



**Sixth Annual Report
Of the
Maternal and Perinatal
Death Review Committee**

**To the
Chief Coroner**

For the Province of Ontario

September 2010

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This report was prepared by Dr. Roger Skinner, Chairperson of the Maternal and Perinatal Death Review Committee; and Ms. Kathy Kerr, Executive Lead – Committee Management.

Introduction

In 2004, Health Canada's Special Report on Maternal Mortality and Severe Morbidity in Canada recommended that all provinces establish a specific maternal death review committee to review all maternal deaths. At the time of that recommendation, the Obstetrical Care Review Committee of the Office of the Chief Coroner reviewed only those maternal deaths that were referred by a Regional Supervising Coroner. Such referrals were requests for the assistance of the committee in the investigation of the deaths. Maternal deaths that were not referred to the committee were not reviewed.

The Office of the Chief Coroner for Ontario accepted the recommendations of the Health Canada report. Effective January 1st 2004, the name and terms of reference of the committee were changed to reflect the fact that all maternal deaths occurring in the Province of Ontario would be reported to the committee in addition to the neonatal deaths and stillbirths already referred by the Regional Supervising Coroners. The name of the committee was changed to the Maternal and Perinatal Death Review Committee of the Office of the Chief Coroner.

The committee's function is to provide assistance to coroners in the investigation of: (1) the deaths of all women who died "during pregnancy and following pregnancy in circumstances that could reasonably be attributed to pregnancy" (2) stillborn cases, and (3) neonatal deaths.

The Coroners Act of Ontario directs coroners to investigate deaths to determine who died and when, the cause of death and the circumstances surrounding it. Once these questions are answered, the coroner is directed to consider whether there are recommendations that could be made, either by the coroner or by an inquest jury, to prevent another death in similar circumstances. Findings of legal responsibility or conclusions of law are not permitted.

The Maternal and Perinatal Death Review Committee case reports are prepared for the Office of the Chief Coroner and are therefore governed by the provisions of the Coroners Act, the Vital Statistics Act and the Freedom of Information and Protection of Privacy Act. As a result, each case review included in the annual report is a summary without identifying details. The recommendations made to the Regional Supervising Coroner and relevant organizations and agencies are included with each case.

It is important to acknowledge that these reports relied upon a review of the records. The Coroner/Regional Supervising Coroner conducting the investigation may have received additional information that rendered one or more of the committee's conclusions invalid. Where that fact was made known to the chair of the committee prior to the production of the annual report, the case review was revised to reflect these findings.

Recommendations were made after a careful review of the circumstances of each death. They are not intended to be policy directives and should not be interpreted as such.

This report of the activities and recommendations of the Maternal and Perinatal Death Review Committee is intended to provoke thought and stimulate discussion about obstetrical care and maternal and perinatal deaths in general in the Province of Ontario.

Questions and comments regarding this report may be directed to:

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Aims and Objectives

1. To assist coroners in the Province of Ontario to investigate maternal and perinatal deaths and to make recommendations that would prevent similar deaths.
2. To provide expert review of the care provided to women during pregnancy, labour and delivery, and to the care provided to women and newborns in the immediate postpartum period.
3. To provide expert review of the circumstances surrounding all maternal deaths in Ontario, in compliance with the recommendations of the Special Report on Maternal Mortality and Severe Morbidity in Canada.
4. To inform doctors, midwives, nurses, institutions providing care to pregnant and postpartum women and newborns, and relevant agencies and Ministries of Government about hazardous practices and products identified during case reviews.

To produce an annual report that can be made available to doctors, nurses and midwives providing care to mothers and infants, and hospital departments of Obstetrics, Midwifery, Radiology/Ultrasound, Anaesthesia and Emergency for the purpose of preventing future deaths.

The Maternal and Perinatal Death Review Committee reviews the deaths of all women who died “during pregnancy and following pregnancy in circumstances that could reasonably be attributed to pregnancy.”

The maternal deaths are classified by the following criteria:

Ante-partum - >20 weeks gestation

Intra-partum - during delivery or immediately following delivery

Post-partum - < 42 days

This report does not review late maternal deaths occurring >42 days, and does not include them in the statistics for the year.

Methodology

Coroners and Regional Supervising Coroners refer cases to the committee for review. At least one member of the committee reviews the information submitted by the coroner and then presents the case to the rest of the committee. After discussion by the committee, a final case report is written consisting of a summary of events, discussion and recommendations (if any), intended to prevent deaths in similar circumstances. The report is sent to the referring Regional Supervising Coroner who conducts further investigation (if necessary) and distributes recommendations to local agencies involved. Recommendations of a more general nature are distributed by the Chief Coroner to agencies and organizations who may be in a position to effect the implementation of such recommendations.

When a case presents a potential or real conflict of interest for a committee member, a temporary member is named from another centre. Alternatively, the committee reviews that case in the absence of the member with the conflict of interest.

When a case requires expertise from another discipline, an external expert reviews the case, attends the meeting and participates in the discussion and drafting of recommendations, if necessary.

Committee Membership (2009)

Dr. Michael Dunn	Neonatologist (Level 3)
Dr. Karen Fleming	Family Physician (Level 3)
Dr. Robert Gratton	Maternal Foetal Medicine
Dr. Steven Halmo	Obstetrician (Level 2)
Ms. Susan Heideman, R.N.	Perinatal Nurse
Dr. Robert Hutchison	Obstetrician (Level 3)
Dr. Sandra Katsiris	Anesthesiologist
Ms. Michelle Kryzanauskas, R.M.	Midwife (Rural)
Dr. Catherine MacKinnon	Obstetrician (Level 2)
Dr. Dilipkumar Mehta	Paediatrician (Level 2)
Ms. Linda Moscovitch, R.M.	Midwife (Urban)
Dr. Toby Rose	Forensic Pathologist
Dr. David Evans Dr. Roger Skinner	Chairperson(s) Regional Supervising Coroners
Ms. Kathy Kerr	Executive Lead

Summary of Cases Reviewed in 2009

This report includes reviews conducted by the Maternal and Perinatal Death Review Committee in 2009. Cases reviewed may involve deaths that occurred in previous years.

Total number of cases reviewed:	46
Total number of recommendations:	69
Number of maternal cases reviewed:	21
Number of recommendations from maternal deaths:	12
Number of neonatal cases reviewed:	16
Number of recommendations from neonatal deaths:	41
Number of stillborn cases reviewed:	9
Number of recommendations from stillborn cases:	16

Case Summaries: Maternal Deaths

Case: 2009-M-1

History

The patient was a 35 year old woman who was at 40 weeks and 4 days gestation when she delivered her first child. She had an uncomplicated spontaneous vaginal birth after 20 hours of labour. An epidural was administered during labour and an episiotomy and repair followed. The baby was a healthy male infant weighing 3900 grams at birth. The patient's white blood cell (WBC) count was elevated at 19.5 on the first postpartum day, however she was afebrile and was discharged in good condition at 42 hours postpartum. The only other health issue was a stable thyroid nodule diagnosed 10 years prior.

On the eighth day post partum, the patient developed a fever and was feeling unwell. She saw her family doctor who suspected mastitis or endometritis and prescribed oral antibiotics. The patient became acutely worse over the following 12 hours with fever, rigors, confusion, lethargy and copious diarrhea. She was brought by ambulance to the hospital and admitted to the ICU with provisional diagnosis of septic shock and possible endocarditis.

The patient's temperature was over 40C while in the emergency room, but there were no localizing findings to identify the source of her infection. She was confused, but did not have a stiff neck or other neurologic findings. She was noted to have some evidence of dehydration, subconjunctival hemorrhages, a mild heart murmur (grade I-II) and a soft nontender abdomen, with no organomegaly. Initial lab tests showed a WBC of 19x10³, Haemoglobin of 108 g, platelets of 178x 10³, creatinine of 148 and AST of 156. Later that day, the patient was noted to have evidence of Disseminated Intravascular Coagulation (DIC) with a low fibrinogen, increased International Normalised Ratio (INR) and Partial Thromboplastin Time (PTT). She also had positive troponins. Her heart was mildly enlarged on chest x-ray. Pelvis ultrasound showed no retained tissue, fluid collections or abscess formation in the pelvis. CT scan of her abdomen on the day of admission showed multiple hyperdense areas in the periphery of her spleen consistent with septic emboli. Echo cardiogram and transesophageal echo were both negative for signs of bacterial endocarditis or vegetations.

The patient was initially treated with triple antibiotic therapy with ampicillin, gentamicin and metronidazole. Vancomycin was added when endocarditis was suggested by CT scan of her spleen showing multiple infarcts and a gram stain from the endometrium showed Staphylococcus Aureus. Metronidazole was continued until her stools proved negative for Clostridium Difficile toxin. Blood cultures also showed staph aureus at 24 hrs of growth. With rehydration and antibiotics, her renal function and DIC improved as did her mental status, however she continued to spike high fevers. CT scan of her head and venous dopplers of her legs after 24 hours were normal. Her cardiac testing confirmed non ST segment elevated myocardial infarct. At 30 hours post admission,

intravenous heparin was commenced as empirical therapy for her multiple splenic emboli and the likely thrombotic myocardial infarction (MI). The possibility of septic thrombophlebitis was also considered given her continued spiking fevers and it was acknowledged that it would require full anticoagulation to clear.

After less than 24 hours of heparin, a marked decrease in the mother's level of consciousness was noted. Coagulation tests had been in the therapeutic range and no elevated blood pressures had been noted. Repeat CT scan of the patient's head revealed a large subarachnoid bleed extending into the lateral third and fourth ventricles. Effacement of the sulci in keeping with increased intracranial pressure was also noted. The patient was intubated and discussions were held with two different neurosurgeons who felt that there was nothing more they could do. The patient died 60 hours after admission.

Postmortem

The significant findings at post mortem examination can all be related to *Staphylococcus aureus* endometritis and sepsis. Sequelae included bacterial vasculitis of the right coronary artery ostium with acute septic myocardial infarction, septic infarcts of the spleen and intracerebral hemorrhage. Disseminated intravascular coagulation had been diagnosed during life.

Cause of Maternal Death:

- *Staphylococcus aureus* endometritis and sepsis with;
- Bacterial vasculitis of the right coronary artery ostium
- Acute septic myocardial infarction
- Septic infarcts of the spleen
- Intra cerebral haemorrhage

Discussion

The mother died of complications of *Staphylococcus Aureus* Endometritis and sepsis.

Recommendations:

None

Case: 2009-M-2

History

The mother was a 34 year old grand multiparous woman who had delivered her tenth baby at home ten days prior to her admission. She had been brought to hospital after

the birth and stayed for 24 hours. She made arrangements for her baby to be adopted. She was re-admitted with lower abdominal and right flank pain as well as fever, leukocytosis and foul smelling lochia. Clinically, the attending obstetrician diagnosis was endometritis. Blood cultures were negative. E. coli was grown from her urine and treated. She was treated with IV antibiotics and the fever and leukocytosis resolved after 72 hours. She was then switched to oral antibiotics.

The patient continued to have marked pain, especially in her right buttock and leg. Physiotherapy helped her with walking aids. The hospitalist was consulted and diagnosed sciatica. She eventually had a CT scan of her back which showed disc herniation at L4-L5. She was morbidly obese and the question of deep vein thrombosis was raised, but venous dopplers of her legs showed no evidence of a clot.

Eventually the pain was improving and she was taking oral medications. Arrangement had been made for her to be discharged home on the day of her death - 22 days postpartum. The patient collapsed while in the washroom. Within two minutes, she was unconscious with no pulse. A code was called and CPR initiated. The code team was unsuccessful in their efforts to restore a pulse.

Postmortem

Autopsy showed evidence of a large pulmonary embolus, which is considered to have arisen from thrombosed parametrial and pelvic blood vessels. Also noted was evidence of acute (mild) and chronic endometritis and thrombosed peri-uterine blood vessels. Note was also made of obesity, polycystic kidneys and a benign thyroid nodule.

Cause of death: Pulmonary Embolus

Discussion

Venous thromboembolism, including deep vein thrombosis and pulmonary embolism, remains the most common cause of maternal death. This woman had additional risk factors in grand-multiparity and morbid obesity as well as infection and immobilization while in the hospital with endometritis. Pulmonary embolism occurs most commonly in the postpartum period. Clinical presentation varies from mild dyspnea, to dramatic cardiopulmonary collapse, as occurred in this case. Proximal pelvic vein thrombosis is difficult to diagnose as compression ultrasound is frequently negative. Magnetic resonance is the only test that has been shown to have a high sensitivity, specifically for iliac vein thrombosis.

Recommendations:

None

Case: 2009-M-3

History

The deceased was a 35 year old G2P1 with an Estimated Date of Delivery (EDD) of March 21, 2007. Her first pregnancy was delivered in 2004 by Caesarean section for a non-reassuring foetal heart rate tracing and meconium for a 3620 gm male infant. The plan was for her to Vaginal Birth After Caesarean (VBAC) in this pregnancy.

Routine prenatal laboratory investigations, Integrated Pregnancy Screening (IPS), second trimester ultrasound and Glucose Challenge Testing (GTT), were normal. Her antenatal course was uneventful. Her past medical history was unremarkable.

Course in Labour and Delivery

The patient presented to the labour and delivery unit at the hospital on March 18, 2007 at 2141 hours in early labour and under the care of her family doctor. On examination, the cervix was 3.5 cm dilated, 100% effaced and the vertex at spines -2. Membranes were intact. The foetal heart was normal. An epidural was placed at 2204 hours. At 2225 hours, the cervix was 4.5 cm dilated. An Artificial Rupture of Membranes (ARM) was performed for a small amount of bloody fluid. Contractions were q 2-3 minutes and mild to moderate in intensity. Mild variable foetal heart rate decelerations were noted at 2313 hours with good recovery to normal baseline. At 0005 hours, the cervix was 5 cm dilated. Foetal heart rate decelerations continued and the attending physician was paged at 0010 hours. The obstetrician attended at 0030 hours and inserted an intrauterine pressure catheter to assess uterine contractions. The obstetrician was subsequently re-paged at 0055 hours because of concerns with the foetal heart rate tracing. A decision was made to proceed with repeat Caesarean section for a non-reassuring foetal heart rate tracing. The patient was noted to be having frequent contractions and a nitroglycerin patch was placed while preparations were made for Caesarean section. At 0109 hours, under epidural anaesthesia, the patient was delivered of a 3175 gm male infant assisted by a vacuum extractor. Apgars were 9 and 9 at one and five minutes and cord gases were normal. There were no tears or extensions and the low transverse uterine incision was closed in two layers and good haemostasis obtained. The patient was transferred to the PACU at 0145 hours.

In the PACU, the patient's blood pressure was 76/40 and pulse 110 beats per minute (bpm). Fundus was firm, lochia was normal and the abdominal dressing was dry. At 0150 hours, blood pressure was 77/42 and pulse 90 bpm. Anaesthesia was paged. Pentaspan 500cc was given IV. At 0205 hours, BP was 83/55 and pulse 106 bpm. Fundus was firm and lochia moderate. The anaesthetist was paged at 0222 hours because of persistent low blood pressure. The anaesthetist attended at 0224 hours. Pentospan 500cc was repeated at 0242 hours. The patient remained hypotensive and her pulse climbed to 115 bpm at 0320 hours. At 0425 hours, the dressing was changed for serosanguinous drainage. Pulse was 125 bpm. Urine output since delivery was 100cc. The anaesthetist was again informed of her low blood pressure and advised that the patient be slowly repositioned into a high Fowlers position. At 0433 hours, fundal

palpation was noted to be painful. At 0512 hours, the dressing was again changed for serosanguinous drainage. The anaesthetist again attended at 0535 hours and the obstetrician was informed of the hypotension. Orders were received for CBC, coagulation screen and cross match. A pressure dressing was ordered for the wound. The obstetrician attended at 0607 hours and observed marked pallor and a distended, tense abdomen. The hemoglobin was reported at 41gm, platelets 173,000. Emergency preparations were made to return the patient to the Caesarean section room for laparotomy for intra-abdominal hemorrhage.

A spinal anaesthetic was placed. Shortly after surgery was commenced, the patient went into ventricular tachycardia and a "Code Blue" was called at 0648 hours. With the resuscitation ongoing, the abdominal incision was opened and approximately two litres of blood was found in the peritoneal cavity. Inspection of the uterine incision revealed venous bleeding only. The incision was re-sutured. The uterine arteries were also ligated. The uterus was noted to be flaccid and fundoplication sutures were then placed to treat uterine atony.

Continued exploration revealed a laceration of the uterine vein at the left margin of the uterine incision. This was the first time that this had been noted and it was felt that it had occurred during the course of the exploration in this procedure, rather than at the time of Caesarean section. The area was sutured as well. Generalized bleeding was encountered, including from the endotracheal tube, consistent with DIC. Surgical lap sponges were then packed between the uterine incision and symphysis pubis in order to apply pressure to the lower uterine segment. The patient received a total of 18 units of packed cells and 6 units of FFP. The patient could not be resuscitated and the code was called at 0802 hours.

Post Mortem

Autopsy revealed a large amount of intraperitoneal blood, but no large uterine vessel injury as a bleeding point. No other intra-abdominal source of bleeding was identified. There was no evidence of retained products of conception or amniotic fluid embolus.

The cause of death was haemoperitoneum following Caesarean section.

Discussion

This patient died from hemorrhagic shock due to intraperitoneal bleeding occurring immediately following repeat Caesarean section for failed VBAC and non-reassuring foetal heart rate tracing.

At the time of Caesarean section, there were no difficulties encountered and the operative report (dictated the following day) indicates that good haemostasis was obtained at the time of closure of the uterine incision.

On arrival in the post-anaesthetic care unit, the patient's blood pressure was low and remained so, despite crystalloid and Pentaspan infusions. The uterine fundus was noted to be firm and vaginal blood loss was within normal limits. Initially, there was no bleeding from the skin incision. It does not appear that consideration was given to the possibility of intra-peritoneal bleeding either by the PACU nurse or the anaesthetist. With no explanation for the hypotension forthcoming, and no response to the Pentaspan infusion, the obstetrician should have been informed of the patient's condition much earlier in the course of events, probably some time between 0200 and 0230 hours.

When the obstetrician was notified at 0535 hours, blood work was ordered and the patient was assessed. At that time, the diagnosis of intraperitoneal bleeding was evident and preparations were made for laparotomy. The surgical approach in such a situation is to resuscitate the patient and control the bleeding concurrently. Unfortunately, by then, the patient's condition had deteriorated to the point that she went into cardiac arrest. As a result, any sites of surgical bleeding may not be apparent due to the extreme hypotension or total lack of circulation. Manual compression of the descending aorta by the surgeon would have been the most effective measure to try to assist in restoring a circulating blood volume while resuscitative measures were being applied. There is no mention of this being done in the operative report. Given the extremely critical condition at the commencement of the surgery, it is unlikely that this intervention would have changed the outcome in this case.

Recommendations

1. The _____ Hospital obstetrical unit should review its PACU protocols, particularly as pertains to notification of unstable vital signs to both the anaesthetist and obstetrician.
2. Obstetricians are reminded of the utility of applying manual compression to the descending aorta to control pelvic bleeding as an aid in the establishment of a circulating blood volume during resuscitation of hemorrhagic shock and Disseminated Intravascular Coagulopathy.

Case: 2009-M-5

History

The deceased was a 39 year old G2 P1 who had her first baby by Caesarean section in Russia. She was admitted to the hospital at 34 weeks gestation in her second pregnancy, with known placenta previa. Posterior placenta previa was identified on a 17 week ultrasound and confirmed on serial ultrasound exams to 37 weeks. These ultrasounds identified some placental infarcts, but there was no suggestion of abnormal placentation. The patient had an elevated alpha-fetoprotein (AFP) in the second trimester, but ultrasound showed a normal baby. She was followed by an obstetrician and a maternal foetal medicine specialist. She did not experience any bleeding and the growth of the foetus was normal.

At 38 weeks gestation, the patient was taken for scheduled Caesarean section under spinal anaesthetic. Note was made that the placenta appeared to be more anterior than was recognized on the ultrasounds. The obstetrician noted that she had to go through the placenta to deliver the baby. A healthy female infant was born, but heavy bleeding ensued when removal of the placenta was attempted. A second obstetrician was called to help and further attempts to remove the placenta and stop the bleeding were made. Hemabate and Misoprostol were used to maintain uterine tone, however the bleeding persisted and a clinical diagnosis of placenta accreta was made. After discussion with the husband, hysterectomy was commenced 30 minutes after the birth of the baby. Bleeding continued to be extremely heavy.

The patient sustained a cardiac arrest approximately one hour after the birth and near the completion of a subtotal hysterectomy. She was successfully resuscitated from the arrest, but continued to bleed heavily. The bleeding persisted even after removal of the uterus and a diagnosis of DIC was now apparent. A vascular surgeon was called and attempts were made to ligate the internal iliac arteries. These attempts were impeded by ongoing massive bleeding and coagulopathy. Over the next four hours, the surgeons made ongoing attempts to control the bleeding with packing and sutures. The anaesthesia team continued the resuscitation with multiple doses of ephedrine, dopamine and vasopressin. The patient received massive volumes of crystalloid and albumin. She was transfused with approximately 60 units of packed red blood cells, 40 units of FFP, 30 units of cryoprecipitate 10 units of platelets and 2 doses of activated factor 7. Eventually, her abdomen was packed, but left open and she was transferred to the intensive care unit.

An hour later, the patient was taken to the radiology department for angiography and an embolization of her left internal iliac artery was performed. The right internal iliac was found to be completely surgically ligated. The patient however, continued to require blood products and attempts to correct her acidosis failed. Haemodialysis was started, but the patient sustained an asystolic cardiac arrest. She was again resuscitated for approximately one hour, but ultimately, all efforts failed and she died about nine hours after the birth of her baby.

Post Mortem

No autopsy was performed.

The pathological examination of the hysterectomy specimen and placenta confirmed the diagnosis of placenta accreta.

The cause of death was multi-organ failure secondary to DIC due to postpartum hemorrhage from placenta previa and placenta accreta.

Discussion

The patient died of complications from placenta accreta including postpartum hemorrhage, DIC and multi-organ failure.

Recommendations

None

Case: 2009-M-6

History

The deceased was a 37 year old woman, 11 weeks post repeat Caesarean section.

Her past medical history was remarkable for pulmonary embolism and two separate episodes of deep vein thrombosis after arthroscopic surgery, adrenal insufficiency, rheumatoid arthritis, and recurrent spontaneous abortion (six in total). After dilation and curettage for one miscarriage, she developed massive bleeding and DIC.

She had one previous term gestation delivered by Caesarean section. This pregnancy was complicated by hyperemesis, arthritic problems, gestational diabetes, and hypertension.

The patient's recent pregnancy was complicated by severe hyperemesis with home IV therapy. Due to her body habitus and difficulty maintaining peripheral IV lines, a Percutaneously Inserted Central Catheter (PICC) was used.

In the mid-trimester, she developed fevers, chill and increased right hip pain. A blood infection with *Serratia* and *Enterobacter* was diagnosed. Hip x-rays failed to show evidence of septic arthritis. An echocardiogram was done. Her PICC line was removed and replaced and she was referred to a tertiary centre. She was treated with six weeks of IV Meropenem for presumptive diagnosis of mixed organ line infection. During this time, she also had some bleeding, but no abruption was confirmed. Her Dalteparin was held, but eventually restarted. Her rheumatoid arthritis was worse during her pregnancy and she was started on Enbrel and Duragesic patches.

A repeat Caesarean was performed in the tertiary centre at term. The mother and healthy baby were discharged at 48 hours post-op on Fragmin, Demerol and Prednisone.

Eleven weeks post partum, she presented to the Emergency Room of her local hospital after a four week history of intermittent fevers. Her medications at the time of her presentation to Emergency included: Prednisone 10 mg by mouth, three times a day, Demerol tabs when necessary, Tylenol #3 when necessary and Plain Tylenol when necessary. She was known to be allergic to contrast media, shellfish and venom.

The mother was admitted to the hospital Emergency Room at 1623 hours with complaints of vomiting, shortness of breath, chest pain and generalized weakness. She gave a history of feeling unwell for about four weeks with intermittent high fevers up to 40C. She reported a productive cough also for four weeks with a recent course of oral Cefazol. After the antibiotics, she reported the sputum had changed from green to clear, but the fever had persisted. For two weeks she had been short of breath. She reported that she had frequent vomiting for the last four weeks, but that this had gotten much worse. The reason she came to the hospital was because she was unable to keep hydrated or take her pills. She reported diarrhea for about five days, with up to nine bowel movements per day which were liquid and orange in colour. She reported left chest pain for six days.

In the Emergency Room, she appeared pale and unwell. Her heart rate was 130 bpm, blood pressure 104/70, respiratory rate 18 and temperature 36.9C. Her heart sounds were normal and her chest was clear, but she was tender in the epigastric area, with no rebound. Lab tests revealed Hb=81, WBC=10.3, Platelets=114, glucose=6.2, urea=14.8, creatinine=123, sodium=126, potassium=5.3, chloride=96, carbon dioxide=18, ALT=240, alkaline phosphatase=148, amylase=49, INR=2.6, PTT=39, d-dimer>4000. The chest x-ray showed an enlarged heart suggesting pericardial effusion. Her urinalysis showed leukocyte esterase=trace, protein=3+, ketone=negative, bilirubin=1+ and blood=3+. Blood cultures were taken. She was assessed by the emergency doctor who referred her to internal medicine.

The internist saw the patient about three hours later and noted the same history. Her blood pressure at this time was 124/43, heart rate 130 and respiratory rate 30. She was again afebrile. Physical exam was unchanged. This doctor made note of the abnormal lab results, except for the chest x-ray and the coagulation which may not have yet been reported when he attended at 1900 hours. The doctor ordered an IV bolus of normal saline, IV hydrocortisone and oral fludrocortisone, morphine, gravol, ferrous gluconate and subcutaneous Dalteparin. The morphine order was changed to Demerol at the patient's request. No morphine was given. The doctor also ordered further blood, urine, sputum and stool cultures as well as clostridium difficile toxin testing. Investigations for the next day included abdominal ultrasound, venous ultrasound of legs, V/Q scan and repeat CBC, electrolytes, blood urea nitrogen (BUN), creatinine, calcium, albumin, thyroid stimulating hormone (TSH) and glucose.

The IV was commenced with difficulty at 2130 hours and the patient received Demerol and gravol through the IV every four hours for pain. Dalteparin was given at 2145 hours. Hydrocortisone was started at 0400 hours. The patient remained afebrile, but the nurses reported that she was hyperventilating at times. At 0835 hours, the patient was taken to ultrasound by stretcher. While in ultrasound, she had a respiratory arrest and became non-responsive. Within minutes, at 0850 hours, she was found to be in cardiac arrest. She was intubated, CPR initiated and a full code performed with five doses of Epinephrine, two doses of Atropine, Solumedrol and Levophed. The patient was stabilized between 0915 and 0950 hours when she had another cardiac arrest. This time, efforts to restore her circulation failed and she was pronounced dead.

The following day, the patient's blood cultures were reported growing gram positive cocci in pairs and chains.

Postmortem

At autopsy, acute infective endocarditis involving mitral, tricuspid and aortic valves. Infected vegetations on the surface of the aortic valve associated with the right coronary artery was found.

The cause of death was Septicemia due to infective endocarditis.

Discussion

The patient died of sepsis from subacute trivalvular endocarditis worsened by her significant immuno-compromised state from her multisystem organ disease, her use of chronic steroids and her underlying adrenal insufficiency. The illness had evolved over the course of four weeks during which time she was treated once with oral antibiotics, but a complete workup was not performed. In light of her adrenal insufficiency and chronic steroid usage, more aggressive investigation and treatment should have been undertaken. When the patient presented to the Emergency Room, she had advanced sepsis syndrome which was unrecognized likely due to lack of fever or leukocytosis. Chronic steroid use is well recognized to mask many signs of sepsis, including fever. The patient did however, have tachycardia, tachypnea, liver failure and DIC. She should have received more intensive therapy at that time with IV fluids and antibiotics as well as close clinical monitoring in a critical care unit.

Recommendations

Caregivers are reminded of the importance of properly educating patients on steroids to seeking care for fevers or other signs of infection. Caregivers are reminded that chronic steroid usage can mask the usual signs of infection. Caregivers are directed to review the "Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008" at www.survivingsepsis.org.

References

Dellinger RP, Levy MM, Carlet JM, et. al: Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008 [published correction appears in Crit Care Med 2008; 36:1394-1396]. Crit Care Med 2008; 36:296-327.

Case: 2009-M-7

History

The deceased was a 26 year old G1P0, pregnant with twins, with an estimated date of confinement (EDC) of September 28th, 2008. On the chart, she is referred to as having a “monochorionic diamniotic twin pregnancy”, however despite the foetuses being both of the same sex, chorionicity does not appear to have been confirmed by ultrasound. The patient was followed throughout her pregnancy by an obstetrician. Her past medical history was unremarkable and her pregnancy had been progressing normally with symmetrical growth as verified by the most recent ultrasound on July 7th, 2008 at 28 weeks gestation. The placentas were “fused” anteriorly and clear of the cervical os.

At 0647 hours on July 30, 2008, the patient presented to triage at the labour and delivery unit of the hospital. She complained of abdominal cramping which had started about 75 minutes earlier. At 0715 hours, the external foetal monitor indicated a foetal heart of 148/?. At 0745 hours, the external foetal monitor again indicated a foetal heart of 145/?. At 0810 hours, an ultrasound was conducted by the attending physician on call.

The foetal heart was not present for one of the twins and apparently bradycardic for the other. A monitor strip was not available for review. At this time, the patient gave a history of vague abdominal cramping with no vaginal bleeding. The patient was sitting up. Vital signs were otherwise normal, with a pulse of 96 and blood pressure of 102/64. The abdomen was soft and only mildly tender. A decision was made to proceed to an emergency Caesarean section for non-reassuring foetal heart.

The patient was in the operating room by 0815 hours. Anesthetic was started at 0825 hours with a rapid sequence induction and intubation. The Caesarean was initiated with a Pfannenstiel incision with the delivery of twin A at 0827 hours. This delivery was 17 minutes after the ultrasound confirmation of the concerning foetal hearts. The twin weighed 2020 grams, had Apgars of 0, 0, 0 and could not be resuscitated. Twin B had advanced resuscitation and was transferred to the PICU.

The newborn record is not available, but according to the coroners investigation statement the second twin did not survive either.

There was “a significant amount of blood in the peritoneal cavity as well as clot.” Approximately 1 to 1½ litres of clot was behind the uterus. After the hysterotomy incision, a “significant amount of serosanguinous fluid” came out. “Placenta at this point was not attached at all to the uterus and literally fell out of the uterus suggestive of a complete and total placental abruption.” After removing a significant amount of blood that was in the posterior pelvis behind the uterus, the splenic and liver areas in the upper abdomen were inspected for any signs of active bleeding. No bleeding could be found. The abdomen was closed with the presumption that that blood in the abdomen had been retrograde through the fallopian tubes.

The anesthetist notes indicate that the patient was stable throughout the procedure and was extubated after the Caesarean section. The procedure ended at 0845 hours. There were apparently two units of uncrossed matched blood that were returned to the blood bank after the end of the Caesarean with the anticipation of waiting for proper crossed products. During the time of transfer to the Post Anaesthetic Care Unit (PACT), the patient became suddenly pale with decreased respiratory effort. She was re-intubated, CPR was initiated and a code blue was called at 0900 hours. The resuscitation efforts continued until 0946 hours at which time the patient was to be transferred to an Intensive Care Unit (ICU) bed.

During the resuscitation, the patient had cardiac compression from 0900 to 0926 hours. There were multiple doses of adrenaline, bicarb and vasopressors as well as blood products. A defibrillator was also used. The patient received 12 units of packed cells by 0921 hours and a further unit of Red Blood Cells (RBC), and one unit of FFP, by 0929 hours.

The patient was received in the ICU at 1015 hours. A thorough exam by the Critical Care Transport Unit (CCTU) fellow at 1100 hours documented the pupils as fixed and dilated, no cough, gag, corneal reflex or movement of limbs, and with positive dolls eyes. For this early time in the CCTU, the patient was unstable and there was concern of increasing abdominal size and potential intra-abdominal bleeding. The hemoglobin was 51 and blood pressure was 60/40. The patient had evidence of DIC with INR greater than 7. She had a further four units of RBC, six FFP, five platelets and ten units of cryoprecipitate.

Due to the distending abdomen and the unstable condition, the obstetrical team was called. A laparotomy was initiated in the CCTU. The patient's staples were removed from the recent incision. There was obviously a large amount of blood coming from the abdomen. A midline incision was performed. The abdomen was evacuated of blood. The clamps were placed on the uter-ovarian and uterine arteries. The abdominal bleeding continued. A vascular surgeon arrived to assist. The patient was then transferred back to the Operating Room (OR). There was oozing from all previous surgical sites. A total hysterectomy was initially performed by the obstetrical team.

The patient became more unstable and required an aortic clamp. Extensive investigation of the abdomen was made to find further bleeding areas. Evidence of a ruptured aneurysm of the splenic artery was found. The abdomen was closed with five sponges remaining in the peritoneal cavity and the retroperitoneum for tamponade.

During the OR, the patient had "massive doses of inotropes and vasopressors", as well as two level one infusers and central lines. She was unstable with two episodes of electromechanical dissociation (EMD) arrest for 2 minutes and 8 minutes respectively during which CPR was required intraoperatively. During this second procedure, the patient required 16 units of red blood cells, 15 of platelets, 2 of FFP as well as factor 7, extensive crystalloid and Pentaspan. She was sent back to the ICU at 1500 hours. The patient remained unstable and died shortly after returning to the ICU.

Post Mortem

Pathology reports showed a normal post partum uterus, diamniotic dichorionic twin placenta with velamentous insertion of cord A, 2.5 cm from the edge of placental plate to the bifurcation. Focal mild villous edema and small subchorionic fibrin thrombus.

Splenic artery aneurysm (1.5 cm diameter), ruptured clinically (repaired); Bilateral severe acute pyelonephritis, bilateral renal tubular necrosis; injuries consistent with resuscitation efforts including fracture of mid sternum, anterior rib fractures (right numbers 2-7, left numbers 2-7)

Final pathological determination of death was complications of intra-abdominal hemorrhage. Sources of hemorrhage: ruptured splenic artery aneurysm (repair); coagulopathy; massive placental abruption (clinically.) Contributing factors: acute pyelonephritis.

Discussion

This patient had a sudden and completely unpredictable onset of an acute complication of her pregnancy at 32 weeks' gestation. On initial assessment in the hospital, one of the foetal hearts could not be determined. This was apparently not checked again for another half hour and an attending physician was not available until 55 minutes after the initial assessment to confirm that one of the foetuses had likely died and there was a significant bradycardia in the other. It is not clear whether this would have made a difference in the outcome, particularly for twin B. The decision was made for a Caesarean due to a non-reassuring foetal heart. The subsequent care was expedient.

As the mother was stable at this point and during the first OR, she would not likely have had any degree of hypotension to affect the well being of the foetuses.

At surgery, it would appear from the OR note that there was an excessive amount of blood in the abdominal cavity already and though an upper abdominal cause of the bleeding was not determined at that time, it is likely that it was already active. It would seem unlikely to have as much blood as was described on the OR report coming out through the fallopian tubes.

It was noted that "serosanguinous" fluid was coming out of the uterus at the time of delivery. It is difficult to tell to what degree intrauterine bleeding contributed to the outcome. As one foetus had already died, and the other significantly stressed, the clinical diagnosis of abruptio placenta appears to be the likely cause of the perinatal death, despite the fact that the pathological examination of the placenta did not reveal any findings supportive of this.

The subsequent cardiopulmonary collapse was not anticipated. The resuscitation was immediate, aggressive and initially successful, though by the time the patient arrived in the ICU, her chance of intact survival was felt to be guarded. Through this time, she

was developing a coagulopathy that contributed to the massive blood loss. Although the patient was in critical condition, it was felt that the only alternative was to take her back to the OR.

The subsequent finding of an aneurysm of the splenic artery and a split in the aneurysm would appear to give a more adequate answer to the haemoperitoneum that was initially found at the time of Caesarean section. Combined affects of blood loss from this, the abruptio placenta and the ensuing coagulopathy, all contributed to her critical state and subsequent death, despite aggressive and appropriate care.

Recommendations

None

Case: 2009-M-8

History

The deceased was a 19 year old G1P0 with a last menstrual period (LMP) of February 20th, 2008, giving an estimated date of delivery of November 27, 2008. She had an ultrasound May 30th which was consistent with a due date of January 12, 2009. The ultrasound was ordered at a clinic visit on May 28th 2008 where she attended for an upper respiratory tract infection. The pregnancy issue was first raised on this visit. The patient was an immigrant from Mexico who next presented to the medical centre on June 9, 2008 for initial prenatal counseling.

The patient returned on June 20, 2008 for a more complete assessment. The pregnancy, as well as her history of significant substance abuse (alcohol, marijuana, cocaine) and a past history of depression, was documented. She disclosed that her partner was no longer involved and she was unemployed living with her parents who were very supportive. She was counseled around substance abuse and declined referral for treatment. She admitted to being depressed currently, but denied current suicidal or homicidal thoughts. She and her parents were provided with crisis numbers and instructions about going to the hospital if any suicidal thoughts evolved. The patient was referred to both a psychiatrist and an obstetrician for further care.

An obstetrician gynecologist saw the patient on July 8, 2008 and subsequently referred her to a clinic for a therapeutic abortion the following week. After the appointment with the obstetrician, the patient went home. She told her mother that she was going to the garage to smoke. When she did not return, her mother went to check on her. The patient was found hanging in the garage. She was vital signs absent (VSA) and her mother called 911.

The patient was taken to hospital where she was resuscitated. She subsequently had three further cardiac arrests and developed Adult Respiratory Distress Syndrome. She was seen by neurology and in view of the grim prognosis, family elected to remove her from life support on July 11, 2008.

Post Mortem

Autopsy was consistent with delayed hanging, with hypoxic ischemic encephalopathy.

Discussion

The deceased was a young woman with a history of depression and drug abuse who, when she found herself pregnant and without a partner, committed suicide. Her caregiver's had appropriately explored her depression and inquired about suicidal ideation. She and her family were offered crisis intervention and referral to a psychiatrist was undertaken.

Recommendations

Obstetrical Care Providers are reminded that psychiatric disease may complicate pregnancy and may be a cause of maternal mortality.

Case: 2009-M-9

History

The deceased was an 18 year old G2T1L1 with an estimated date of confinement of December 14, 2007. She was followed in this pregnancy by her family doctor. She was seen regularly and was doing well at her most recent visit on November 28, 2007. Her prenatal testing had been normal. She had asthma due to second hand smoke and used Ventolin and Flovent as necessary. Her health has otherwise been unremarkable.

Her past medical history included the uncomplicated pregnancy and vaginal birth of a 6 pound 6 ounce infant at term in 2006. Her partner for both of these pregnancies is known to be Human Immunodeficiency Virus (HIV) and Hepatitis C positive. She continued to have unprotected sex on occasion. She had testing for HIV over the last two years, including in this pregnancy, and these tests were negative. She had recent testing, just prior to her delivery. She had been living in a challenging situation with her partner and his six children who she was anticipating adopting.

The patient presented in labour at approximately 38 weeks gestation on November 30, 2007. She went on to have an uncomplicated spontaneous vaginal birth approximately eleven hours later. She delivered a 2.97 kg male infant with Apgars of 7 and 9. There was no episiotomy or vaginal tears.

On admission, the patient's temperature was 37.6 C, with other vital signs within normal limits. Approximately one hour prior to delivery, her temperature was 37.7 C. The temperature subsequently came down to normal, shortly after admission and remained normal, as did other vital signs until the time of discharge.

The patient had a vaginal/rectal Group B Streptococcus culture on November 9, 2007 which was negative for group B streptococcus and therefore did not warrant prophylactic antibiotics.

Lab testing approximately six hours after admission, while in active labour, revealed a White Blood Cell count of 22000, a haemoglobin of 137 and normal platelets.

The patient did well post partum, was breastfeeding her infant and was afebrile. She was discharged home later in the day on December 1, 2007.

The patient subsequently presented to the emergency department of the hospital on the morning of December 3, 2007. She gave a history of starting to feel unwell the night before with vague malaise. On awakening in the morning, she felt very unwell, was weak, was unable to move her right leg and was unable to ambulate. She was complaining of dyspnea and stomach pain. Paramedics were called and arrived at her house at 0730 hours on December 3, 2007. She was tachycardic, cool, mottled and tachypnoeic at the time. An intravenous was started and she was taken to the emergency department where she arrived at 0807 hours.

On assessment by the emergency doctors, she was felt to be septic. She had a white cell count of 2.1, lactate of 4.2 and bicarbonate of 10. The intensive care staff were consulted and the Intensive Care Unit (ICU) fellow, and his attending physician, assessed her at 0850 hours. During this time, the patient had central lines placed. She had multiple antibiotics started and aggressive multi-system treatment, including care for renal failure as she was anuric. She was transferred to the ICU at 1100 hours. She was intubated because of increasing difficulty breathing. Despite aggressive treatment with vasopressors, inotropes, care for her renal impairment, intubation, ventilation, and broad spectrum antibiotics she continued to deteriorate and subsequently passed away at 1240 hours on December 3, 2007.

Blood cultures that were taken shortly after the patient's admission grew Group A Streptococcus (S) Pyogenes with normal sensitivities.

Post Mortem

Summary of findings:

1. Severe myometritis with inflammation of the cervix
2. Necrotizing fasciitis involving the vulva, right thigh and upper inner thigh
3. DIC with micro-thrombi in various organs
4. Intravascular bacterial dissemination
5. Acute tubular necrosis
6. Early centrolobular necrosis of the liver
7. Premortem blood cultures positive for strep A pyogenes

8. Generalized edema and effusions in pleural cavities, peritoneal cavity and pericardium
9. Bilateral pulmonary edema

Pathology of Placenta

This was a normal placenta, without evidence of infection.

The cause of death was determined to be septic shock due to invasive Group A Streptococcus Pyogenes infection with evidence of Uterine infection and necrotizing fasciitis of the vulva and upper thigh. It was felt that the most likely source of infection being from the vagina.

Discussion

This young woman had a recent uncomplicated pregnancy and vaginal birth. Her temperature was mildly elevated in a recent labour; however she became afebrile afterwards and for the duration of her hospital stay. This is not unusual in labour. She had a high white cell count from a sample taken while she was in active labour. This too is a very usual finding and does not indicate a subsequent fatal infectious illness. Her clinical course during labour and in the early post partum time was otherwise normal.

When the patient presented to the emergency department, she was in severe septic shock. It is not uncommon in this state to have low white cell count along with all the other findings that were determined during her assessment and testing.

This was an unpredictable illness due to a bacteria for which screening is not recommended. The case-fatality rate for Group A Streptococcus sepsis is high.

The care that she received was expedient and intensive, but her degree of septic shock was not recoverable.

Recommendations

None

Case: 2009-M-10

History

The deceased was a 28 year old G1P2 of uncertain menstrual dates. An ultrasound on November 11, 2008 gave a gestational age of 7 weeks 3 days. The uterus was markedly enlarged due to the presence of uterine fibroids, the largest measuring 11.9 X 15.1 X 9.0 cm. She had seen a gynaecologist in 2006 because of uterine fibroids,

menorrhagia and dysmenorrhoea. After reviewing the management options, it was recommended that she consider undergoing myomectomy. This was declined.

The patient was re-evaluated on March 3, 2008 for her gynaecological complaints. She was found to have a uterine size consistent with a 26-27 week gestation. She was again offered surgical intervention.

The patient returned on November 3 having decided to have surgery. A pregnancy test was ordered and on November 10, she was informed that it was positive. She was seen again on November 14 for review of the earlier ultrasound and discussion regarding the risk factors for her pregnancy. During this time, she had been experiencing intermittent abdominal pain.

The patient presented to the emergency department at the hospital on November 24 complaining of severe pelvic pain. She was not having any vaginal bleeding. Vital signs were stable. Her haemoglobin was 83 gm, PTT and INR were normal. An ultrasound showed a viable pregnancy at 9 weeks 4 days and a moderate size subchorionic bleed. The pain was likely due to degeneration of a fibroid and the subchorionic bleed. Her pain improved over the course of her stay in the emergency department and she was discharged home.

On November 25, the following day, she spent a lot of time sleeping. Her husband stayed home from work because of her condition. At approximately 1600 hours, he heard a thump in the adjoining room and went in to find her sitting on the ground slumped forward. He helped her up, but she fell again, became unconscious and stiffened. The husband called 911 at approximately 1626 hours then attempted to revive her. She gained consciousness and decided to go to the hospital without waiting for the ambulance. While taking the elevator down to the parking garage, she had to lean against the elevator wall for support. During this time, officials from the management office had been contacted by the 911 operator and they asked to speak with the husband. When he returned, his wife was unconscious, her eyes were closed and she was jerking. The ambulance arrived almost simultaneously. The paramedics found the patient to be vital signs absent (VSA) and full resuscitation was commenced. She was transferred to the emergency department where resuscitation efforts were continued, but she could not be revived. The patient was pronounced dead at 1717 hours.

Post Mortem

The autopsy revealed bilateral central and peripheral pulmonary emboli and thrombosis of the pelvic veins.

The uterus contained a 9 week pregnancy and fibroids. The total weight of the uterus was 3.55 Kg.

Discussion

This pregnant woman died suddenly as a result of a pulmonary embolus originating from thrombosed pelvic veins. Predisposing risk factors for the development of pelvic

vein thrombosis were the pregnancy and venous stasis secondary to the large fibroids. Although she had been having intermittent pelvic pain for approximately three weeks before the event, and an episode of severe pain the day before, it was reasonable to attribute this to discomfort caused by the fibroids and possibly threatened abortion. A diagnosis of pelvic vein thrombosis and consideration of imaging studies pertaining to this diagnosis would not normally be undertaken in this setting.

Recommendations

None

Case: 2009-M-11

History

The mother was a 41 year old G6P4 with one previous spontaneous abortion. Approximately 14 months prior to her presentation, she had her fourth baby by low transverse Caesarean section for failure to progress in labour and atypical foetal heart monitoring. Her three other births were spontaneous vaginal births. In this pregnancy, she had planned to have a repeat Caesarean and tubal sterilization. The current pregnancy was uneventful. Blood pressures on her antenatal records were 110-120/59-67. She was followed by an obstetrician who had booked her for Caesarean on the day after her presentation.

She was 39 weeks when she came to the hospital with contractions in active labour with ruptured membranes. Her vital signs are recorded as 'stable' and the electronic foetal monitor tracing was normal. Her cervix was found to be 6 cm dilated. The obstetrician on call discussed with her the options of proceeding with Caesarean birth or continuing with labour as a vaginal birth after Caesarean. The mother requested Caesarean and it was completed 90 minutes after her arrival in labour.

Spinal anaesthesia was used. The anaesthetist recorded an initial blood pressure of 125/70 and 100/50 for the last 30 minutes of the surgery. Pulse was 85 bpm and O2 saturation was 98% throughout. The baby was a healthy male, 3.27 kg, with Apgar scores of 9 and 9 at one and five minutes. The Caesarean low transverse incision, repaired in two layers, and sterilization, was performed with Filshie clips. The operative note does not report any complications with the surgery. The estimated blood loss was 700 ml.

The mother arrived in the recovery room at 0130 hours. Her initial blood pressure was 88/44, but improved to 100/50 and pulse 80-90 bpm for the next hour. At 0250 hours, the nurse called the obstetrician to come to see the mother as her blood pressure was 70/40, pulse 100 bpm and there was little urine output. At 0315-0330 hours, the obstetrician assessed the mother and irrigated, and ultimately replaced, the catheter.

At 0345 hours, the mother was noted to be short of breath with blood pressure 70/38. The nurse gave her oxygen at 10 litres/minute and called the obstetrician who ordered blood tests which were taken at 0425 hours. At 0400 hours, the nurse noted the mother was restless and diaphoretic and still short of breath. Her blood pressure was 67/35 and small to moderate vaginal loss was noted, with about 20 ml of dilute urine. The obstetrician was again requested to assess the mother.

The obstetrician reassessed the mother between 0400 and 0430 hours. The situation was discussed with the mother's regular obstetrician on the phone. The on call obstetrician was advised by the mother's obstetrician to transfuse two units of blood and request an abdominal ultrasound in the morning. By 0430 hours, the nurses were having difficulty recording a blood pressure, assessing O2 saturation and starting a second IV for blood. The previously ordered complete blood count had been taken with difficulty at 0425 hours. The obstetrician on call performed a bedside ultrasound and diagnosed intra abdominal hemorrhage. According to the obstetrician's notes, more blood was ordered and the other obstetrician, the operating room nurses and anaesthetist were called in.

The nursing notes suggest a somewhat different timing. They indicate that the obstetrician was called again and again and did see the mother at 0500 hours, but gave no new orders. They then note that the mother was combative at 0500 hours and that they had still not been able to start another IV. At 0517 hours, the mother is noted to be unresponsive and the nurses note that the obstetrician had left the ward. At this point, the nurses called a code blue and paged the obstetrician to return. The anaesthetist notes being called by the obstetrician at 0447 hours and being advised that they were planning to take the patient back to the OR because of internal bleeding. The nurses note his arrival at 0519 hours and the arrival of the second obstetrician at 0535 hours, but do not indicate that they were aware of the plan to return the mother to surgery.

The anaesthetist intubated the mother on his arrival in the labour room recovery area, placed a central line and commenced blood and crystalloid. About 25 minutes later, the mother sustained a cardiac arrest and full code was carried out in the labour room with chest compressions, two defibrillations and Epinephrine, Atropine and Amiodarone were given, as well as Vasopressin. The mother received three units of red blood cells, three litres of normal saline and 500 ml of Pentaspan through the central line. She was transferred to the operating room with cardiopulmonary resuscitation in process by the anaesthetist.

The abdomen was opened at 0603 hours and 3-4 litres of blood were evacuated. The mother regained a central pulse at this point and cardiopulmonary resuscitation was stopped for a few minutes between 0620 and 0632 hours. However, the pulse was lost again and she required chest compressions at 0632 to 0642 hours, 0715 to 0727 hours, 0803 to 0815 hours, and 0846 to 0902 hours. The mother's usual obstetrician performed the surgery with the on call obstetrician assisting. The surgeon identified an area of bleeding on the right side of the uterine incision. With further exploration, it appeared that there was "some extension down in the right angle of the incision" so a subtotal hysterectomy was carried out. He felt that the mother had DIC and attempted

internal iliac artery ligation, but this was abandoned as the mother arrested again. The abdomen was closed, but had to be reopened as it was clearly filling with blood again.

A second anaesthetist joined the team and a haematologist was consulted over the phone. At this point, the mother had received 13 units of cross-matched blood and 10 units of O-negative blood. The hospital's blood bank was now empty and more blood was being moved from another institution. The haematologist ordered cryoprecipitate. At the re-laparotomy, the incision was extended into the upper abdomen which was also explored. No specific bleeding site could be identified and the abdomen was packed with four large sponges and again closed. During these surgeries, the mother received a total of 23 units of red blood cells, 16 units of FFP, 10 units of platelets and 1 unit of cryoprecipitate.

The mother was transferred to the intensive care unit at 1015 hours. She remained very unstable and comatose with DIC multi-organ failure. She arrested at 1055 hours and again at 0633 hours the following morning. She could not be resuscitated from the last cardiac arrest.

Post mortem

Pathologic examination of the hysterectomy specimen revealed widespread foci of myometrial haemorrhage with no evidence of retained products of conception or placenta.

The post mortem exam revealed recent subtotal hysterectomy with haemorrhagic surgical bed and multiple sutures in situ. No site of origin of the hemorrhage was identified. No underlying cause of the DIC (such as amniotic fluid embolism) was identified.

Cause of Death: Intra-abdominal bleeding and DIC due complications of Caesarean section.

Discussion

The mother died of massive post partum hemorrhage after Caesarean section. It is not possible to determine with certainty the cause of the bleeding, but the obstetricians did identify a possible bleeding site and possible extension of the uterine incision at laparotomy. At this time, however, the mother already had a coagulopathy, thus making such a diagnosis, difficult.

The signs of internal bleeding were present for two hours prior to the patient's first cardiac arrest and return to the operating room. By the time they were recognized and acted upon, she had lost a massive amount of blood and had DIC and multi-organ failure.

Recommendations

1. All health care providers are reminded to review the patients' vital signs for symptoms of postoperative blood loss and respond to the nurses concerns with other investigations to confirm the problem or reassure the other caregivers.
2. The nurses of this hospital should be aware of the options they have for calling the department chief or chief of staff if they are concerned about a patient and feel the attending physician is not acting appropriately.
3. The hospital is requested to do a Quality of care review of his case.

Case: 2009-M-13

History

The patient was a 35 year old gravida 2 who died six days post partum. The recent pregnancy was complicated by pre-existing hypertension with preeclampsia, gestational diabetes (for which she was on insulin) and morbid obesity. She also had asthma and one previous Caesarean section.

The patient received shared prenatal care by her family doctor and an obstetrician. All routine investigations were normal. She declined genetics investigations.

She was morbidly obese with a body mass index (BMI) over 50 at delivery. She weighed 149 kg (327 pounds) at 15 weeks and increased another 10 kg to 159 kg (349 pounds), by 34 weeks.

At 28 weeks, a 50 gram GCT was 11.0 mmol/L. This was followed by 75 gram tolerance test which showed the fasting sugar and one hour PC to be abnormal. She was referred to an endocrinologist and to the Diabetes Education Centre. She was diagnosed with gestational and possible type 2 diabetes. She was started on insulin at 30.5 weeks.

Her blood pressure was elevated from 20 weeks, but was also quite labile. At 32 weeks, she was admitted to the hospital for control of both her diabetes and blood pressure. Labetalol was started orally and insulin increased to three times a day.

The patient was home five days and re-admitted at 34.5 weeks, again due to blood pressure with new symptoms of headache and scotoma. Her Labetalol was increased and she was monitored with urine collections for protein, platelet, coagulation and liver enzymes tests. All of the tests were normal. The patient's sugars were well controlled at 5-7 mmol/L. Her baby was monitored with daily NST and twice weekly BPPs, all of which were normal. Prophylactic heparin was given antepartum.

The patient was booked for repeat Caesarean at 38 weeks gestation, but by 37 weeks, her blood pressures had again increased, so a Caesarean section was undertaken at that time. She had a spinal anaesthetic and a healthy male infant was delivered weighing 4.1 kg (9 lbs). Post-operatively, she developed a small wound haematoma which was drained and the incision was irrigated and packed daily. She was not continued on either heparin or insulin post partum. She continued to require some

Labetalol. The patient developed a superficial phlebitis which was treated with ibuprofen.

On the sixth day post partum, the patient was expected to be discharged with home care for her wound if the baby was ready. She had been to the nursery to feed her baby and returned to her room at 0300 hours. She told the nurses she felt well, but asked for some Percocet. The nurses brought her the medication and checked her vital signs at this time. Her blood pressure was 138/85, temperature 36.8C, pulse 89, respirations 20 and oxygen saturation 100% on room air.

At 0415 hours, the patient rang the bell for the nurses as she was having trouble breathing. She felt she was having an asthma attack and asked for her puffers from her purse. The nurse helped her use the puffers, but they did not help. The patient was given oxygen and Ventolin by mask and the obstetrician was called.

When the patient's oxygen saturations decreased into the 40's, a "code blue" was called. The code team arrived and cardiac arrest was diagnosed. CPR was commenced and IV access was established. The patient was intubated with some difficulty on the third attempt and hand ventilated. She was given four doses of Epinephrine, two doses of Atropine, one dose of sodium bicarbonate and a dopamine drip was started. Despite these efforts, she remained asystolic. Lack of cardiac contractility was confirmed with emergency echocardiography and the code was concluded after 32 minutes. The patient was pronounced dead.

Post Mortem

An autopsy was conducted and the cause of death was determined to be hypertensive heart disease.

The significant findings at autopsy were:

- Bilateral Pleural Effusions.
- Lungs wet and congested.
- Foamy secretion – trachea.
- Heart - cardiomegaly, 600 g.
- Liver – 3090 g (soft, tissue gas).
- Splenomegaly – 850g

Discussion

The patient had a peripartum cardiomyopathy. This is a rare condition that can result in sudden cardiac death. She had risk factors for peripartum cardiomyopathy that included: advanced maternal age, chronic hypertension and preeclampsia. It is

however, unusual that she had no symptoms and no signs of cardiac problems as of 90 minutes prior to her death. The patient died of sudden cardiac collapse, without warning. Given the lack of symptoms or signs, it is not likely that anything could have changed the outcome.

Recommendations

None.

Case: 2009-M-14

History

The patient was a 21 year old female with a known cranio-facial syndrome, as well as congenital heart disease. She had a cranial reconstruction surgery as a child and had been followed in the Pediatric Cardiology Service in Toronto. She had bioprosthetic mitral and aortic valve replacements in 2004 and recurrent atrial ectopic and junctional tachyarrhythmias. She was mentally challenged with a learning disability. Since the age of 5, she was under the care of the Children's Aid Society (CAS). As an adult, she was in a common-law relationship.

The patient was followed by the Maternal Foetal Medicine service and two cardiologists at a large hospital. She was also seen by Internal Medicine, Genetics, and Pediatric Cardiology. She was constantly non compliant with medical follow up and taking her cardiac medications, despite constant aggressive input from physicians and social services. Social work in the community and in hospital, Public Health, and CAS were involved and tried to support and encourage her compliance with prenatal care.

The patient was admitted on June 5, 2008 for 24 hours with atrial tachycardia. She was found to have increased liver enzymes and jaundice. GI medicine saw her and outpatient follow up arranged to try and reach a specific diagnosis.

The patient was admitted to hospital on June 15, 2008 with a complaint of headache, chest pain and upper abdominal pain. At the time of admission, she was approximately 33.5 weeks gestation. Maternal tachycardia was treated with B-blockers. She experienced some shortness of breath overnight, but her oxygen saturation and vital signs remained stable. On nursing rounds at 0630 hours, she was found to have foetal bradycardia with foetal heart rate determination in the 60's. An emergency Caesarean section was arranged. There was delay in getting the patient's consent. Anesthesia was concerned about her airway, so spinal anesthesia was rapidly performed. Her blood pressure was liable and then difficult to maintain towards the end of the Caesarean section. Immediately post-operatively, the patient suffered a cardiac arrest. Attempted resuscitation recovered a heart rate and she was transferred to the intensive care unit for subsequent management. The patient was determined to be brain dead and all further resuscitative efforts were not successful.

Post Mortem

At autopsy, multiple congenital abnormalities of the head and face were noted. There were multiple scars of the scalp and the scalp was noted to be very adherent to the skull. The initial autopsy demonstrated the presence of near occlusive thrombus involving the prosthetic mitral and aortic heart valves.

Microscopic examination of the various tissues obtained at the time of the general autopsy demonstrated the presence of hemorrhage into the adrenal glands, a micro-abscess of the spleen, clusters of Gram positive bacteria within the liver parenchyma, micro-abscess in the kidneys and alveolar edema of the lungs.

The cause of death was attributed to sepsis due to infective endocarditis involving both prosthetic mitral and aortic valves. The micro-abscesses in the various other tissues including the brain reflect the disseminated nature of this process.

The cardiac pathology consultation also found prosthetic valve endocarditis involving both the mitral and aortic valves. There were abundant Gram positive organisms identified. There was a previous myocardial infarction and focal eccentric atheromatous stenosis of the left anterior descending coronary artery. The predominant feature on histologic examination of the organs was disseminated sepsis with a Gram positive organism. This was evident in the brain, heart, liver, spleen, and kidneys.

Discussion

This is a case of a maternal death following emergency Caesarean Section for foetal bradycardia. The patient had presented less than 24 hours prior with symptoms that were being investigated appropriately. The patient had significant medical risk factors and multiple care providers were diligent in attempting to get her to understand her risks and to have her comply with medical care. Multiple social services were involved to this end as well. Despite this, the patient was non compliant with medical care and treatment. Autopsy revealed the unexpected finding of infective endocarditis with disseminated septic foci.

Recommendations

None

Case: 2009-M-16

History

The deceased was a 36 year old G1P0 with an EDD of November 14, 2008. This was an Intrauterine Insemination (IUI) pregnancy. The medical records provided did not note the indication for infertility treatment. An ultrasound on April 14, 2008 confirmed a viable singleton intrauterine pregnancy of 7 weeks 3 days gestation. Routine prenatal laboratory investigations, IPS and second trimester ultrasound were normal. The patient

was Rh negative and was given Rh immune globulin at 28 weeks gestation. The GCT was elevated and a Glucose Tolerance Test (GTT) was diagnostic for gestational diabetes. The patient was placed on a diabetic diet and her blood sugars were well controlled without insulin. An ultrasound on October 6, 2008 at 34 weeks gestation confirmed normal foetal growth and amniotic fluid volume. BPP was 8/8.

The patient's medical history was significant for obesity and hypertension. Her pre-pregnancy weight was 219 pounds (99.34 kg) and she gained 24 pounds (10.89 kg) during the pregnancy. She was on Labetalol 200 mg, twice daily, at the start of the pregnancy. Her blood pressure was well-controlled until October 22 at 36 weeks 5 days, when her blood pressure at a scheduled prenatal visit was 130/90 with 1+ urine protein. Pregnancy Induced Hypertension blood work was ordered and was normal. On November 5, at 38 weeks 5 days, her blood pressure was 160/100 with no proteinuria.

The patient's family history was significant for stroke; the patient's mother had a stroke at age 46 from which she recovered. The patient had a brother with a cognitive developmental delay.

The patient was admitted to hospital on November 6, 2008 at 38 weeks 6 days, for worsening hypertension.

At the time of admission, the patient's cervix was 1 cm dilated and thick, with the vertex at sp-3. She received three doses of Cervidil without labour becoming established by the morning of November 8. She went on to delivery by Caesarean section under spinal anaesthesia at 1029 hours for a healthy male infant. Apgars were 9 and 9 at one and five minutes. The birth weight was not recorded on the Labour and Delivery Summary.

During the immediate post partum course in hospital, the patient's blood pressure remained elevated and was associated with intermittent headache. On November 12, the Labetalol was increased to 300 mg, three times daily, for a blood pressure of 177/96. Adalat XL 30 mg daily was added on November 14 for a blood pressure of 161/94. On November 15, her blood pressure was 150/88. She complained of a frontal headache that morning, but was otherwise well. She was discharged home on Labetalol 300 mg, three times daily, and Adalat XL 30mg daily.

The patient attended the breast feeding clinic on November 20 and when leaving the hospital, she developed blurring of her vision and a right-sided headache. She presented to the Emergency Department at 1218 hours. She had transient spells of left-sided facial droop and slurred speech. Her blood pressure was 181/104. CT of the brain was normal. A neurological consultation was obtained. Findings on neurological examination were inconsistent. A diagnosis was not forthcoming at that time and she was admitted to hospital and observed.

At 1600 hours, she again had some left-sided arm weakness and drowsiness. She was re-assessed by the neurologist and again findings were inconsistent as the left arm movements had improved. She continued to have left-sided symptoms and requested analgesia for headache during the night and early on the morning of November 21. A repeat CT scan at 0820 hours showed extensive infarction of the right fronto-parietal, temporal and occipital lobes. There was marked crowding of the sulci with slight mass

effect on the ipsilateral ventricle and slight midline shift. There was also slight effacement on the basal cisterns relating to increased intracranial pressure. There was no intracerebral hemorrhage.

A consultation was obtained with the Neurosurgical Unit of a larger hospital through Criti-Call. Arrangements were made to transfer the patient to the larger hospital. The patient was intubated prior to transfer for airway protection.

The patient was admitted to the receiving hospital at 1310 hours. A repeat CT scan showed increasing mass effect and brain swelling. An emergency decompressive craniotomy was performed with placement of a subdural drain. Her neurological status did not improve. After discussing the situation with the family, the decision was made to withdraw ventilator support and provide comfort measures only. The patient died on November 24.

Post Mortem

An autopsy was not performed even though the discharge summary from the hospital indicates that one was done. The dictated discharge summary letter by the ICU physician indicates that the coroner was notified at the time of death. Documents however, indicate that the coroner's office was not contacted until May 20, 2009 – several months after the death.

Discussion

The patient died from a massive ischemic stroke of the right hemisphere 16 days post partum. Her pregnancy had been complicated by chronic hypertension which worsened towards the end of the pregnancy, in the absence of super-imposed pre-eclampsia leading to induction of labour at 38 weeks 6 days. Her blood pressure continued to be elevated post partum requiring the dose of Labetalol be increased and the addition of Adalat. With medication, her blood pressure was for the most part controlled. In the absence of an autopsy, the underlying reason for the stroke cannot be determined. The possibilities of a thrombophilia or carotid artery disease were considered by the neurosurgical team, but investigations were not done presumably due to the rapid deterioration in her clinical course.

Recommendations

1. The Chief Coroner's office should investigate the circumstances relating to the delayed reporting of this maternal death and the reason why an autopsy was not performed.

History

The deceased was a 36 year old G1P0 with an EDC of November 11, 2008. She had recently moved from China and the first prenatal care she received was when she arrived in Canada, at 24 weeks gestation, with the obstetrician who subsequently cared for her in labour and delivery. The patient was followed regularly and her last recorded prenatal visit was on October 30, at 38 weeks gestation.

The patient was diagnosed with gestational diabetes and subsequently followed in the diabetic clinic at Hospital A. She maintained normal sugars on diet therapy alone. Routine prenatal blood testing was remarkable in that she was positive for Hepatitis B surface antigen. There were no recorded abnormalities in the patient's previous medical history. However, the patient did not speak English well and this may have had an impact on the accuracy of the health history obtained.

The patient was admitted to hospital on the morning of November 10 with a history of having had ruptured membranes approximately ten hours earlier. She was Group B streptococcus (GBS) negative and was in labour at that time. There was slow progress, so three hours later, she had syntocinon augmentation. She was fully dilated by 1620 hours and had an effective epidural anesthetic for analgesia. Continuous foetal monitoring remained normal. As there was no progress with pushing in the second stage, at 2115 hours and approximately five hours after being fully dilated, the option of Caesarean section was discussed and decided upon. The foetal heart was stable and the patient was afebrile. A Caesarean section was planned for approximately an hour later when the anesthetist was available.

A 5 pound 4 ounce infant was born by low segment Caesarean section at 2308 hours. There was thick meconium at the time of delivery. Neonatal staff was available. The baby had Apgars of 2, 5 and 8 at one, five and ten minutes. The baby was resuscitated, given CPAP and taken to the NICU.

During labour, vital signs were normal and temperatures ranged from 37.1 to 37.5. A catheter was inserted at 2130 hours, approximately one hour before the delivery by Caesarean and "hematuria noted". Vital signs during the operative procedure were unremarkable.

Overnight until noon the next day, the patient's temperature remained low at less than 35 degrees. The vitals were otherwise unremarkable. Approximately three hours after the Caesarean, the abdominal dressing revealed blood oozing through and required reinforcement. There was normal post partum lochia.

At 0800 hours the following morning, the patient was noted to be sleeping. At 1230 hours, the attending nurse commented on the decreased temperature and the fact that no report had been given from the previous night shift. At that time, the patient's temperature was 34.3 C, BP 115/81 and pulse 150. The attending obstetrician was made aware. The RN applied warm blankets and warm IV solutions were started. The pulse decreased to 103 by 1300 hours, but remained 100 for the day. The patient's

temperature rose to 35.5 C by 1845 hours. The attending physician was asked to see the patient at 1915 hours due to persistent oozing through the dressing. At that time, the hemoglobin was 91, down from 117 on admission and platelets were 77 000, down from 169 000 on admission. WBC was 31.6, up from 11.3. Liver function tests showed significantly elevated enzymes including bilirubin, compatible with the obvious jaundice that she was now displaying.

Blood tests were completed. The internist on call was consulted and attended the patient shortly after midnight on November 12, 2009. The patient's status was reviewed and it was noted that she had a low blood pressure at 65/38, increased pulse at 160, drowsiness, jaundice and significantly abnormal blood findings. The ICU consultant saw the patient and immediately transferred her to the intensive care unit. A central line was instituted and the patient required fluids, blood products and pressor agents in order to achieve some degree of hemodynamic stability. Over the next 24 hours, the patient seemed to stabilize, although her LFT's remained elevated as did her ammonia, PT and PTT. By the afternoon of the November 13, the patient became less responsive and a CT of the head showed what appeared to be cerebral edema. Arrangements were made for transfer to a tertiary centre for hepatic support and consideration of a transplant.

The patient was intubated for transport to the tertiary centre. At that time, the etiology of her hepatic failure was unclear. HELLP or TTP had been entertained, although thought to be unlikely with the given presentation.

The patient was seen on the evening of November 13 by the liver transplant team at Hospital B. At that time, the working diagnosis was either HELLP or acute fatty liver with associated DIC. It was felt that the patient had a reasonable chance of recovery and did not fulfill criteria for transplant. The patient remained intubated and ventilated and was empirically treated with broad spectrum antibiotics. Further consultations were done with hematology and obstetrical medicine. The continuing consensus was that this was likely a severe case of HELLP with associated liver failure. Hematology continued to follow the patient and she was tested for red cell fragments for possible TTP. Plasmapheresis was considered as a management strategy. Repeated testing did not confirm this possibility. On November 15, the patient required further blood product replacements due to increasing INR, decreasing platelets and decreasing hemoglobin. A transjugular liver biopsy was done in preparation for a possible liver transplant.

The biopsy showed "extensive hepatocyte damage", but there was no mention of underlying pathology. The patient was listed as a "status 4F" for transplant. On the morning of November 16, the patient further deteriorated and became less responsive with an acute drop of blood pressure that required pressor drugs. The patient was felt to have lost all brain stem activity. A CT scan of the head demonstrated significant brain edema and she was declared brain dead at 1520 hours on November 16. The patient died the following day, on November 17, 2008.

Post Mortem

The post mortem examination revealed jaundice and submassive hepatic necrosis. No underlying cause for the hepatic necrosis could be determined. It was not felt that these findings, or those of the liver biopsy, were compatible with HELLP or fatty liver. Other findings included early hypoxic-ischemic encephalopathy. There was no evidence of retained products of conception, endometritis, injury to uterus, cervix or vagina, or amniotic fluid embolism.

The pathological cause of death was given as, "hypoxic ischemic encephalopathy due to submassive hepatic necrosis of unknown etiology after a recent Caesarean section at term."

Discussion

This 36 year old patient had recently arrived in Canada from China at approximately 24 weeks gestation. She appeared to have an unremarkable pregnancy until shortly after her delivery. Neonatal blood testing revealed positive hepatitis B surface antigen. She was diagnosed with gestational diabetes and placed on the required diet treatment.

The patient appeared to be well prior to going for Caesarean section. On catheterization prior to the section, she was noted to have hematuria. In the early post operative time, she developed oozing from her incision. The patient was somewhat hypothermic during the night after delivery. When assessed approximately 20 hours after delivery, she was noted to be jaundiced. There was a significant change in her blood testing which revealed thrombocytopenia, coagulopathy and significantly abnormal liver function. She had expedient consultation with internal medicine, then critical care, and was quickly moved to the intensive care unit. Over the next 24 hours, the patient seemed to stabilize, however she became less responsive and required intubation and transfer to a tertiary center for consideration of hepatic support and possible transplant.

At Hospital B, the patient remained intubated and on a ventilator in the intensive care unit. She had multiple consults, including the liver transplant team, hematology and obstetrical medicine. The working diagnosis was of a pregnancy related HELLP or acute fatty liver with the possibility of recovery. At the point of being put on the liver transplant list (approximately six days after delivery and three days after transfer to the tertiary center), the patient further deteriorated and was declared brain dead.

The final pathology confirms extensive liver damage, though its etiology is unknown and not compatible with HELLP. The illness leading to this death was sudden, unexpected, severe and with no hope of recovery.

Recommendations

None

History

The deceased was a 29 year old gravida 2 at 38.5 weeks gestation who was admitted for repeat Caesarean section. She had no past medical or surgical history, aside from one prior Caesarean for placenta previa. Prenatal care was provided by her family doctor and obstetrician and there were no concerns noted. She was a non-smoker with an allergy to codeine. She did not use any medications or street drugs.

The patient was well upon presentation for her surgery. After the spinal anaesthetic was placed, she developed hypotension and bradycardia. She was treated for this by the anaesthesiologist and then developed a tachycardia with ventricular tachycardia and ventricular fibrillation which was quickly corrected. However, because of foetal concerns with persistent hypotension, the Caesarean was completed urgently and the paediatrician was called to attend the birth. The baby was female, healthy and responded well to minimal resuscitation.

After the surgery was completed, the patient developed profound shortness of breath and was hypoxic. She was given general anaesthesia and was intubated. At this point, marked pulmonary edema was noted and an amniotic fluid embolism was suspected. The intensive care physician was consulted and attended in the operating room. After intubation, gross pulmonary edema was still apparent. The patient had a wide complex bradycardia and cardiac arrest while still in the operating room. CPR was performed and she was treated with epinephrine, atropine, and bicarbonate. A pulse was restored, however she remained hypotensive and was treated with levophed and more epinephrine. She was stabilized and moved to the ICU where she remained very unstable with hypotension, and hypoxemia.

A cardiologist was consulted. An echocardiogram revealed severe left ventricular dysfunction with an ejection fraction of 15%. The patient was receiving massive doses of inotropes and vasopressors and frequently required CPR. Arrangements were made for transfer to another hospital for extracorporeal membrane oxygenation (ECMO). The patient sustained another cardiac arrest during the transfer and was admitted to the cardiovascular ICU in very unstable condition. ECMO cannulas were placed, but the patient developed progressive refractory shock and passed away about 8 hours after the birth of her baby girl.

Post Mortem

Summary:

- Acute pulmonary edema and pulmonary hemorrhage and ARDs changes;
- Post partum uterus with secondary changes from Caesarean section.

Cause of death was acute cardiovascular collapse, probably from amniotic fluid embolism.

Discussion

Amniotic fluid embolism is a rare complication of pregnancy. Even with intensive therapy there is an extremely high mortality rate.

Recommendations:

None

Case: 2009-M-19

History

The deceased was a 26 year old G1P0. On October 26, 2009, at 38 weeks gestation, she arrived at hospital with term pre-labour rupture of membranes (term PROM). She had an uncomplicated antenatal course. She spontaneously ruptured her membranes at 0500 hours on October 26, 2009 for clear fluid. She was GBS positive and was induced with Oxytocin as she was only contracting mildly with a posterior uneffaced cervix.

The patient was started on IV penicillin for GBS prophylaxis. The IV Oxytocin was started and she was 2 cm and fully effaced by 1645 hours. Foetal heart tracing was normal. She was 3-4 cm dilated by 2055 hours and was offered pain relief options to facilitate labour. She progressed to 5 cm by 2220 hours when Oxytocin was discontinued for a deep variable deceleration. The patient had blood drawn at that time to be cross-matched for two units of blood. The Oxytocin was restarted and at 0140 hours on October 27, 2009, she was examined again and found to be unchanged. The pain relief options were reviewed and she elected to have morphine and gravol. She was fully dilated by 0330 hours. The patient pushed for 1.5 hours when a mediolateral episiotomy was undertaken for resistant tissues. The baby delivered at 0357 hours.

The patient went on to have a post partum hemorrhage which was initially managed by the delivering doctor. She was given two IV bolus' of Oxytocin and four doses of hemabate over the next 1.5 hours at 0519, 0540, 0555 and 0625 hours. At 0540 hours, another doctor was called to assist as the initial two units of blood were transfused for hypotension and ongoing blood loss. The patient received another two units of blood at 0635 and 0650 hours. A general surgery consultation was undertaken for exploration under anesthesia to assess for other causes of the bleeding. The patient was transferred to the OR at 0705 hours. Bleeding was noted from a cervical laceration and from the episiotomy site. The cervix was sutured and the episiotomy site was re-sutured. The bleeding was thought to be under control.

The patient was transferred from the OR and developed cardiac arrest. A code was called at 0821 hours. Bleeding resumed and blood, fresh frozen plasma and albumin were all utilized. There were further attempts to control the bleeding with additional doses of hemabate and misoprostyl. The patient was taken back to the OR at 1030

hours for a Bakri balloon placement in an attempt to control the bleeding. The patient ultimately developed resistant DIC and was pronounced dead at 1251 hours, despite aggressive resuscitation measures.

Post Mortem

Postmortem examination was consistent with massive post partum hemorrhage secondary to uterine atony.

Discussion

The deceased developed a post partum hemorrhage after a spontaneous vaginal delivery following induction with Oxytocin for term PROM. Post partum hemorrhage affects up to 5% of deliveries and remains a cause of maternal mortality. The primary cause of bleeding in this case appeared to be uterine atony, although note was made of retained membranes and a cervical laceration.

The patient, as expected in a young healthy woman, compensated for her blood loss until she decompensated from hypovolemic shock with a BP of 61/41. It was at this point that aggressive fluid resuscitation was begun. She had two IV's started and she received blood products. She initially received two boluses of IV Oxytocin, then hemabate thirty minutes into the post partum hemorrhage . The patient was not explored for other sources of bleeding or retained products until three hours after delivery as uterine atony was assumed to be the mechanism. The patient's rapid and ongoing blood loss contributed to her development of DIC which ultimately resulted in her death, despite aggressive use of blood products and attempts to control bleeding with a Bakri balloon.

It is not clear whether embolism or hysterectomy were considered. It is also difficult to assess whether more aggressive initial or early resuscitation, and more intense use of hemabate or misoprostyl, would have changed the outcome. There may have been a delay in exploration because the patient initially seemed to respond quickly to uterine massage and medications and therefore it seemed less likely that lacerations were the cause of the blood loss.

Recommendations

1. Obstetrical care providers are reminded that post partum hemorrhage complicates up to 5% of deliveries and remains a cause of maternal morbidity and mortality. All obstetrical care providers at the hospital should consider review of the ALARM course and develop protocols dealing with obstetrical emergencies.
2. Obstetrical care providers are reminded that otherwise healthy women can tolerate a significant blood loss prior to becoming symptomatic and aggressive attention to the ABC's is required.

Case: 2009-M-20

History

The deceased was a 24 year old G1P0 with an EDD of January 26, 2009. This was an unplanned pregnancy. Routine prenatal laboratory investigations, IPS and ultrasounds were normal. GCT was normal and she was GBS positive.

The patient's medical history was significant for scleroderma and pulmonary fibrosis. She was under the care of a rheumatologist and her last pulmonary function studies were done in 2007. At that time, her FEV1 was 64% and TLC 62%. She had started Plaquenil in early 2008, but this was discontinued in May 2008 because of the pregnancy. She was seen by her rheumatologist during the pregnancy. The patient was on Prednisone 10 mg daily and the scleroderma was stable throughout the pregnancy.

Because of her connective tissue disorder, her obstetrician referred her to a specialized Medical Disorders Clinic at Hospital A. On August 21 at 17 weeks 4 days, she complained of swelling in her legs. An echocardiogram was normal. The antenatal course was otherwise uneventful. She had an anaesthetic consultation on December 10.

Course in Labour and Delivery

On December 29, at 36 weeks gestation, membranes ruptured spontaneously for meconium-stained fluid. The patient was given intravenous Penicillin G for positive GBS status. She became uncomfortable with the contractions and an epidural was started. She subsequently required oxytocin augmentation and became fully dilated at 1330 hours with the vertex at spines +3. During the second stage, baseline foetal heart rate went up to 160-170's. The patient remained afebrile and the meconium was darker. The decision was made to expedite delivery using vacuum extraction because of the atypical tracing. The patient was delivered of a 4 lb. 4 oz. female infant with Apgars of 9 and 9 at one and five minutes over a right mediolateral episiotomy and a third degree tear. Her post partum course was uneventful and she was continued on Prednisone 10 mg daily.

The patient was seen at the Special Pregnancy Program at Hospital A for a post partum assessment on February 12. She complained of frequent headaches, back and joint pain. She was also experiencing a feeling of chest pain described as a pressure feeling. She had seen her rheumatologist the week before, although records from this visit were not noted on file.

The patient presented to the Emergency Department at Hospital B on April 20 at 1721 hours with chest pain and shortness of breath. She had been experiencing shortness of breath and orthopnea for a month which had worsened over three days prior to coming to the Emergency Department. A chest x-ray showed diffuse lung infiltrates and pleural effusions. An ECG confirmed sinus tachycardia. Internal medicine was consulted. A CT angiogram showed no evidence of a pulmonary embolus.

Laboratory investigations showed a Hb 91gm, WBC 10.4, platelets 659,000, potassium 5.1, creatinine 88, CK 292 and troponin 0.11. The patient was hypoglycaemic. She was given Kayexelate, D10W, solucortef and IV antibiotics.

The patient was admitted to the Intensive Care Unit and placed on 100% O₂ and given Lasix 40 mg IV at 0600 hours and a Foley catheter was inserted. Repeat laboratory investigations showed potassium 6.0, troponin 1.5 and lactic acid 9.6. Liver enzymes were elevated and serum creatinine increased. Cardiology, nephrology and rheumatology were consulted.

At 0800 hours, the patient became bradycardic, hemodynamically unstable and then arrested. A "Code Blue" was called. She was resuscitated with Atropine, adrenalin, bicarbonate and calcium.

A hemodialysis access line was inserted and arterial blood gases showed a pH of 7.19, pCO₂ 34, pO₂ 139 and bicarbonate 13 on 100% O₂. Placement of a Swan-Ganz catheter was attempted, but was unsuccessful. Placement was then attempted under fluoroscopy which showed that the catheter could not be carried through into the pulmonary artery due to the very low cardiac output. An echocardiogram at 0905 hours showed a grade III-IV left ventricular dysfunction and a small pericardial effusion. The patient had recurrent bradyarrhythmias which were again treated with Atropine, adrenalin, calcium and bicarbonate.

At 1040 hours, the patient had a recurrent event and could not be resuscitated. Her husband was present and requested termination of the resuscitative efforts and she was pronounced dead at 1055 hours.

Post Mortem

A post mortem examination was not performed.

Discussion

The deceased died from heart failure almost four months after delivery of her first pregnancy. Her medical history was significant for scleroderma and the major manifestations were pulmonary fibrosis, arthritis and skin changes. She had no previous history of heart disease and an echocardiogram done in the second trimester was normal. Her pregnancy and delivery were relatively uneventful and post partum, she continued on Prednisone. At her post partum visit on February 12, she complained of headaches, back and joint pain and a pressure feeling in the chest. It was noted that she had been seen by her rheumatologist the week before. The medical records provided do not indicate if there was any investigations or follow-up arranged at that time.

The patient presented to Hospital B Emergency Department where she was seen in triage at 1721 hours on April 20 with shortness of breath and chest pain. She was found

to be alert. Her pulse was 137 bpm, RR 24 and BP 114/87. SpO2 was 95%. She was assigned a triage Level 2. The patient was assessed at 2025 hours by the Emergency Room nurse and her pulse was 143 bpm, RR 40, BP 123/96. Initial blood tests were drawn at 2041 hours. The Emergency Room record appears to indicate that the patient wasn't seen by the Emergency Room physician until 2300 hours. A subsequent chest x-ray showed pleural effusions and cardiomegaly. An ECG at 0032 hours showed sinus tachycardia with low voltage QRS. A referral was then made to internal medicine at 0050 hours. A CT angiogram was done at 0140 hours to rule out a pulmonary embolus. She was admitted to the ICU at 0350 hours where she was treated for fluid overload and hyperkalemia. Laboratory investigations over the course of the early morning hours showed developing renal and hepatic failure. A Foley catheter was inserted and the patient was given Lasix 40 mg IV at 0600 hours. She arrested at 0800 hours on April 21 and was resuscitated. An echocardiogram showed severe left ventricular dysfunction. Attempts were made to perform emergency dialysis, but she had recurrent cardiac events from which she ultimately could not be resuscitated. The question was raised as to whether peripartum cardiomyopathy was the underlying cause.

Four criteria are needed to meet the definition of peripartum cardiomyopathy:

1. development of cardiac failure during the last month of pregnancy or within five months of delivery;
2. absence of an identifiable cause for the cardiac failure;
3. absence of recognizable heart disease prior to the last month of pregnancy;
4. left ventricular systolic dysfunction.

The presentation made by this patient was consistent with the diagnosis of peripartum cardiomyopathy. The differential diagnosis shortly after admission to the ICU was pulmonary infection or fluid overload from an underlying cardiomyopathy from her rheumatological condition. She was treated for fluid overload with one dose of Lasix, but there was no diuresis. Her creatinine rose from 88 when first seen in the Emergency Room, to 134 at 0450 hours. Her serum potassium on admission was 5.0 and 6.1 at 0450 hours. With the persisting fluid overload and electrolyte abnormalities, she continued to deteriorate until she arrested at 0800 hours. After successful resuscitation, lines were placed for haemodialysis, but she suffered from recurrent arrests before haemodialysis could be carried out.

Recommendations

1. Health care providers are reminded that peripartum cardiomyopathy can occur up to five months post partum.
2. The Chief of Emergency Medicine at Hospital B should review this case particularly with regard to triaging and timelines for assessment.

Case Summaries: Neonatal Deaths

Case: 2009-N-1

History

The mother of the deceased was a 29 year old G2P0 who came to the midwifery practice on December 27, 2007 at 34 weeks gestation. Prior to moving to Canada, she had been receiving prenatal care in China. Her EDD was February 1, 2008. Her first pregnancy ended as a result of an ectopic pregnancy. Her past medical history was non-contributory.

The patient's routine prenatal laboratory investigations were normal and she was HIV negative. Her GCT was normal and GBS was negative. During one of her prenatal visits with the midwife, a foetal heart rate irregularity was noted. A referral was made to a paediatric cardiologist on February 1. A foetal echocardiogram was normal and the cause was felt most likely to be intermittent premature beats.

At the prenatal visit of January 23 at 38 weeks, she reported having some bloody show, but no contractions.

Her antenatal visits were otherwise uneventful. Her blood pressure was normal throughout her pregnancy. Symphysis-fundal height measurements were up and down. An ultrasound on January 29 showed normal foetal growth and AFI with a BPP score of 8/8 and estimated foetal weight of 3642 gm. There was no evidence of a nuchal cord.

Course in Labour and Delivery

The patient attended the midwife's clinic on February 7 at 40 weeks 6 days gestation with spontaneous rupture of membranes occurring at 0630 hours that morning. Contractions were q4-5 minutes, lasting 40-50 seconds. The cervix was 6 cm, 70% effaced, vertex at spines -3 in the LOT position. Foetal heart rate was 145 bpm. She was sent to Labour and Delivery at the health centre.

She was assessed at 1300 hours and electronic foetal monitoring (EFM) was commenced. Variable decelerations were noted and an obstetrical consultation was obtained at 1315 hours. The patient was assessed by the obstetrician at 1320 hours. On review of the tracing, the baseline foetal heart rate was noted to be 140-150 bpm with intermittent decelerations down to 80 bpm lasting 40 seconds. A foetal scalp clip was applied. The cervix was noted to be 6-7 cm dilated with the vertex at spines -1. It was conveyed to the patient through her mother acting as translator, that if the decelerations were deep, persistent or recurrent with each contraction, that delivery by Caesarean section would have to be considered. The pattern continued with severe decelerations occurring, but not with each contraction. Position change, fluid bolus and oxygen were given.

The patient was re-assessed by the obstetrician at 1355 hours and found to be 9 cm dilated. She had the urge to push. At 1405 hours, she was moved to the delivery room

and anaesthesia was paged. The obstetrician reviewed the tracing at 1413 hours and an epidural was placed at 1430 hours. At 1455 hours, she was fully dilated and as the epidural was effective, she no longer had the urge to push. The contractions were noted to be more variable in strength and frequency. Her temperature at 1545 hours was 38.1° C and meconium was noted. The midwife requested that the obstetrician assess whether Oxytocin augmentation was indicated.

The patient was reassessed by the obstetrician at 1600 hours. Contractions were noted to be every 4 minutes. The vertex was at spines. The foetal heart rate tracing was noted to be as before. Oxytocin augmentation was initiated shortly after 1600 hours and care was transferred to the obstetrician.

The foetal heart rate pattern continued to show deep decelerations, but these were not consistent. The patient started pushing at 1700 hours. At 1720 hours, the midwife advised paediatrics that their attendance would be requested at the delivery because of a maternal temperature of 38.1° C and the presence of meconium.

The obstetrician attended at 1728 hours and noted at 1750 hours that the presenting part was visible with pushing. The obstetrician left the room at 1755 hours to attend to another patient. At 1810 hours, the foetal heart rate decelerated to 85 bpm. The midwife applied scalp stimulation and the foetal heart recovered. With continued pushing efforts, the patient went on to a spontaneous vaginal delivery at 1817 hours of a female infant weighing 3080 gm (6lb 12oz). The cord was around the neck once loosely. The baby was suctioned on the perineum for meconium.

The delivery was attended by the midwife and the obstetrician was present at 1818 hours. The baby was flat at birth with no respiratory effort and a heart rate of 50 bpm. Bagging was commenced and a "code pink" was called. The paediatrician arrived at 2 minutes after delivery and the baby was intubated at approximately 5 minutes. There was no response to resuscitative efforts including epinephrine via the endotracheal tube and at 15 minutes via an umbilical catheter. After consultation with the mother, support was withdrawn at approximately 30 minutes and the baby was pronounced at 1850 hours. Cord gases were obtained, but there was insufficient blood for analysis.

Post Mortem

Autopsy findings revealed growth parameters appropriate for gestational age. Examination of the lungs showed evidence of meconium aspiration. The heart was anatomically normal. There was cerebral congestion and edema with recent focal periventricular white matter infarcts.

The placenta showed meconium exposure effects with acute chorioamnionitis, chorionic vasculitis and acute funisitis. Blood cultures were negative. Placental cultures showed a scant growth of a coliform and mixed anaerobes. Lung culture grew *S. Viridans*.

The cause of death was hypoxic-ischemic injury. Additional or potentially contributing factors were acute chorioamnionitis with acute funisitis and chorionic vasculitis.

Discussion

This infant died as a result of a hypoxic-ischemic injury. The autopsy findings indicate that the injury occurred near the time of delivery. Such events could occur just prior to, or during labour. The intrapartum foetal heart rate tracings and cord gases can be useful, but are not necessarily conclusive, in trying to determine whether the event occurred during labour.

Unfortunately, although cord gases were obtained in this case, there was insufficient blood to run the analysis. On review of the foetal heart rate tracing, the pattern was abnormal as defined by the classification of intrapartum EFM tracings published by the SOGC in 2007(1) based on the occurrence of repetitive (>3) complicated variable decelerations. The recommended action is to review the overall clinical situation, obtain a scalp pH if appropriate/prepare for delivery. In this case, the care givers were cognizant of the concerning appearance of the tracing, but further testing of foetal well-being (scalp gases) or intervention to expedite delivery did not occur. Oxytocin augmentation without reassurance of foetal well-being was ill-advised. Foetal scalp stimulation was done during a deceleration by the midwife and the subsequent recovery to baseline may have been interpreted as being reassuring of foetal well-being. This was a misapplication of this test and as such, may have been falsely reassuring. Alternatively, it may have been felt that despite the decelerations, there was no significant deterioration in the tracing in terms of variability or baseline as is often seen with worsening foetal acidosis. (The baseline did increase to 160 bpm but this may have been due to the maternal temperature and by definition was not tachycardic). Otherwise, the rationale for the approach that was taken cannot be determined by this review, nor can it be determined that intervention leading to earlier delivery would have changed the outcome.

References

JOGC Volume 29, Number 9 September 2007 Supplement 4.

Recommendations

1. Obstetrical care providers are advised to become familiar with the current classification of intrapartum electronic foetal monitoring and the recommended actions in the setting of atypical and abnormal tracings.
2. Care providers are reminded that augmentation of labour in the presence of concerns regarding foetal well-being should be commenced with extreme caution.

Case: 2009-N-2

History

The mother of the deceased was a 35 year old G6P3A1. She was followed by her GP obstetrician for both prenatal and intrapartum care. She delivered a developmentally delayed girl in 1994 at 42 weeks and a boy in 1996 at 38 weeks. She subsequently had another girl at 40 weeks in 2006 with a new partner (same as current case). Her pregnancy was complicated by obesity with a Body Mass Index (BMI) greater than 30 (height 5'1" and weight 220 lbs). Her weight gain was 40 lbs. She had a LMP date of May 5 2007 with an EDD of February 9 2008 and a due date by ultrasound at 11+ weeks of February 15, 2008.

The patient underwent prenatal testing which was abnormal. She had a Second Trimester Screen with a risk of trisomy 21 of 1 in 160 (Down's risk due to maternal age alone was 1 in 360). It is unclear from the GP's antenatal records or office chart what counseling was undertaken around this result. She had a level II ultrasound at the district hospital at 26 weeks and the indication given on the report was "MSS findings". It is unclear why this occurred so late in the pregnancy when the recommended timing for that ultrasound would be 18-20 weeks. There is a consultation note on file from her obstetrician gynecologist, done at 32 5/7 weeks, apparently for the abnormal screening. The note contains documentation of an ultrasound done at 31+ weeks showing single intrauterine pregnancy at 30 5/7 weeks.

The plan was to have the patient return to the clinic as needed after the delivery of the baby. It appears that neither the GP obstetrician nor the obstetrician gynecologist recognized this as an at-risk pregnancy. There was no evidence in the records that a 50 gram GTT was administered despite BMI and positive family history of diabetes (both parents). The patient screened positive for GBS (no susceptibility testing despite known penicillin allergy). The Antenatal Record 2 documents weekly visits up to February 20 2008 (i.e. 40 5/7 by ultrasound), with no increase in SFH since 35 weeks (36 cm) and no documentation of any discussion of foetal well being or plans for surveillance or induction.

It was only from a hospital chart from the February 21 2008 admission that the failed induction attempts are noted. The records include foetal heart tracings from February 3, 4 and 11, 2008 which were reassuring, but the reason they were done is unknown as there are no notes. The first Cervidil attempt appears to be on February 11, 2008. There were no notes, only the foetal heart tracing. It appears as though no further ultrasounds were obtained, despite the pregnancy being at-risk. Documentation on Antenatal 1 and 2 is scant with no lab results documented and no final EDB documented.

The patient was admitted to hospital on February 21, 2008 for induction of labour with Oxytocin after two failed Cervidil attempts at induction (the most recent being February 20, 2008 at 1910 hours). She presented to the delivery suite at 0815 hours for Oxytocin and was found to be 2 cm 60% effaced. The Cervidil had fallen out overnight at 0300 hours on February 21, 2008. The foetal heart tracing was non-reassuring from the start

on February 21, 2008. The tracing became more concerning after Oxytocin was started at 0900 hours and was discontinued at 0940 hours after a prolonged deceleration. The GP obstetrician consulted with the family physician and a general surgeon regarding an emergency Caesarean section. Plans for "stat c-section" began at 0942 hours. The patient underwent the Caesarean section under spinal anesthesia at 1140 hours. The procedure took place two hours after the decision was made and documented in the chart. The baby weighed 2.38 kg (growth restricted), head circumference 32 cm and length 47 cm. Apgars were noted to be 5 at one minute and 8 at five minutes. Tubal ligation was performed with the Caesarean section.

Post Natal Course

There was meconium staining and evidence of post maturity. Initially, there was low tone, pallor and poor air entry, but the infant appeared to improve and was transferred to the nursery.

At 1205 hours, the infant was noted to have shallow respirations and became hypotonic. Oxygen was given via face mask, but the heart rate (HR) dropped to below 100. A code was called. The infant was given Intermittent Positive Pressure Ventilation (IPPV) with 100% oxygen with a bag and mask.

At 1210 hours, the HR dropped to the 90's and O2 saturation to 80%.

At 1221 hours, intubation was attempted and failed. The HR dropped below 60 and CPR was commenced. The HR improved to 80 with IPPV and a second attempt was made to intubate. The baby did not tolerate the procedure, resulting in bradycardia of 60, but improved with bagging. An umbilical arterial catheter was inserted.

At 1224 hours, the first dose of Epinephrine 0.5 ml (1:10000) was given via umbilical catheter, followed by normal saline bolus of 20 cc x 2. Once again, CPR was started after attempted intubation. Another saline bolus was given at 1227 hours and a second dose of Epinephrine 0.5 mls was given 3 minutes later.

At 1231 hours, the anaesthetist intubated with a # 2.5 ETT following a dose of atropine. HR was 60-70 and CPR was maintained. Criti-Call was notified. The ETT was removed because of a leak at 1236 hours and a third dose of epinephrine 0.5 ml given, with HR of 85.

At 1240 hours, the infant was was intubated with a #3.5 ETT and showed better air entry. HR improved to 100, but laboured respirations were documented. At 1245 hours, the HR was between 100 and 120

By 1251 hours, her temperature had dropped to 34.1°C (Axillary) and glucose was 1.9. Poor air entry was noted on the left side, so ETT was pulled back 1 cm.

At 1310 hours, 10 cc of 5% Dextrose was given. A chest x-ray was done showing ETT placement at the carina and consolidation of right lower zone and lesser extent on the left.

By 1315 hours, her temperature was still low at 34.4°C, but the HR was up to 120-130 and her colour and tone were improving. Blood glucose was 3.8.

At 1325 hours, the infant's HR was 140 and BP 60/40. Ampicillin and Gentamicin were given. NICU was contacted at 1330 and advised to commence 10% Dextrose infusion at 6cc/hr. ETT was suctioned for thick brown fluid.

At 1405 hours, arterial gas ws obtained: pH 7.04, pCO₂ 53, PO₂ 41, HCO₃ 11, B.Ex - 19.1 Electrolytes Na⁺ 137, K⁺ 3.3 (low), Cl⁻ 106, Blood count Hb 172, WCC 17.9, N 0.4, L 0.53.

At 1420 hours, a peripheral IV was established for dextrose infusion and arterial catheter locked (subsequent catheter infusion with 1 cc NaCl was started at 1539 hours).

At 1432 hours, the capillary gas showed: pH 7.11, pCO₂ 44, pO₂ 49, HCO₃ 12, B. Ex - 18, rectal temperature of 38°C.

By 1445 hours, the HR was 160 and the infant appeared pink. Systolic BP was 60 and O₂ saturation was 88-90% in 100% oxygen. Peripheral perfusion had improved. A further 25 cc bolus of saline was given at 1505 hours. The infant's vital signs at 1523 hours showed a good HR of 150, temperature of 37.5°C, glucose of 4, but O₂ saturation was down to 79%.

At 1551 hours, capillary gas showed: pH 7.07, pCO₂ 55, PO₂ 42, HCO₃ 12, B.Ex -17.

At 1630 hours, the infant was still being given IPPV with bag/mask using 20 cc of H₂O pressure, PEEP of 5 and rate of 50. HR was 150.

At 1640 hours, capillary gas was: pH 7.13, pCO₂ 52.4, pO₂ 46.5, HCO₃ 14, B.Ex -13.2.

At 1710 hours, 9 cc of NaHCO₃ 4.2% was given over two hours using a second IV. However, the infant went dusky within 10 minutes with O₂ saturation of 70%. Fentanyl bolus of 1.25 mcg was given and infusion started at 1730 hours. Oxygen saturation remained at 70%. Glucose and temperature were normal.

At 1840 hours, arterial gas showed: pH 7.18, pCO₂ 53.9, pO₂ 34.8, HCO₃ 16, B.Ex -9. O₂ saturations dropped to 60%. Fentanyl infusion was discontinued due to poor respiratory effect and low tone. O₂ saturation remained in the low 50's, but improved with bagging. Another saline infusion of 38 cc was given at 1915 hours. O₂ saturation dropped to 30's and HR to 60's. CPR was started and a chest x-ray showed a right pneumothorax which was initially drained with a #25 butterfly needle (30 cc of air aspirated).

At 1950 hours, a fourth dose of Epinephrine 0.5 ml was given and at 1955 hours, #23 butterfly was replaced by #21 and a total of 35 cc of air was aspirated. HR dropped to 50 and then to 30, requiring a fifth dose of Epinephrine.

At 2003 hours, CPR was in place and air aspiration continued. A sixth dose of epinephrine 0.5 ml was given.

At 2014 hours, 10 ml of NaHCO₃ was given over five minutes. Surgery was called and a chest drain was started using a #6 FG feeding tube since a chest drain was not available.

At 2020 hours, a seventh dose of Epinephrine 0.5 ml given.

At 2030 hours, the HR was down to 60 and peripheral pulses were not palpable. The Transport Team arrived at 2031 hours and gave another dose of Epinephrine 0.5 mls. The infant's temperature dropped again to 34.9°C and the ninth dose of Epinephrine 0.3 ml was administered. She also received NaHCO₃.

At 2031 hours, blood gas had deteriorated significantly to: pH 6.52, pCO₂ 163, pO₂ 27.2, HCO₃, B.Ex -37.2. The infant was in asystole, appeared moribund, had areflexia and pupils were dilated and fixed. After discussion with the parents, resuscitation was stopped at 2049 hours.

Post Mortem

External examination:

1. Gestation consistent with post term baby.
2. Birth weight, head circumference and length consistent with Small for Gestational Age infant.

Flat facies, bradycephaly, slightly up slanting palpebral fissures, intracanthal distance 2.5 cm and interpupillary distance 3.6 cm, bilateral epicanthal folds, depressed nasal bridge and flat philtrum. Small low set ears and short fingers.

Chromosomal diagnosis confirmed of 47xx – 21x (Down's syndrome).

Internal examination:

Lungs: Left lung somewhat hypoplastic, airless, congested and collapsed. Lung volumes lower than normal – 49 cm³ (normal 60 cm³). Evidence of congestion and minimal local intra-alveolar haemorrhage. Meconium present.

Heart: weight 20 gm – large for body size. Complete, balanced atrioventricular septal defect. Valvular anomaly – common atrioventricular valve, estimated annular circumference of right sided portion 30 mm (N 38 + /-5).

Patent ductus arteriosus.

Liver: 93.1 gm (N 185 gm).

Macrovascular steatosis and congestion.

Adrenals: Right 2.9 gm and left 3.2 gm. (normal combined wt 10.5 gm). Marked vacuolisation and steatosis.

Kidneys: Right 8.6 gm. Left 9.5 gm (N combined weight 21.2 gm).

Brain: 323 gm (normal for age is 387 ± 61 gm).

Global, acute hypoxic–ischaemic injury.

Placenta: fetal circulation shows vascular obstruction on both arterial and venous sides.

Chorionic villus– chorionic vascular karyorrhexis. Avascular villi, dysmature and show chorangiosis – usually associated with diabetes but placental is small and therefore not suggestive of diabetes. Chorionic non-occlusive thrombi.

Discussion

The mother should have been identified as an at-risk pregnancy by virtue of her increased BMI and her positive Second Trimester genetic screening test. It is unclear from the documentation what counseling she received and her options to pursue further non-invasive (i.e. foetal echo) or invasive (amniocentesis) testing. Her level II ultrasound did not occur until 26 weeks, which is after the recommended window of 18-20 weeks. It was an incomplete assessment secondary to body habitus and foetal positioning. The need for counseling and/or increased foetal surveillance secondary to the abnormal screening does not appear to have been recognized by either her GP obstetrician or the gynecologist.

Intrauterine growth restriction developed and was not recognized; surveillance with ultrasound was not undertaken and maternal body habitus was such that SFH measurements were not particularly helpful (e.g. 5 foot 1 inch with final pregnancy weight 260 lbs).

Induction was attempted at 39+ weeks and again at 40+ weeks with Cervidil. The foetal heart monitoring prior to February 21st, 2008 was reassuring, but was non-reassuring at admission February 21, 2008. It is unclear why Oxytocin was started in the setting of a non-reassuring foetal heart tracing. It is also unclear why, once the decision for stat Caesarean section was made, it took two hours to begin. It seems that the trisomy 21, underlying heart abnormalities and growth restriction impacted this fetus' ability to withstand the challenges of this labour and subsequent resuscitation.

This infant died as a result of a number of factors:

- The infant had intrauterine physiologic distress associated with passing of meconium and aspiration. This is evident with placental histology. She also had a complete Atrioventricular Septal Defect (associated with Down's syndrome), which contributed to her demise.
- The metabolic acidosis did not resolve despite aggressive therapy, leading to persistent bradycardia and hypoxia.
- Although BP is recorded on two occasions, there is no documentation of mean BP and there is a strong likelihood of significant hypotension. The unsuccessful attempts at intubation were detrimental and prolonged hand-bagging may have contributed to the pneumothorax. Although multiple doses of epinephrine were given

with some success, a continuous infusion of an inotropic medication may have been more effective. Cord gas was not done to assess foetal well-being and the first blood gas was done at two hours of age. In general, the resuscitation was appropriately administered.

Recommendations:

1. Obstetrical care providers are reminded of the importance of clear documentation.
2. Obstetrical care providers are reminded of need to identify early at risk pregnancy and offer as timely as possible consultation to help plan further management. This is especially important in smaller centres which do not have on site access to more specialized care.
3. Obstetrical care providers are reminded of the importance of identifying and acting on abnormalities in fetal heart monitoring.
4. Paediatric health care providers are reminded that early blood gas estimation is essential in dealing with compromised babies as well as close monitoring of vital signs.

Case: 2009-N-3

Maternal History

The mother of the deceased was a 31 year old primigravida. She had no problems in her pregnancy to date. Two ultrasounds were done at 14 and 20 weeks which confirmed her dates. No abnormalities were detected and there was no placenta previa. She declined genetics screening. At 27 weeks and 4 days, she presented to the emergency room at 2100 hours with constant supra-pubic pain for one hour and discomfort with sitting. Her pulse was 98 bpm and blood pressure 136/88. She was transferred by the nurse from the triage area to the labour room for assessment. The foetal heart rate was auscultated at 100-104 bpm. No foetal activity was noted. Abdominal tightenings were palpated. At 2154 hours, the obstetrician on call was contacted and informed of the mother's pain, the foetal heart rate and the uterine activity. The nurse twice requested that the obstetrician attend to assess the mother. The obstetrician declined to attend, but ordered a urinalysis and asked the nurses to call him with the result.

The nurse obtained the urine sample and commenced external EFM. The tracing was abnormal with a baseline of 120 bpm and minimal variability. There were no accelerations and no decelerations. The nurse noted that the mother was very tender on her abdomen and reacted to the slightest touch. The mother was described as weepy and uncomfortable. At 2218 hours, the nurse again called the obstetrician about the abnormal EFM, which she described as "almost a flat line". The nurse also informed the obstetrician of the tenderness of the mother's abdomen and increasing pain. The nurse asked the doctor to attend and assess the mother. The doctor ordered that the

nurse perform a vaginal exam on the patient. The exam was completed and the doctor was re-contacted at 2232 hours and informed that the nurse had difficulty finding the cervix due to an irregular fold in the vagina. After some further discussion of the patient's symptoms over the telephone, the doctor ordered Demerol, Gravol, Ampicillin and Serax. The nurse again reported the abnormal EFM to the doctor.

At 2247 hours, the nurse discontinued the EFM for the mother's comfort and she administered the Demerol and Gravol at 2300 hours. Between 2300 and 2330 hours, the IV was started and Ampicillin was given. The Demerol was noted to be effective. At 2330 hours, the monitor was again applied and an abnormal pattern was again detected. There was undetectable variability and repeated late decelerations with a baseline of 140 bpm.

At 2357 hours, the obstetrician was again contacted and advised staff that he was on his way in. The obstetrician arrived at 0050 hours and assessed the mother and the EFM tracing and discussed with her the need for transfer to a tertiary centre. Morphine was ordered and given at 0104 hours. The obstetrician made a diagnosis of preterm labour at 27 weeks and possible urinary tract infection. No note was made by the obstetrician of the abnormal EFM. The obstetrician made arrangements through Criti-Call to transfer the mother to a tertiary centre. She was given Celestone IM at 0123 hours. The EFM tracing continued to be abnormal with undetectable variability, repeated late decelerations and a baseline of 150 bpm. The ambulance departed with the patient and a nurse at 0148 hours.

At 0250 hours, the mother arrived at the tertiary centre. She was assessed by the obstetrics resident and a nurse. The mother's blood pressure was 133/93, her pulse was 98 and she was afebrile. A bedside ultrasound revealed the fetus to be in breech presentation with normal amniotic fluid and an inhomogeneous placenta. The EFM was abnormal with undetectable variability, a baseline of 140 bpm and repeated late decelerations. Her cervix was closed. At 0335 hours, the perinatologist on call reviewed the mother's condition. A diagnosis of concealed abruption and abnormal EFM was made and an emergency Caesarean section was recommended.

Spinal anaesthetic was placed at 0432 hours and surgery commenced at 0441 hours. A Pfannenstiel skin incision was used and a mid transverse incision was made in the uterus. The baby was delivered as a complete breech extraction at 0447 hours. The placenta delivered soon after the baby and was followed by a large clot suggestive of placental abruption. There was a blue hue to the serosal surface of the uterus suggestive of a Couvelaire uterus, but excessive bleeding was not encountered. The patient's post operative course was uneventful and she was discharged on the second day. She was advised to follow in the high risk clinic for future pregnancies and deliver by repeat Caesarean.

Neonatal History

The infant was described as flat at delivery. Resuscitation with bag and mask ventilation was commenced with poor response. The baby was intubated at one minute

of age and there was a slow improvement in heart rate with intermittent positive pressure ventilation. Tone and reactivity were slow to improve and first gasp did not occur until 6 minutes of age. Apgar scores awarded were 2 at one minute, 3 at five minutes and 4 at ten minutes of age. Cord gases revealed severe acidosis with pH 6.92, pCO₂ 76 mmHg, HCO₃ 15 mmol/L and base deficit of 21 mmol/L. The infant's general condition remained poor and she was transferred to the NICU for further assessment and management. She was placed on a ventilator on arrival.

The infant had a birth weight of 890 g. There were no apparent dysmorphic features and external physical examination was appropriate for her gestational age. She remained poorly responsive with little independent respiratory effort. She was placed on a ventilator and lines were inserted. She was described as dusky and poorly perfused. Oxygen saturation was below 80% in 100% oxygen. Surfactant was given with little effect. Blood pressure was low and she was given two normal saline boluses. She was switched to high frequency oscillatory ventilation and started on a dopamine infusion. Antibiotics were given after blood cultures were drawn. The first arterial blood gas taken at two hours of age showed pH 6.87, pCO₂ 69 mmHg, pO₂ 20 mmHg, HCO₃ 13 mmol/L and base deficit of 22 mmol/L. She was started on inhaled nitric oxide with very transient improvement. The presumed diagnoses included hypoxic ischaemic encephalopathy, persistent pulmonary hypertension and systemic hypotension.

The baby remained extremely unstable in spite of the provision of full, aggressive intensive care measures. Investigations included an echocardiogram which confirmed the presence of pulmonary hypertension and cerebral ultrasound which showed large intraventricular haemorrhages bilaterally. She exhibited seizure activity that was treated with anticonvulsants and sedation. Hypotension was persistent and refractory. There was evidence of renal impairment and a bleeding diathesis, presumably secondary to disseminated intravascular coagulation. She experienced several serious bradycardic spells requiring full CPR. A hopeless prognosis was delivered to the parents.

The infant was extubated and passed away at 0550 hours on June 19, 2007.

Post Mortem

No congenital abnormalities or evidence of bacterial or viral infection were identified. The brain showed subependymal matrix hemorrhage with intraventricular extension involving all ventricles. Marked subarachnoid hemorrhage was noted at the base of the brain stem. There was evidence of hyaline membrane disease of the lungs. No evidence of maternal cocaine use was identified on fetal hair and meconium testing. Cytogenetics testing revealed a normal female karyotype.

Death was attributed to "acute hypoxic ischaemic encephalopathy due to severe prematurity and placental abruption".

Examination of the placenta showed no reason for the abruption and no evidence of infection.

Discussion

The mother presented at 27 weeks' gestation to her local hospital with evidence of abnormal fetal status on EFM and clinical signs and symptoms highly suggestive of placental abruption. There were several delays in her diagnosis and treatment. She waited almost four hours to be assessed by the local obstetrician. After that assessment, a decision was made to transfer her to a larger centre which took another two hours and although emergency Caesarean was recommended shortly after her arrival in the tertiary centre, the baby was not delivered until almost two hours after her arrival.

In the case of the local hospital delay, the nursing staff were clearly very concerned about this woman and her baby, but could not convince the obstetrician on call to come to see her immediately. They did not however, use other avenues such as calling the department head or the Chief of Staff, to have this patient assessed.

In the case of the delay at the tertiary centre, the chart does not indicate why it took so long to mount an emergency Caesarean.

At delivery, the baby was severely depressed and it eventually became clear that there had been a profound prenatal insult. The infant was in extremis throughout her hospital stay and failed to respond in spite of full intensive support. Cerebral ultrasound revealed large, bilateral intraventricular haemorrhages. The paediatric team provided expert, skilled and sensitive care, but it is clear that no treatment provided postnatally would have been able to improve the outcome.

Recommendations

1. Obstetrical health care providers are reminded of their duty to assess, treat and document in a timely fashion.
2. Nursing staff are reminded that if the doctor fails to respond appropriately they may seek other care for the patient.
3. The local hospital should establish guidelines for nurses to use when they are unable to get a satisfactory response from an attending physician.

Case: 2009-N-4

Antenatal History

The mother of the deceased was a 30 year old G3P2. Her LMP was June 15, 2006, but she had a history of irregular cycles. A dating ultrasound on August 29, 2006 gave a gestational age of 8 weeks 1 day, giving an EDD of April 9, 2007. Routine prenatal laboratory investigations were normal including second trimester genetic screening. There is no record of a GCT being done, although a random blood sugar completed on October 6, 2006 was within normal limits. A GBS culture was not done.

The patient's past obstetrical history was for two term pregnancies delivered in Yugoslavia. The first was an uncomplicated pregnancy delivered at 40 weeks of a 3.5 kg female infant in 1997. The second pregnancy was delivered at 40 weeks of a 3.2 kg female infant in 1999 and was complicated by abruption and induction of labour.

The patient's medical history was significant for chronic hypertension diagnosed in 2003. She was on Metoprolol 50 mg daily at the beginning of the pregnancy. At the first prenatal visit on August 21, her BMI was 36 and blood pressure was 140/100. The Metoprolol was discontinued and she was prescribed Labetalol 100 mg, twice daily. Subsequent blood pressure recordings at prenatal visits ranged from 130-140/80-100. At her last prenatal visit recorded on the Antenatal Record 2, no blood pressure was recorded. The SFH was 36 cm. The notation under "Urine Prot." is illegible.

At 37 weeks gestation, the patient presented to a walk-in clinic with decreased foetal movement. She was sent to the hospital for foetal assessment. The NST was non-reactive and a decision was made to induce labour.

Course in Labour and Delivery

The patient was admitted to hospital and 1 mg Prostin gel was given intravaginally. She started having uterine contractions and the heart rate went down to 100-110 bpm. The obstetrician was notified, attended and performed an ARM for a large amount of fluid and some blood clots. A foetal scalp clip was applied. The foetal heart rate was noted to go down slowly and did not recover. A decision was made to perform an emergency Caesarean section. It was noted that the placenta appeared to be full of blood clots going into the placenta proper.

The female infant was delivered at 1741 hours with a birth weight of 3.72 kg. The paediatrician had been summoned and was in attendance. No vital signs were detected. Resuscitation was commenced with bag and mask ventilation and cardiac compressions. There was no response, so the infant was intubated and a dose of epinephrine was given via the Endo Tracheal Tube. Positive pressure ventilation and cardiac compressions were continued. Intravenous access was secured through the jugular vein. Four additional doses of epinephrine were administered, including one given directly intracardiac. A bolus of normal saline was given IV. At 35-40 minutes of age, the baby was seen to take a gasp and a low heart rate was detectable. The heart rate slowing climbed to greater than 100 bpm. Additional medications were administered, including calcium gluconate, sodium bicarbonate, two additional doses of epinephrine and another normal saline bolus. Her colour and perfusion improved, but she remained flaccid and unresponsive. Apgar scores were 0, 0 and 0 at one, five and ten minutes. Cord arterial blood gases revealed severe acidosis with pH < 6.8 and pCO₂ > 115 mmHg.

The baby was transferred to the NICU for further management which included mechanical ventilation and blood pressure support with intravenous fluids and infusions of Dobutamine and Dopamine. A blood culture was drawn and antibiotics given. The first blood gas (capillary) from the baby at approximately 2 ½ hours of age, showed

ongoing severe acidosis with pH 6.93, pCO₂ 40 mmHg, HCO₃ 8 mmol/L and base deficit 24 mmol/L. The local tertiary Neonatal Intensive Care Unit was contacted and a transfer was requested. Full support was continued and the Transport Team arrived at 2300 hours when the infant was 5 ½ hours of age.

The Transport Team assessed the baby and described her as very flaccid and hypotonic. There was some bruising over the chest and a large bruise in the left groin area. Umbilical lines were inserted and fluids adjusted to maintain blood sugar in the normal range. The baby exhibited some abnormal movements felt to represent seizure activity and Phenobarbital was given. Blood pressure remained low, so she was given additional fluid and started on an infusion of epinephrine. Fresh frozen plasma was given after INR and PTT were checked and found to be prolonged. Ventilation was adjusted and infusion of sodium bicarbonate given in order to improve the blood gases and oxygen saturation. The infant was transported and arrived at the NICU in stable, but critical condition.

At the NICU, the baby remained neurologically obtunded with severe hypotonia and absent reflexes. Cardiovascular status had stabilized, but she remained fully ventilator-dependent. An Electroencephalogram (EEG) was performed and showed virtually absent cortical activity. The medical staff all agreed that the infant had sustained such severe central nervous system (CNS) injury that survival with a reasonable quality of life would be virtually impossible and recommended withdrawal of life-sustaining medical therapy. The family agreed to this approach and the infant was extubated at 1400 hours on March 15, 2007. The infant was pronounced dead at 1423 hours.

Post Mortem

Significant findings at autopsy related to changes of hypoxic-ischemic encephalopathy (slight).

The hospital pathology report of the placenta showed a marginal insertion of the umbilical cord. There was no meconium staining and no significant gross haematoma. The forensic report showed evidence of intervillous hemorrhage.

The cause of death was stated as hypoxic-ischemic encephalopathy due to abruption placenta.

Discussion

This infant died as a result of acute asphyxia from placental abruption. Antenatally, risk factors present for placental abruption were a past history of abruption in the mother's second pregnancy and chronic hypertension. Her blood pressure was treated with Labetalol 100 mg twice daily, and appears to have been well controlled. There was no additional foetal surveillance testing during the third trimester such as NST or ultrasound/Bio Physical Profile as would usually be done given the risk factors. Also, clinical assessment of foetal growth is severely compromised in the setting of a high

BMI and although ultrasound can be technically difficult, it is of more utility than SFH measurements.

Review of the foetal heart rate monitor strips shows commencement of the tracing at 1240 hours. The tracing is essentially flat until the development of subtle decelerations around 1700 hours. Shortly thereafter, the tracing deteriorated further to a terminal bradycardia pattern. The tracing ends at approximately 1720 hours with the notation “to OR”.

The administration of a cervical ripening agent is clearly contraindicated in this setting as there was no indication of foetal well-being. On the contrary, preparations for Caesarean section should have been made shortly after admission. It cannot be determined by this review if such intervention would have led to a more favourable outcome.

Review of the paediatric records indicates that the baby was effectively stillborn, with no detectable vital signs and no response to standard resuscitative measures. Only after multiple doses of epinephrine, including an intracardiac injection, did the baby’s heart begin to beat again. There was a period of over 30 minutes during which the baby remained asystolic in spite of full resuscitative efforts. Under such circumstances, intact survival would be virtually impossible, even with the subsequent return of cardiac function. It is clear that no interventions applied after delivery could have resulted in survival with a reasonable quality of life. Current Neonatal Resuscitation Protocol guidelines state, “If there is no heart rate after 10 minutes of complete and adequate resuscitation efforts, and there is no evidence of other causes of newborn compromise, discontinuation of resuscitation efforts may be appropriate. Current data indicate that, after 10 minutes if asystole, newborns are very unlikely to survive, or the rare survivor is likely to survive with severe disability.”

The paediatric team at the first hospital provided prompt and skilfully applied resuscitative and post-resuscitation measures. After the heart beat was restored, the providers took the necessary steps to support the infant until such time as an independent assessment by clinicians at the local tertiary care centre could be performed. The local paediatric care providers, the transport team and the providers at the tertiary centre provided care of high standard, but nothing could be done postnatally to change the outlook. After the poor prognosis was confirmed through assessment of the history, physical examination of the baby and EEG, withdrawal of life-sustaining medical therapy was recommended and undertaken. Post-mortem examination failed to reveal any alternative explanation for the baby’s demise, other than acute hypoxic-ischemic encephalopathy.

Recommendations

1. The obstetrical care providers in this case should be required to demonstrate competence in the interpretation and management of foetal heart rate tracings.

2. Obstetrical care providers are reminded that cervical ripening agents are contraindicated in the absence of normal tests of foetal well-being.

Case: 2009-N-5

History

The mother of the deceased was a 34 year old G2P0 with an EDD of December 20, 2007. Routine prenatal laboratory investigations, IPS and GCT were normal. Ultrasounds done at 7, 13, 19 and 23 weeks were normal and in agreement with the EDD. Her medical history indicated that she had Stevens-Johnson Syndrome.

On November 28 at 36 weeks 6 days gestation, GBS culture was done and subsequently reported as negative. At that visit, her blood pressure was 120/80 and continued in that range for the duration of the pregnancy. There was no proteinuria. SFH was 37cm.

On December 12, 2007 at 38 weeks 6 days, the mother reported decreased foetal movement. SFH was 40 cm. A NST and BPP were ordered and subsequently reported as normal.

On December 20 at 40 weeks gestation, the patient complained of a visual disturbance and was sent to triage for assessment. NST and Pregnancy Induced Hypertension (PIH) laboratory investigations were normal and she was discharged home.

On December 27 at 41 weeks gestation, the cervix was a fingertip dilated, 70% effaced and the vertex at spines -3. An NST was performed and reported as normal. PIH laboratory investigations were done and were normal. Plans were made for induction of labour on December 31 at 41 weeks 4 days, to be preceded by cervical ripening on December 30, 2007.

Course in Labour and Delivery

The patient began having uterine contractions at 2100 hours on December 28, 2007. She reported feeling foetal movement prior to the onset of contractions, but none after the onset of contractions. She presented to the labour and delivery unit of the hospital, located in Northern Ontario, at 0150 hours. She was placed on an EFM and was noted to have variable decelerations. The obstetrician was notified at home at 0230 hours and requested that the tracing be faxed to him. At 0235 hours, while reviewing the fax, he received a phone call informing him of a prolonged deceleration. He ordered preparations be made for emergency Caesarean section after being informed that the patient was 1 cm dilated. The obstetrician contacted the anaesthetist on call who was busy in the operating room. Arrangements were made for the second on call anaesthetist to come in.

The obstetrician arrived at 0245 hours followed by the anaesthetist at 0255 hours. Under general anaesthesia, the patient was delivered at 0303 hours of a 3775 gm female infant through thick meconium. The umbilical cord was described as thin and

was wrapped around the shoulder. Apgars were 0, 0, 0 and 3 at one, five, ten and fifteen minutes. Cord gases were arterial pH 7.07, pCO₂ 70, pO₂ 42, HCO₃ 20, base deficit 10.7 mmol/L and venous pH 7.11, pCO₂ 63, pO₂ 38, HCO₃ 20 and base deficit 10.1mmol/L.

The paediatrician had been summoned and was in attendance along with other members of the paediatric team. The baby was immediately intubated and meconium suctioned from the upper airways. Resuscitation was instituted with bag and mask ventilation and cardiac compressions. The infant was reintubated at two minutes of age and positive pressure ventilation and cardiac compressions continued. Two doses of epinephrine were given via the Endo Tracheal Tube (ETT), followed by a dose intravenously. Heart rate was first detected at 15 minutes of age, but output remained poor. A bolus of normal saline was given resulting in an improvement in the pulses. Umbilical catheters were inserted, blood cultures drawn and antibiotics administered.

The first spontaneous respiratory efforts were observed at one hour of age. The first arterial blood gas from the baby drawn at one hour of age showed a profound acidosis, with pH 6.8, pCO₂ 44mm Hg with a serum HCO₃ and base deficit that were incalculable. The infant was given sodium bicarbonate intravenously and continued on mechanical ventilation. Initial CBC was normal, showing no evidence of acute haemorrhage or infection.

Initial physical examination revealed a well grown term neonate with birth weight of 3.77 kg. Although spontaneous respirations were becoming steadier, the infant remained poorly responsive to any form of stimulation. Suspected seizure activity was observed at around six hours of age and she was treated with Phenobarbital. Tests revealed evidence of multiorgan damage with elevated liver enzymes, evidence of a DIC, elevated creatinine, poor urine output and haematuria. After discussion with physicians at the tertiary hospital in Southern Ontario, it was decided that she should be transferred there for further assessment and management. Transfer was affected to 1700 hours on December 29 when the infant was 14 hours of age.

On arrival at the tertiary centre, the infant was stable from a cardio-respiratory perspective, but remained neurologically obtunded. She subsequently required treatment with dopamine to support her blood pressure. Seizure activity was detected and she was treated with additional anticonvulsants. Systemic hypothermia was instituted until she was 72 hours of age in an attempt to attenuate the CNS injury. However, little improvement in her clinical condition ensued.

EEG and MRI were performed and suggested severe hypoxic ischaemic encephalopathy. The findings and poor prognosis were relayed to the family and upon discussion with the clinical team, it was decided that the infant should be transferred back to the birth hospital in Northern Ontario, where life-sustaining medical therapy would be withdrawn and palliative care provided. This was arranged and the infant was flown back on January 3, 2008 at six days of age.

The infant arrived intubated and ventilated with an infusion of dopamine running to support her blood pressure. She was described as flaccid with poor reactivity and no

gag reflex. Although the parents initially expressed uncertainty about the decision to withdraw life-sustaining treatment, they eventually agreed that this would be the most appropriate course of action. The infant was extubated on January 5 at 8 days of age. Palliative care was provided and she remained with her parents until she passed away on January 6 at 1130 hours.

Post Mortem

At autopsy, there were findings of perinatal asphyxia with meconium staining of the skin, acute tubular renal necrosis and global, severe, subacute hypoxic-ischemic brain injury.

The cause of death was hypoxic-ischemic complications of perinatal asphyxia of undetermined etiology.

Discussion

This infant died eight days after birth from perinatal asphyxia. Autopsy findings did not reveal an underlying cause. This was a postdates pregnancy and it is recognized that perinatal mortality is increased in this setting. Specifically, pregnancies beyond 41 weeks gestation have a one third greater incidence of neonatal mortality compared to 38-40 weeks gestation. Asphyxia, meconium aspiration and intrauterine infection all contribute to the excess perinatal deaths. This discussion will therefore focus on this possible etiology and the important clinical factors in antenatal care that are particularly germane, namely dating of the pregnancy and foetal surveillance and timing of delivery.

The mother's LMP was March 15 and ultrasounds at 7 and 13 weeks were consistent with an EDD of December 20. Therefore the EDD was well established and the pregnancy was 41 weeks 2 days at the time of delivery.

It is recognized that pregnancies that extend beyond 41 weeks gestation are associated with an increased risk of perinatal mortality. While the optimal gestational age for initiating foetal monitoring has not been determined, the Society of Obstetricians and Gynaecologists of Canada (SOGC) and the American College of Obstetricians and Gynaecologists (ACOG) recommend testing between 41-42 weeks. The most commonly used tests include foetal kick counts, Non Stress Testing, Bio Physical Profile (BPP) and the modified BPP (NST plus Amniotic Fluid Volume assessment). The SOGC Clinical Practice Guideline (June 2000) states that no single method of antenatal surveillance has been shown to be superior to another. Evaluation of amniotic fluid volume was identified as being especially important because of a demonstrated increase in adverse outcomes in the setting of oligohydramnios.

Optimal timing of delivery has not been clearly established. Most obstetrical care providers would recommend delivery before 42 weeks gestation.

Increase foetal surveillance applied in this pregnancy consisted of NST's at 38 weeks 9 days, 39 weeks 5 days and 41 weeks. On review, these tracings meet the criteria for normal. A BPP done at 39 weeks 5 days scored 8/8 with an Amniotic Fluid index of 144.

Although postdates was not the indication for the tests being done, they were reassuring of foetal well-being up to 41 weeks. Poor perinatal outcomes are rare within 24-48 hours of a normal NST in the absence of an acute event such as a placental abruption or cord occlusion. A possible mechanism explaining the outcome in this case is cord compression aggravated by low amniotic fluid volume. There is no consensus however for performing a test of amniotic fluid volume prior to 41 weeks 2 days.

The response of the obstetrical care providers to the foetal heart rate tracing was timely and appropriate.

The infant was well managed by the neonatal team at the birth hospital and the transfer hospital. Once ongoing clinical assessment with EEG and neuroimaging confirmed the severe neurological injury, the parents agreed to the provision of palliative care only.

Care provided by the paediatric team in birth hospital was of a very high standard. Resuscitation efforts were promptly and expertly applied. The investigation and treatment of the baby's condition was of a very high standard. The parents were fully informed and involved. When it was recognized that the infant was exhibiting evidence of severe CNS and multiorgan failure, consultation with, and transfer to the tertiary centre was undertaken. At the tertiary centre in Southern Ontario, the infant's neurological status failed to improve in spite of a trial of systemic hypothermia for neuroprotection. It became clear that she had sustained severe, irreversible neurological injury and that her long term prognosis was extremely poor. The parents agreed to transfer the infant back to the birth hospital in Northern Ontario for the provision of palliative care only.

Life sustaining medical therapy was withdrawn and the infant died at 8 days of age.

Although it is clear that the infant was severely compromised from the time of birth and it is likely that no change in the postnatal management would have resulted in a different outcome, there are a few issues that warrant comment. Firstly, while hypothermia for neuroprotection was not considered standard care for babies with severe perinatal hypoxic-ischemic encephalopathy at the time this infant was born, evidence has accumulated such that there appears to be consensus in the neonatal community that this treatment should be offered to eligible neonates. The team at the tertiary centre applied a cooling protocol in this case, but it was not commenced until after the baby arrived, well after the accepted optimal timing of initiation. All studies to date that provide support for hypothermia in this setting have applied it at less than 6 hours of age and, although there may be some benefit in selected cases if hypothermia is applied after this age, it is clear that it is best instituted soon after birth.

While it is difficult to know whether hypothermia applied sooner after birth might have resulted in a reduction in central nervous system damage in this case, it appears that the baby had sustained significant hypoxic ischemic injury prior to delivery and it is very unlikely that the damage would have been ameliorated. If systemic hypothermia for neuro-protection of compromised neonates is to be adopted, a system-wide approach will be required so that this therapy can be applied soon after birth, regardless of where the baby is born.

There appears to have been a terminal event that produced the sustained bradycardia and subsequent asystole in the infant, but there is no clear mechanism. The cord gases were not profoundly disturbed as would be expected when a baby is born compromised to this degree. The blood gas taken from the baby at one hour of age indicates severe tissue hypoxia with lactic acidosis. Since there appears to have been no omissions or problems in the postnatal management, the best explanation is that there was an acute cord accident of some sort resulting in complete cord occlusion on utero. The umbilical cord was described as being wrapped around the shoulder at delivery. It is plausible that, if tightly wrapped, complete occlusion may have been the cause of the CNS injury to the baby.

Recommendations

None

Case: 2009-N-6

Maternal History

The mother of the deceased was a G4T2A1L1 with an EDC April 30, 2008 based on good menstrual history and first trimester ultrasound. She had two previous uncomplicated pregnancies with vaginal birth at term in 1998 and 2000. Her pregnancy to date had been normal, as had all screening tests and ultrasounds.

The patient had a spontaneous rupture of membranes on the morning of January 28, 2008. She went to Hospital A as this was the closest hospital. Since there was no level 3 hospital available for transfer, she was sent by ambulance to the Hospital B which was a level 2 hospital. She had undergone a speculum examination to confirm her ruptured membranes. An initial dose of betamethasone IM and intravenous ampicillin were given. Ultrasound confirmed a vertex presentation with decreased amniotic fluid volume. The patient was not in labour at this time and arrangements were made, and accepted, for transfer to Hospital C.

The patient was admitted on January 28, 2008 at 2130 hours to Hospital C where preterm premature rupture of membranes (PPROM) at 27 weeks and 2 days was confirmed. The course of Celestone was completed and the patient was given a 7 day course of oral erythromycin. Vaginal/rectal culture for haemolytic group B strep was reported as positive on January 28, 2008. Repeated complete blood counts were done. The white blood cell count was 13.4 on admission and went as high as 14.2 on January 31, 2008 and as low as 9.8 on February 11, 2008. On the date prior to discharge, the white cell count was 14. The neutrophil count was above the normal range for most of the hospital stay. Several biophysical profiles were done during the hospital stay with the most recent being done on February 26, 2008 - six days prior to discharge. Tests confirmed decreased amniotic fluid volume and a pocket less than 2 x 2 cm. Doppler studies were normal and fetal growth was appropriate.

The course during the hospital stay was unremarkable apart from one transfer to labour and delivery when there was a small amount of spotting. A note on the chart on February 26 indicates that there was communication with an obstetrician at Hospital B who was to be on call March 4, when the patient was at 32 weeks gestation. The plan was to transfer the patient to Hospital B at that point, assuming expectant management continued until that time. A transfer note was written indicating the acceptance of this patient at 32 weeks gestation with PPRM. The note indicated that documentation regarding issues dealt with during the care and copies of admission records, including ultrasounds, were being sent.

The patient was transferred to the secondary level centre, Hospital B, at 32 weeks and 1 day gestation. She was admitted to the maternity ward and monitored for signs of infection. A complete blood count was done every two days. Fetal movement count and NST were done daily and weekly biophysical profile and doppler studies, as well as full ultrasound for foetal weight, were done every two weeks. All investigations were reassuring until 10 days after her transfer.

At 33 weeks and 4 days gestation, the patient's temperature at 1145 hours was 36.7°C. Her pulse was 100 bpm, which was not unusual for her. She had her NST done at 1230 hours. This tracing was normal. The patient complained of feeling crampy at 1530 hours and at 1600 hours EFM was commenced. The tracing showed a baseline of 140 to 145 bpm with moderate variability and accelerations. There were occasional small variable type decelerations. The nurses noted that mild contractions could be palpated every 2 - 4 minutes. The toco also picked up uterine activity every 2 minutes. Near the end of the 30 minute tracing done on the maternity ward, the baseline increased to 145 to 150 bpm and the variability remained moderate.

The patient was transferred to the Labour Room at 1630 hours. The obstetrician on call examined her at 1650 hours and found her cervix to be a fingertip and the vertex was noted to be high. Her temperature was 37°C at 1700 hours. An IV was started and penicillin G was given. Mild tightenings were noted by the nurses. The patient declined analgesia until 1730 hours. It appears that the fetal monitor was off from 1648 to 1734 hours.

The patient received morphine and Gravol at 1730 hours and EFM was re-commenced. The baseline was now 170-175 bpm with moderate variability and decelerations occurring after each contraction. The toco recorded uterine activity every 2 minutes. At 1750 hours, the obstetrician examined the patient and found the cervix to be 1-2 cm dilated. The plan was to proceed with an epidural, wait for the second dose of pen G, then commence oxytocin. From 1800 to 1830 hours, there continued to be decelerations after each contraction with slow return to baseline and a loss of baseline variability to minimal. The baseline was about 170-180 bpm. The nurse's notes document, "variable decelerations occurring after contractions" and that the obstetrician was aware.

The anesthetist arrived at 1830 hours, but had to restart the IV prior to starting the epidural. At 1840 hours, the patient's temperature was 39.2°C and she was noted to be anxious and hyperventilating. At 1850 hours, after the epidural was placed, the patient

was repositioned on her left side. The baseline heart rate was between 90 and 110 bpm. The nurse examined her and found the cervix to be 2 cm. The patient's temperature was 39.5°C and her pulse was 140 bpm at 1900 hours. The obstetrician was called and it appears from the nursing notes that Caesarean section was discussed at about 1920 hours. The obstetrician also documented the decision to proceed with Caesarean, but there is no time on the note. The obstetrician also documented that calls were made to the pediatrics, anesthesia and assistant on call, and that IV access and blood drawing continued to be difficult.

The patient was transferred to the operating room at 1958 hours. A Caesarean section was commenced at 2018 hours using a midline abdominal incision. The baby was born at 2021 hours. The mother was treated with antibiotics post operatively and recovered without complication.

Pediatric History

The infant girl was born at 33 weeks gestation, following an emergency Caesarean section that was performed due to fetal tachycardia and maternal fever (chorioamnionitis).

A "code pink" was called at birth. The infant had poor colour, no spontaneous movements, but a normal heart rate (>120). Resuscitation was commenced with intermittent positive pressure ventilation using a bag/mask and she was subsequently intubated orally with a #3.5 ETT by anesthesia. In spite of 100% oxygen, the oxygen saturation remained at 60%, the colour was poor and the infant had gasping respirations. The Apgar scores were 3, 5, 5 and 6 at one, five, ten and fifteen minutes. The estimated weight was 2.5 kg, head circumference 32 cm and length 49 cm.

The heart rate remained stable and air entry and colour were both poor. Narcan was given since the mother had received morphine 4 hours earlier. Cord gases showed: pH 6.85, PCO₂ was 86, HCO₃ was 11, and Base deficit was -21.

A chest x-ray showed possible right upper lobe streaking/collapse. The infant was given 5 ml of surfactant with improvement in air entry and oxygen saturation of 79%. A second dose of surfactant 5 ml was given with oxygen saturation improving to 95%. Mechanical ventilation was commenced.

The infant was started on ampicillin and cefotaxime. In view of the chest x-ray, vancomycin was added. At two hours of age, the heart rate was 164 bpm, BP right arm was 40/26, mean of 33 and left arm 48/27, mean of 35; respiratory rate was 40-50 and oxygen saturation was 97% on 100% FiO₂. Dopamine infusion at 5 mcg per kilogram per minute was started.

The Transport Team for the childrens' hospital arrived at 0006 hours on March 16, 2008. The initial assessment showed the baby was tachypneic (RR 70's) plethoric with cyanotic undertone, displayed poor perfusion, but HR was normal. The infant opened her eyes and appeared to tolerate handling. The infant was started on a Cavitron (HSC ventilator). Up slanting eyes were noted, indicating the possibility of Down's syndrome.

The infant was transferred to the childrens' hospital. The assessment at the time was:

- Prematurity - 33 weeks
- History of Preterm Premature Rupture of Membranes
- Respiratory distress – poor oxygenation.
- Likely Perinatal Pulmonary Hypertension of the Neonate
- Presumed sepsis
- Metabolic acidosis
- Suspected trisomy 21

The infant was placed on high frequency oscillation (HFO) ventilation, Umbilical vein and umbilical artery lines were inserted and she continued to receive ampicillin, cefotaxime, and inotropic support. Nitric oxide was initiated. Dobutamine was started and further saline bolus was given.

Gram negative growth on the blood culture was reported from Hospital A. Tobramycin was added in view of the gram negative sepsis and total parenteral nutrition was started.

On March 17, 2008, the infant remained on HFO, needing 32% FiO₂ and maintaining oxygen saturation of 99%. Colour was pale pink and capillary refill time was less than 3 seconds. Cardiovascular findings were stable. However, abnormal movements were noticed: thrusting and lip smacking was suggestive of seizures. The infant was given phenobarbitone at 20 mg per kg.

On March 18, 2008, the infant remained stable from the cardiovascular system and respiratory point of view. Dobutamine and hydrocortisone were being weaned off. Electroencephalogram was abnormal with occasional rhythmic theta activity. Cardiac ECHO showed a large patent ductus arteriosus. Abdominal U/S showed air in the liver (portal venous gas) and coarse echo texture of the organ. Kidneys showed PC dilation. The blood culture confirmed a growth of E. Coli. Phototherapy began for increasing jaundice

On March 19, 2008, seizure activity was noted and there was one episode with desaturation to 80% oxygenation. Lorazepam was given.

A MRI showed white matter ischemia in keeping with cytokine release. This result was discussed with the parents regarding very poor neurological outcome. The family agreed to extubation in the morning.

The infant's condition deteriorated and she was subsequently pronounced dead at 1810 hours.

Post Mortem

Death was due to complications of perinatal asphyxia due to placental chorioamnionitis and cord vasculitis with *E. coli* septicaemia and preterm premature rupture of the membranes.

Discussion

The mother of the deceased had PPROM at 27 weeks gestation and was appropriately transferred to a level 3 centre where she remained over the next five weeks. Her course during that time was normal for this type of complication. Oligohydramnios had developed and was persistent during the stay and she was at risk for developing chorioamnionitis due to the prolonged ruptured membranes. Both of these factors would increase the possibility of having a non reassuring fetal heart when she did subsequently go into labour.

Due to the limitation of both maternal and neonatal beds in a level 3 centre, it is normal practice to consider transferring a pregnant woman to a site nearer her home that can offer the level of care that is needed.

A transfer was therefore arranged at 32 weeks' gestation and accepted by the attending obstetrician at Hospital B. While a centre such as this should have the capabilities of caring for patients at this gestational age, they would have infrequent experience in situations of prolonged PPROM and with the subsequent increased chance of a non reassuring fetal heart and occult chorioamnionitis which may precipitate labour. A high index of suspicion must therefore be maintained as this remained a high risk pregnancy despite the fact that, by gestational age criteria, it would be appropriate for care for in a hospital that has a level 2 nursery.

The mother had spontaneous rupture of membranes at 27 weeks, but did not enter into labour. She was assessed and monitored in a tertiary centre and transferred to a secondary centre after 32 weeks. Observation continued to be reassuring until the day she delivered. She developed uterine activity, secondary to intra-uterine infection with *E coli*. Labour proceeded slowly and the fetal monitoring became abnormal. There were delays in identifying the fetal compromise and further delays in performing the delivery by Caesarean section. When the baby was delivered, she had suffered severe perinatal asphyxia and was septic with *E coli* sepsis.

The infant died of gram negative septicaemia causing multiple organ damage and severe neurological deficit. This is evident in the findings of chorioamnionitis, vasculitis, and funisitis which initiated a sequence of events that led to hypoxic ischemic damage to fetal tissue and severe acidemia. Despite initial improvement in the respiratory status and cardiovascular function, and appropriate antibiotic therapy, this child went on to die. To avoid high mortality, early and aggressive management of prolonged rupture of membranes may be the only effective treatment.

There were no concerns identified with the resuscitation and subsequent therapy at either institution.

Recommendations

For the secondary centre:

1. The obstetrician should review fetal health surveillance to ensure that they are able to identify abnormal tracings and proceed with appropriate interventions.
2. The hospital should review fetal health surveillance with the labour room staff and with the obstetrician to ensure that staff are able to identify abnormal tracings and proceed with appropriate interventions
3. The hospital should review their policy for fetal heart rate monitoring to be sure that continuous electronic fetal monitoring is used when preterm labour is suspected or confirmed and that documentation and terminology is consistent with current standards and begins with admission to the labour room.
4. The hospital should review their policy for medication administration in labour to ensure that a fetal heart rate assessment is done prior to the administration of narcotics.
5. Caregivers are reminded that all notes and orders should be dated and timed.

Case: 2009-N-7

History

The mother of the deceased was a 23 year old G1P0 with an EDD of January 30, 2008. Routine prenatal laboratory investigations were normal. Ultrasound on September 11 at 19 weeks showed normal foetal anatomy, no placenta previa, but evidence of uterine fibroids. The largest fibroid measured 4 cm. The prenatal record does not indicate if a glucose challenge test was done. Culture for GBS was done on January 7.

The patient's past medical history was unremarkable. She was a non-smoker and the family history was negative for hypertension.

The patient's blood pressure was not recorded on the prenatal record at her first visit on July 6 at 10 weeks 2 days. On her second visit at 15 weeks 5 days, her blood pressure was 130/90. Her blood pressure remained elevated during the pregnancy. On November 29 at 31 weeks 1 day, blood work was ordered for investigation of pre-eclampsia. The results of that test were normal. Her blood pressure was 145/100 on December 29 at 35 weeks 3 days. There was no proteinuria. Liver enzymes and platelet count remained normal. On January 2, her blood pressure was 160/120 and she was started on labetalol 100 mg, twice daily, after consultation with an obstetrician. The following day, the dose was increased to 200 mg, twice daily. After discussing the case with an obstetrician on January 3, the plan was to induce labour at 37 weeks gestation provided there were no signs of pre-eclampsia before then.

Non stress tests done on November 29, December 10 and 29, January 2, 3, 6 and 10 were normal. Ultrasound on December 29 at 35 weeks 3 days showed foetal growth

consistent with 34 weeks 2 days, Biophysical profile 8/8 with amniotic fluid index (AFI) 160 and normal uterine artery doppler flows normal. Ultrasounds on January 2 and 10 for amniotic fluid assessment were normal (AFI 122 and 96 respectively).

The patient was scheduled for induction on January 11 at 37 weeks 1 day, but was admitted in labour at 2355 hours on January 10.

Course in Labour and Delivery

At the time of admission to hospital, contractions were q2-3min. The patient's blood pressure was 149/100, cervix was 1-2 cm dilated with the vertex at spines -2. Foetal heart was normal. Membranes ruptured spontaneously at 0005 hours.

At 0045 hours, the patient was given morphine and Gravidol.

At 0330 hours, the cervix was 5-6 cm dilated and an epidural was placed.

The patient was assessed by her doctor at 0600 hours. The epidural was working well and her blood pressure was stable. Artificial rupture of membranes (ARM) was postponed pending results of the GBS. At 0900 hours, she was given 300 mg of labetalol for a blood pressure of 156/96. By 0905 hours, results were still not obtained. The cervix was 8 cm dilated and ARM was performed for clear fluid.

The patient was fully dilated at 1050 hours and started pushing shortly thereafter. After one hour of pushing, the presenting part was at spines -1 to 0 with the head was felt to be somewhat deflexed. The foetal heart tracing showed variable decelerations with contractions with good recovery and normal baseline between contractions. At 1220 hours, the GBS culture result was available and was positive, so the patient was given 2g ampicillin IV.

The obstetrician on call was consulted because of failure of descent despite good pushing efforts for 1 hour and 10 minutes and concerns about the head being deflexed. The obstetrician ordered oxytocin augmentation to start while awaiting his arrival. An epidural top up was given at 1233 hours, but was ineffective. The patient was then given fentanyl 100mcg IV.

A foetal bradycardia occurred prior to the arrival of the obstetrician. The patient was placed on her side, given oxygen and the oxytocin was discontinued. When the obstetrician arrived, it was decided to proceed with operative vaginal delivery for a non-reassuring foetal heart rate tracing. The vertex was 1 cm below spines in the left occiput transverse to left occiput anterior position. The anaesthetist was not available at this time and because the epidural was not working, a pudendal block was placed. The Kiwi cup vacuum extractor was applied and traction applied over the next three contractions. As it was difficult to maintain a seal during the third pull, the vacuum extractor was abandoned and Kjelland forceps were applied. The delivery summary does not mention the position prior to the forceps application, but rather indicates that during the application of the anterior blade, there appeared to be spontaneous rotation to what turned out to be a direct occiput-posterior position. With the next contraction and

minimal traction, she was delivered over a mediolateral episiotomy of a 2.2 kg (5 lb 2 oz) male infant at 1303 hours. Meconium was noted on delivery. There were no cord complications.

Postnatal Course

The infant was apnoeic and had no muscle tone or reactivity. A heart beat was documented at both one and five minutes, but the method of ascertainment was not stated. Resuscitation was initiated with bag and mask ventilation and cardiac compressions. There was no improvement in tone or colour. A “code pink” was called. Apgar scores awarded were 2 and 2 at one and five minutes. The baby was intubated by the respiratory technician at about 8 minutes of age and, in spite of correct placement and good lung inflation, the heart beat was lost. Cord arterial blood gases revealed severe acidosis with pH 6.94, pCO₂ 92mm Hg, HCO₃ 19.8 and base deficit 13.8mmol/L.

After arrival of the “code pink” team, ongoing resuscitative measures were continued. This included checking endotracheal tube placement and air entry, continued bag ventilation, cardiac compressions and epinephrine and atropine administered via the endotracheal tube. Multiple attempts to secure intravascular access were unsuccessful until the arrival of the paediatrician when the baby was 25 minutes of age. On the second attempt by the paediatrician, an umbilical venous catheter was placed. Intravenous administration of normal saline, epinephrine, sodium bicarbonate and atropine failed to restore the heart beat. The resuscitation was called off and the baby pronounced dead at 1340 hours at 37 minutes of age.

Post Mortem

Post mortem findings indicated a weight of 2.36 kg (vs. 2.20 kg from medical record) which was in the 5-10th percentile for 37.5 weeks gestation.

There were no congenital malformations.

Birth injuries were identified in three areas:

1. There was evidence of cranial trauma with marked hemorrhagic caput succedaneum, ruptured bilateral cephalohaematomas, tears in the left lambdoid and saggital sutures and a fracture of the right lateral portion of the occipital bone.
2. There was marked congestion and oedema of the brain with no evidence of acute intracranial or intracerebral haemorrhage.
3. There was acute bruising of the posterior neck and subcapitis muscles and anterior neck muscles.

The placenta had to be removed manually and showed extensive disruption. The weight of all placental material obtained was 310 g, which was in keeping with placental growth

restriction. There was evidence of recent mild meconium staining. There were no specific morphological features of impaired utero-placental perfusion.

The cause of death was stated as perinatal asphyxia due to undetermined cause with additional and/or potential contributory factors of birth injury in the form of cranial trauma and soft tissue injury to the neck and back.

Discussion

This infant died shortly after birth from an acute asphyxial event. Autopsy findings place the timing of this event within a matter of hours before delivery. There was evidence of birth trauma on autopsy, but it could not be determined to what degree, if any, these findings contributed to the outcome.

The American College of Obstetricians and Gynaecologists and the American Academy of Paediatrics have listed five criteria that collectively suggest an intrapartum timing (within close proximity to labour and delivery) of the event, but are non-specific to asphyxia insults. These are:

- A sentinel hypoxic event occurring immediately before or during delivery;
- A sudden and sustained foetal bradycardia or the absence of foetal heart rate variability in the presence of persistent late or variable decelerations, usually after an hypoxic sentinel event when the pattern was previously normal;
- Apgar scores of 0 to 3 beyond 5 minutes;
- Onset of multi-system involvement within 72 hours of birth;
- Early imaging studies showing evidence of acute, non-focal cerebral abnormality.

Review of the foetal heart rate tracing indicates that the nature of the tracing abruptly changed at 1030 hours following which there were decelerations and a changing baseline. Then at 1236 hours, after the attempted epidural top up, there was a prolonged foetal bradycardia. It cannot be determined if this started earlier as the tracing was lost starting at 1224 hours. The oxytocin augmentation had been started at 1215 hours. These findings, together with the Apgar score and the cerebral changes at post mortem, meet the criteria outlined above. In addition, the cord gas indicates a metabolic acidosis with a base deficit $>12\text{mmol/L}$. The one inconsistency in these findings is the last part of the foetal heart rate tracing which does not have the appearance of an ominous or terminal tracing. That aside, the appearance of the tracing starting at 1030 hours was such that an obstetrical consultation should have been considered at that time. Also, given the appearance of the tracing, consideration should have been given to notifying anaesthesia so that if an emergency Caesarean section became necessary, advanced planning could have taken place. The decision to start oxytocin augmentation at 1215 hours was ill-advised.

The birth trauma identified at autopsy, although significant, did not cause any gross cerebral traumatic injuries and therefore the fractures were unlikely to have impacted on

the outcome. The findings however, are consistent with the possibility that there was an undue amount of force applied in the form of compression from the forceps. Such compressive forces could have contributed to the cerebral oedema and congestion identified at autopsy. The circumstances surrounding the delivery procedure placed the baby at risk for birth trauma. The risk factors include mid-station failed vacuum followed by forceps and an incorrect, or lack of determination, of the position prior to application of the vacuum/forceps. The obstetrician's delivery summary indicates that there was satisfactory descent obtained with the vacuum as evidenced by the rectal emptying that occurred. This is not recognized as a determinant of the progress in descent of the presenting part. Also, the summary indicates that after the application of forceps, delivery occurred with the next contraction with very minimal traction. While it is possible that adequate descent had been obtained with the vacuum such that the forceps procedure was as easy as stated, the circumstances as noted above and the post mortem findings, are not consistent with this description.

A resuscitation team consisting of a RN and RRT was in attendance and provided appropriate resuscitative measures. A number of emergency physicians responded to the "code pink" call. There was some delay in contacting the paediatrician on call who did not receive a page until 20 minutes after delivery. Once called, the paediatrician attended quickly. Other than some difficulty with vascular access and medication administration that is somewhat out of step with current Neonatal Resuscitation Program guidelines, there were no concerns about resuscitation. It is clear that the infant was extremely compromised at birth and apparent that no change in post natal management would have resulted in a different outcome.

In order to ensure optimal neonatal resuscitation in the future, it may be helpful for providers charged with this responsibility to review NRP guidelines, especially in relation to this case. Specific deviations from current recommendations include treatment with atropine (via ETT or IV) or sodium bicarbonate. Providers should also review methods to access the umbilical vein with particular attention given as to how the stump is cut and the vessel opening exposed.

Recommendations

1. Obstetrical care providers are reminded that foetal well-being should be assured in the form of a normal foetal heart rate tracing prior to the commencement of oxytocin.
2. Obstetrical care providers are advised to be familiar with the current guidelines for action in the setting of atypical and abnormal EFM tracings.
3. Obstetrical providers should, to the degree possible given the resources within their hospital, coordinate activities with anaesthesia and paediatrics in advance if there is a high risk situation in the delivery room.
4. Obstetrical care providers are reminded of the importance of determining foetal position prior to performing an instrumental delivery.

5. The _____ Hospital should ensure that health care providers who may be called upon to resuscitate newborns should have up to date NRP provider status.

Case: 2009-N-8

History

The mother of the deceased was a 35 year old P3G2 whose pregnancy was uncomplicated until 34 weeks gestation. She had normal routine lab testing, IPS and ultrasound investigations. Her blood pressure was normal (125/68) in her obstetrician's office two weeks prior to her presentation at hospital.

The patient came to the hospital with abdominal pain at 34 weeks gestation. She was noted to have an elevated blood pressure of 160/92 and irregular contractions. Her lab tests were normal, except for a platelet count of 132. Fetal monitoring was normal. She was admitted to the antepartum ward and started on labetalol and given Celestone. Her blood tests were repeated daily. The platelet count initially rose to 150 and a 24 hour urine collection for protein showed 10 grams per day.

Forty eight hours after admission, the patient was brought in for induction of labour. She received Prepidil gel and oxytocin and ARM was done. She had an epidural and was also given magnesium sulphate. The baby delivered spontaneously after four hours of labour. The mother's post partum course saw development of mild hemolysis, elevated liver enzymes and low platelet (HELLP syndrome) and some pleural effusions, as well ongoing hypertension. She recovered spontaneously and was discharged on the sixth postpartum day with outpatient follow-up by her obstetrician.

The male infant was born in good condition with a birth weight of 2.16 kg. His size and appearance were appropriate for the gestational age of 34 weeks. No resuscitation was required and Apgar scores of 9 and 9 were given at one and five minutes respectively. He was transferred to the neonatal intensive care unit (NICU) for observation and further management.

The admission note indicates that the baby was a normal and healthy late preterm infant. There was a brief period of grunting respiration that resolved spontaneously. Blood glucose monitoring was commenced according to protocol and a slightly low bedside glucose value of 2.4 mmol/L was detected at just over one hour of age. A small feed of 6 cc of formula was given via nasogastric tube. There was no notation regarding the nasogastric tube insertion. A repeat glucose test 45 minutes later was lower at 1.9 mmol/L, so a bolus of D10W was given intravenously followed by an infusion of D10W at 7 cc/hr. There was one further episode of mild hypoglycemia at nine hours of age and a second bolus of D10W was given. There were no further low blood glucose measurements.

By the next morning, the infant appeared well, tolerating 9 cc of feed every three hours. The intravenous glucose was weaned. Over the next two days, feeds were gradually advanced and nipple feeding was attempted. There were no documented episodes of feeding intolerance or difficulty with nasogastric tube placement.

In the early morning hours of the third day of life, the baby suddenly deteriorated with recurrent apneic spells and lethargy. The pediatrician on call was informed and noted that the infant's abdomen was distended with erythema around the umbilical stump. There was some blood-tinged aspirate from the nasogastric tube. The pediatrician placed the baby on nasal continuous positive airway pressure (CPAP), started treatment with caffeine, performed a blood culture and started antibiotic treatment with ampicillin and gentamicin. Abdominal and chest x-rays were ordered and revealed free intraperitoneal gas indicative of gastrointestinal (GI) perforation. Necrotizing enterocolitis with perforation was suspected. The baby was ordered NPO (nothing by mouth). IV fluids were commenced and the children's hospital was contacted to arrange transfer. Metronidazole was added to the antimicrobial coverage.

The transport team arrived shortly thereafter and assumed responsibility. The abdomen was grossly distended and the baby was poorly perfused. An arterial blood gas revealed a mixed acidosis with pH 7.09, pCO₂ 66 mmHg, pO₂ 46 mmHg, HCO₃ 19.1 mmol/L and base deficit of 13.3 mmol/L. The infant was intubated and placed on mechanical ventilation. A normal saline bolus was given intravenously and he was started on fentanyl. The infant was transferred to the children's hospital for further management.

On admission to the children's hospital, the infant was described as pale and poorly perfused. The abdomen was grossly distended and erythematous. Abdominal x-ray revealed massive pneumoperitoneum. He was assessed as a "critically ill three day old 34 week gestational age infant with small bowel perforation of unknown etiology who was hemodynamically unstable with poor perfusion probably secondary to peritonitis." He was given additional fluid boluses and started on dopamine to support the blood pressure. He was seen by general surgery and taken to the operating room for an exploratory laparotomy.

At laparotomy, a large amount of gas was released and the peritoneal cavity was suctioned for a large amount of milky fluid. An isolated gastric perforation on the lesser curvature of the stomach was identified and oversewn. After irrigation, the abdomen was closed and the baby returned to the NICU in critical condition. The infant was continued on triple antibiotic therapy with ampicillin, tobramycin and metronidazole. He was hypotensive despite volume expansion with saline, albumen and plasma. He was started on inotropic agents and hydrocortisone and was switched to high frequency oscillatory ventilation. He received several infusions of sodium bicarbonate for refractory metabolic acidosis.

By the next day, the infant remained critically ill with evidence of multiorgan failure, DIC and shock. CBC revealed leukopenia and thrombocytopenia. The infant was unresponsive to ongoing aggressive interventions. The groin area and lower abdominal wall became discoloured with a blistered appearance suggestive of necrotizing fasciitis. A gram stain of fluid from one of the blisters in the groin revealed many gram negative bacilli. The antibiotic regimen was changed to clindamycin, penicillin G and meropenem. The infant continued to deteriorate and passed away early in the evening of January 29, 2008, at 5 days of age.

Bacterial culture from a groin blister and blood culture drawn shortly before death revealed a heavy growth of multiple antibiotic resistant E. coli, sensitive only to meropenem and amikacin.

Post Mortem

The significant findings at post-mortem examination included:

- acute gastric perforation, status post surgical treatment
- necrotizing fasciitis, involving abdominal wall, genitalia and groin region
- acute fibrino-purulent peritonitis
- hypoxic-ischaemic encephalopathy, severe, recent

Post mortem bacterial cultures of blood, lungs and peritoneum revealed a heavy growth of E. coli with the same multiple resistant strain as seen in the premortem cultures.

Death was attributed to “septic complications of acute gastric perforation.”

Discussion

This infant died from severe septic complications arising from a gastric perforation that developed on day 3 of life. He was born slightly preterm at 34 weeks gestation after induction of labour for maternal welfare. He was in good condition for the first two days when he suddenly deteriorated and was found to have free intraperitoneal gas indicative of a perforated viscus. He was stabilized and transported to the childrens’ hospital where an exploratory laparotomy was quickly performed. An isolated gastric perforation was found and oversewn, and the peritoneal cavity irrigated. The infant was returned to the NICU in critical condition and continued to deteriorate until he passed away two days later in spite of aggressive treatment. His clinical course was characterized by severe refractory hypotension, metabolic acidosis, DIC, leukopenia and multiorgan failure suggestive of septic shock. Necrotizing fasciitis developed over the abdomen. Bacterial cultures from multiple sites taken on the day of demise revealed a multiple antibiotic resistant E. coli.

Gastric perforation in a neonate is a rare, potentially fatal condition. Predisposing factors appear to be prematurity, gastrointestinal malformation and corticosteroid treatment. In most cases, perforation with an intragastric tube has not been implicated and the perforation appears to occur spontaneously, often around day 2-3 of life. Treatment involves surgical repair and supportive treatment. Published mortality rates are reported at approximately 50%.

For this infant, everything appeared to be going well until the perforation occurred on day 3 of life. There was no apparent difficulty with nasogastric tube insertion or position and the baby had been tolerating increasing feedings nicely. When he began to show signs of difficulty, the pediatric team at the initial hospital responded quickly and

appropriately. The gastrointestinal perforation was quickly detected and referral made to the children's hospital for ongoing management.

A gastric perforation was detected at laparotomy and was oversewn. The infant exhibited cardiovascular instability from the time of presentation which failed to abate with surgery. The infant was in critical condition after the surgery and steadily deteriorated from that point, despite aggressive intervention. The cause of death appeared to be systemic sepsis as a complication of the gastric perforation. The predominant organism recovered in clinical and post-mortem samples was a multiple drug resistant *E. coli* that would not have been effectively treated by the antibiotic regimens in place prior to the infant's final few hours.

Multiple antibiotic resistant organisms (ARO's) are becoming increasingly problematic in modern health care. Traditional antibiotic choices to cover certain conditions may be ineffective if ARO's are the responsible pathogens. In most instances, broad spectrum antibiotics are prescribed to cover the majority of agents responsible for the condition being treated. If the patient fails to respond, infection with an ARO should be considered, additional studies performed and a switch to an antibiotic that would be effective against ARO's considered. The use of such antimicrobial agents needs to be restricted, but should be considered in such a setting.

In this case, there is no report of the blood culture taken at the initial hospital and no indications in the notes from the children's hospital that results were found. There appear to have been no additional blood cultures drawn at the children's hospital until just before the infant's demise despite ongoing, refractory septic shock. Consideration of a change in antibiotic coverage did not occur until that time and the infant's illness was too far advanced to allow any recovery. Whether the ARO could have been detected in earlier clinical samples and whether a change in antibiotic coverage would have resulted in a different outcome, is uncertain. It is possible that earlier treatment with meropenem and amikacin could have resulted in an improvement in the baby's clinical condition.

Recommendations

1. Pediatric care providers are reminded that infants with refractory sepsis might be infected with an antibiotic resistant organism and standard antibiotic regimens might be ineffective. Empirical treatment with higher-level antibiotics should be considered under these circumstances.
2. Blood cultures and other appropriate clinical samples should be taken prior to institution of antibiotic treatment in neonates with suspected sepsis and repeated if the baby's clinical condition fails to improve as expected. A revision to the antibiotic regimen should be considered after samples are collected.

History

The mother of the deceased was a 35 year old G4P1. Her first child was born 19 years ago by Caesarean section at 36 weeks gestation due to fetal distress. Her second pregnancy ended as a miscarriage and her third was an ectopic pregnancy for which she had a left salpingectomy.

During this pregnancy, she was followed by an obstetrician from 14 weeks. She had two early ultrasounds which confirmed her dates and a genetic amniocentesis which was normal. The 18 week ultrasound revealed a posterior, low lying placenta which was clear at a 28 week, 2 days follow-up scan. This last scan was done seven days prior to her presentation in the labour room. The scan showed some dilation of one of the posterior horns of the baby's ventricles and referral to a tertiary centre was being arranged for further assessment. The remainder of the ultrasound was normal.

At 29 weeks and 2 days, the mother presented to the labour room with concerns of a pink discharge and cramping. She was assessed by the obstetrician on call who ordered an urgent ultrasound. The ultrasound did not reveal any anatomic concerns, but did note the internal os of the cervix to be open with a "small amount of umbilical cord extending into the cervix". The amniotic fluid volume was normal on this scan. A non-stress test showed a foetal heart rate baseline of 155-160 bpm, with moderate variability and accelerations, as well as some variable decelerations. The obstetrician examined the patient and felt the cervix was in fact 2 cm long. No notes were made about the presence of umbilical cord, nor was there any suggestion that rupture of membranes was likely. A swab was taken which later showed heavy growth of GBS. The patient was given the first of two Celestone injections and was discharged. She returned the following day for her second injection, but no notes from this visit can be located.

The patient presented again to the triage area of her level 2 centre the following day at 29 weeks and 4 days gestation, with concerns of possible preterm labour and possible rupture of membranes. She was assessed by another on call obstetrician about 40 minutes after her arrival. The on call obstetrician confirmed the rupture of membranes and found the patient's cervix to be 1 cm dilated. An IV and ampicillin was ordered for the patient and Criti-Call was contacted for transfer to a level 3 centre. EFM showed a baseline foetal heart rate of 155-160 bpm with minimal variability. Periodic decelerations were noted, but as the toco was not working well, these cannot be timed with uterine activity. An hour after her presentation, it was determined that there was no bed available to transfer her to at a tertiary centre. Ultrasound was completed and cord presentation was ruled out.

The patient was admitted to the labour room, two hours after her initial presentation. The tertiary paediatric transport team was contacted about attending the birth, but were not available for several hours. The foetal heart rate persisted at 155-160 bpm, with minimal variability and some decelerations which now appeared to be late in timing. Three hours after her presentation, the on call obstetrician was notified of this abnormal

foetal heart rate pattern. Four hours after her presentation, the mother was complaining of contractions every two minutes. The obstetrician was called, reassessed the patient, and decided to proceed with a Caesarean section. The patient was transferred to the operating room five hours after she came to the hospital.

Spinal anaesthesia was used and the paediatrician was in attendance during the Caesarean section. The baby was female and weighed 1.805 kg. The Apgar scores were 1 and 1 for heart rate only. A full code was run for 25 minutes during which time the baby was noted to have a distended abdomen and oedema. The baby could not be resuscitated and the efforts were stopped.

Post Mortem

This baby died of congenital toxoplasmosis with necrotizing pneumonia. This is a rare fatal congenital infection which is very difficult to diagnose antenatally due to very few maternal symptoms. However, this baby also suffered from birth asphyxia. The abnormal foetal heart rate was present for some time prior to the decision to proceed with delivery. The team was initially hoping to transfer the patient to a tertiary centre, then when it was confirmed that there were no beds, they attempted to arrange for a paediatric team to attend the birth.

Earlier delivery would not likely have changed the outcome; however caregivers did not know this prior to the birth, resuscitation and autopsy.

Recommendations

1. Obstetrical caregivers are reminded of the importance of acting on atypical and abnormal foetal status in preterm infants even while transfer is arranged.
2. The Ministry of Health and Long Term Care should urgently review the bed capacity for severely sick neonates needing transfer to neonatal units throughout the province. A review should also be conducted on the availability of transport teams that service neonatal units.

Case: 2009-N-10

History

The mother of the deceased was a 25 year old G4P3 with an EDD of October 4, 2008. Her LMP was December 29, 2007. She was seen at a walk in clinic on March 18 with bleeding. An ultrasound on March 20 was consistent with a viable pregnancy at 12 weeks 1 day. There was evidence of a subchorionic haematoma. A referral was made to an obstetrician.

The referral note indicated that the patient's medical history was significant for narcotic abuse related to the treatment of back pain stemming from a motor vehicle collision injury. She had been investigated for a pulmonary embolus in November 2007. Her past

obstetrical history included term pregnancies in 2000 and 2004. A third pregnancy delivered prematurely at 34 weeks in 2006. The baby tested positive for morphine at which time the patient admitted to being addicted to Percocet. She was placed on methadone. Following investigation by the Children's Aid Society, the children were placed with extended family. In October 2007, the patient was hospitalized for OxyContin and cocaine addiction, but discharged herself against medical advice. Subsequently, both the patient and her partner were required to engage in a drug treatment program and submit to drug screens. A number of the screens were positive. In March 2008, the couple entered a short term residential drug treatment facility and during this time it was discovered that she was pregnant.

The patient saw the obstetrician on March 31 at 13 weeks 1 day. She had not had any further bleeding. She was counseled with regards to genetic screening testing and this was declined. Risk factors identified included 1st trimester bleeding, smoking (3-4 cigarettes a day) and previous preterm delivery. Recent OxyContin use was documented, but not identified as a risk factor. Medical records from the investigation for the pulmonary embolus were obtained and the results were negative. The Antenatal Record 2 does not include test results for rubella, HBsAg, VDRL or HIV. The patient was asked to return in two weeks, but she did not keep the appointment.

The next prenatal visit occurred on June 9 at 23 weeks 2 days. SFH was appropriate and the foetal heart was heard. The patient denied any OxyContin use since 12 weeks gestation. There is no record of a routine 2nd trimester ultrasound being done.

On the morning of July 4, at 27 weeks, she presented to the emergency room complaining of abdominal cramps. She was found to be 2 cm dilated. She was treated for a urinary tract infection and discharged. She saw her obstetrician later that day and informed him about the cramping and urinary tract infection diagnosis. The cervix was found to be 1-1.5 cm dilated. She was given 12 mg of Celestone and instructed to return in 24 hours for a second dose. She used OxyContin at home for the pain.

Course in Labour and Delivery

The patient was admitted to hospital via ambulance at 0020 hours on July 5 in acute distress and complaining of rectal pressure. She was assessed by the obstetrician on call and was found to be fully dilated with bulging membranes. An artificial rupture of membranes was performed at which time a transverse lie was diagnosed. Preparations were made for emergency Caesarean section.

Under general anaesthesia through a classical Caesarean section scar, the patient was delivered of a male infant weighing 1.09 kg at 0055 hours. The presenting shoulder was noted to be bruised. There was evidence of lateral extension of the vertical scar. Apgars were 3 and 6 at one and five minutes. The baby was described as flaccid with extensive bruising to his face, head and trunk and showed no respiratory effort. Heart rate was less than 60 bpm. The paediatric team was in attendance. The paediatrician placed an endotracheal tube and commenced positive pressure ventilation. When the heart rate failed to improve, chest compressions were given for 15-20 seconds, the endotracheal

tube was replaced and positive pressure ventilation resumed. With this, the heart rate increased to approximately 120-130 bpm and the baby's colour improved. Apgar scores awarded were 3 at one minute and 6 at five minutes (although 2 points were awarded at five minutes for respirations when the baby was being artificially ventilated). Cord arterial blood gases revealed pH=7.10, pCO₂=79 mmHg, HCO₃=24 mmol/L, BE= -5 mmol/L. The infant was transferred to the Neonatal Intensive Care Unit (NICU) for further management.

In the NICU, heart rate was 188 bpm, oxygen saturation 82% on 100% oxygen with bag ventilation. Physical examination revealed bruising of the left shoulder, no dysmorphic features, good air entry and decreased muscle tone. The infant was placed on a ventilator and preparations were made for umbilical line insertion. Occasional gasping respirations were noted at 25 minutes of age.

Umbilical lines were inserted at 0140 hours, blood work was drawn and infusion of fluids commenced. Arterial blood gases revealed a marked metabolic acidosis with pH=7.19, pCO₂=33 mmHg, pO₂=37 mmHg, HCO₃=12 mmol/L. The haemoglobin concentration was normal at 156 g/L, but there was marked neutropenia with an absolute neutrophil count of only 260 per mm³. Glucometer reading was normal at 6.7 mmol/L. A blood culture was also sent. Initial arterial blood pressure was very low at 19/8 and a normal saline bolus was ordered. While the lines were being sutured, the baby exhibited an acute deterioration with desaturation and bradycardia to 77 bpm. Air entry to the chest was very diminished so the endotracheal tube was placed on traction. This resulted in an improvement in air entry and heart rate with a gradual increase in oxygen saturation. The transport team from the childrens' hospital had been alerted and arrived at 0203 hours, when the baby was just over one hour of age.

When the transport team arrived, the baby was being hand-bagged in 100% oxygen and had an oxygen saturation of 39%. After noting diminished breath sounds on the left side of the chest, the endotracheal tube was withdrawn and re-taped with subsequent improvement. With a switch to mechanical ventilation, the oxygen saturation improved and the baby tolerated some weaning in supplemental oxygen. Chest x-rays taken at this point showed the endotracheal tube with the tip at the carina and granular lung markings consistent with RDS.

At 0315 hours, FIO₂ had increased to 1.00 again. Point of care arterial blood gases showed pH= 7.05, pCO₂=57 mmHg, pO₂=30 mmHg, HCO₃= 15.7 mmol/L and BE=-15 mmol/L. Serum lactate was elevated at 10.3 mmol/L. Haemoglobin concentration was 133 g/L. A dose of surfactant was given via the endotracheal tube and a second normal saline bolus started. The infant tolerated the procedure fairly well with a drop in FIO₂ requirement to 0.46, but deteriorated again when the team attempted to transfer him to the transport ventilator. The umbilical arterial catheter was removed due to poor circulation to the lower limbs. Ampicillin and gentamicin were given intravenously.

From this point on, the infant's condition was very critical. He was ventilated manually with 100% oxygen. His arterial blood pressure was difficult to pick up, so an infusion of dobutamine was started. He was unresponsive with painful procedures. Just after 0600 hours, it was decided to transport the infant by ambulance to the childrens'

hospital. Air entry to the chest was described as equal and clear. Oxygen saturation during transport was documented at around 78%.

The infant arrived at the childrens' hospital just after 0700 hours. His chest was described as symmetrical with good air entry bilaterally. He was in extremis with bradycardia and low oxygen saturation. A "code pink" was called and aggressive resuscitative measures undertaken, including treatment with four doses of epinephrine, sodium bicarbonate, increase in dobutamine and cardiac compressions. Point of care blood work revealed a blood gas with improved pH and PCO₂, but ongoing base deficit. Haemoglobin concentration had dropped to 78 g/L. In spite of ongoing efforts, a normal heart rate could not be restored and the code was called off at 0743 hours. The baby was pronounced dead at 0800 hours.

Post Mortem

Significant findings were:

- evidence of recent birth trauma with bruising involving the left scalp from forehead to occiput, midline forehead and right outer canthus.
- bruising extending from the left neck onto the left shoulder and also the deep surface of the sternocleidomastoid
- left pneumothorax without tension, uneven aeration of the right lung and early bilateral focal hyaline membrane formation
- subependymal germinal matrix hemorrhage of the right frontal lobe extending into the right lateral ventricle and congestion with patchy multifocal subarachnoid hemorrhage

Examination of the placenta revealed severe chorioamnionitis and organizing subchorionic and basal plate haematomas. Nucleated erythrocytes were identified in the fetal circulation.

Toxicology studies were positive for oxycodone at a level of 0.26mg/ml. This level can cause death in non-tolerant adults.

The cause of death was given as complications of prematurity. Contributing factors were acute chorioamnionitis and chronic decidual hemorrhage.

Discussion

This infant died approximately seven hours after birth from complications of prematurity following premature labour and delivery at 27 weeks gestation. The pregnancy was at risk for premature labour based on a past history of premature labour, early pregnancy bleeding and maternal smoking. There was also a history of drug abuse. On the day the mother went into premature labour, she had presented on two occasions with the complaint of cramping; the first to the emergency department and the second to her

obstetrician. Although she was not diagnosed with premature labour, her cervix was 1-2cm dilated and her obstetrician was concerned enough to administer Celestone. Given her presentation and risk factors, she should have been considered for a period of close observation either in an obstetrical triage unit or admission to labour and delivery. It cannot be determined from this review if the emergency department of the hospital she presented to has a policy requiring such pregnant patients be assessed in an obstetrical triage unit. Also, if available at this site, fast foetal Fibronectin (fFN) testing may have been particularly helpful in identifying a significant risk of premature labour and the need for hospitalization and/or transfer to a tertiary care centre. It cannot be determined if any of these measures would have changed the outcome.

Although birth injuries were identified, the post mortem report did not find that they contributed to the baby's death. It is recognized however, that the baby can be at risk for serious birth injuries in the setting of prematurity and a transverse lie. It is most likely this consideration that lead the obstetrician to perform a classical incision in the uterus. The technical difficulty can further be increased by the lack/decrease of amniotic fluid. For this reason, with no presenting part in the pelvis, it would have been preferable to determine the foetal position using a portable ultrasound (if available) prior to the ARM.

This death was also reviewed by the Paediatric Death Review Committee (PDRC) as there was Children's Aid Society (CAS) involvement with mother and siblings of the deceased infant. The PDRC recommended that the report of the Maternal and Perinatal Death Review Committee (MPDRC) address the issue of the lack of transfer of information between physicians regarding the mother's drug use/abuse. Although the referring physician did identify the problem, the full extent of the history of the drug abuse was not realized by the obstetrician. This may have been more clearly identified and the history documented if the patient had been under the care of a family physician familiar with the patient's admission for drug abuse, entry into addiction treatment programs and CAS involvement with the children. It cannot be determined from this review why the patient did not have a family doctor, but possibilities include a physician shortage in the community and doctor-patient relationship breakdown due to drug seeking behaviour.

Although the OxyContin use on the day the mother presented in premature labour was not a factor in the neonatal death, it potentially could result in obstetrical care providers not appreciating the strength of the uterine contractions and the risk of premature labour.

The baby had severe respiratory failure with refractory hypotension and hypoxia despite intensive supportive measures provided by the paediatric team at the birth hospital, the transport team and the receiving team at the childrens' hospital. Babies born at 27 weeks gestation in good condition have very high survival rates (greater than 90%) with the receipt of modern neonatal intensive care. This baby's rapid downhill course suggests that there were one or more underlying conditions in addition to prematurity that resulted in a much more guarded outlook.

The most likely explanation for the baby's poor condition and outcome would be perinatal asphyxia. The infant was born in a depressed state, requiring aggressive

resuscitative measures. Although there was some difficulty extracting the baby at delivery and he was quite bruised, the poor response to resuscitation and intensive care suggests a more protracted intrauterine stressor. The chronic decidual haemorrhage may very well have resulted in chronic intrauterine hypoxia-ischaemia and foetal compromise. In any event, poor response to appropriately applied resuscitative measures and ongoing encephalopathy with poor responsiveness and hypotonia strongly supports a diagnosis of perinatal asphyxia. The cord arterial blood gases were only moderately deranged, but the first arterial gases from the baby clearly showed severe metabolic acidosis. The infant appeared to suffer from refractory hypotension which could certainly have resulted from acute myocardial ischaemia.

Another possible explanation for the baby's poor condition at birth and poor response to resuscitation and intensive care would be congenital sepsis. Microscopic examination of the placenta revealed severe chorioamnionitis; the baby suffered from refractory shock and the CBC showed profound neutropenia. These findings would support a diagnosis of sepsis. However, the blood culture taken from the baby at the time of line insertion showed no growth. Results of post-mortem microbiologic analysis are unavailable in the report provided for review, but the final report by the pathologist contains no mention of positive results.

The findings of elevated OxyContin levels at autopsy were indicative of recent use by the mother, but would have not contributed to the death of the baby. Toxic levels of narcotics present with respiratory depression and this baby was being appropriately treated for respiratory failure of any aetiology.

The significance of the pneumothorax detected on post mortem has also been raised. A chest x-ray taken at just over an hour of age showed no evidence of pneumothorax, so the air leak must have occurred at some point after this. There were a couple of instances in which an acute deterioration took place with a drop in saturation and heart rate which could have been indicative of a pneumothorax. Difficulty with air entry was described on several occasions, but manipulation of the endotracheal tube resulted in an improvement. At all other times, air entry was described as clear and equal, a finding that would be uncommon if a tension pneumothorax were present. The pneumothorax could not have been the root cause of the baby's problems, but may possibly have been a contributing factor to the terminal event. Pneumothorax should be considered in any ventilated preterm neonate with respiratory failure, especially after an acute deterioration. The team did not appear to consider this possibility. Transillumination or a follow-up chest x-ray, would generally be indicated in this setting. However, if a pneumothorax had been detected and treated earlier, it is very unlikely that the final outcome would have been different.

Recommendations

1. Obstetrical care providers are reminded to have a high index of suspicion for premature labour, particularly in the presence of risk factors.

2. The _____ Hospital should review its guidelines as to which pregnant patients presenting to the emergency department would best be assessed in a dedicated obstetrical triage unit.
3. Obstetrical care providers are reminded of the utility of foetal fibronectin testing in the assessment of possible preterm labour.
4. Paediatric care providers are reminded to consider the possibility of pneumothorax in any ventilated neonate who experiences an acute cardiorespiratory deterioration.
5. This report should be forwarded to the Chair of the Paediatric Death Review Committee.

Case: 2009-N-11

History

The mother of the deceased was a 28 year old G2P0 with an obstetric history that included a therapeutic abortion, with no complications, in 1998. She had her tonsils and adenoids removed in 1987 and the rest of her medical history was unremarkable.

Summary of Prenatal Care

The prenatal care was provided by an obstetrician from 10 weeks to approximately 35 weeks and a midwife from 36 weeks to 41 weeks. The woman's LMP was September 10, 2007, having just discontinued use of her contraceptive patch that same month. Her correct EDD of July 2, 2008 was determined by ultra sound at 7 weeks. The record indicates July 5, 2008 as the due date.

The mother of the deceased was blood group A positive with no antibodies. She was 157.5 cm (5' 2") and had a pre pregnant weight of 62.2 kg (136.8 lbs) and gained approximately 20.2 kg (44.4 lbs) throughout the course of the pregnancy. Her care was well documented and she was seen very frequently. Records indicate that the patient was seen ten times by her physician between 10 weeks - 34 weeks 4 days and five times by the midwife to 40 plus weeks.

The patient had negative first trimester and maternal serum screens, was rubella immune, HIV negative, Hepatitis B antibody positive and was syphilis negative. She had a history of negative PAP smears. Urine culture and microscopic at 30 weeks was negative. Swabs were negative for gonorrhoea and chlamydia. There is a record of glucose present in her urinalysis in 6 of 15 visits. Her random glucose at intake was 5.0 mmol/L and the 1 hour oral glucose challenge test was 5.9 mmol/L at 30 weeks. Hgb was 126g/L at intake and 104g/L at approximately 30 weeks. The patient had three ultra sounds. The ultrasound at 7 weeks 3 days indicated a July 2, 2008 due date. A nuchal translucency at 11 weeks 5 days gave a July 4 due date with a fundal placenta. A normal foetal morphology exam at 20 weeks 3 days indicated an anterior placenta and confirmed an EDD of July 5.

The care was transferred to a midwife at 36 weeks. The record indicates the patient was planning a home birth and was also hoping to give birth in water. She was GBS positive on swab at 37 weeks and declined intrapartum antibiotics stating she would consider it if additional risk factors presented during labour. At her last antenatal visit at 40 weeks 6 days on July 8, a vaginal examination revealed vertex at -1 spines, cervix 5 cm dilated and 60% effaced and show noted. She reported pre labour contractions and mucous discharge.

Summary of Labour and Delivery

July 8 - At 2030 hours on the day of the 40 week 6 day antenatal visit, the patient called to report contractions Q4 to 6 minutes for 60 to 75 seconds, talking and walking with back pain. She was given instructions to eat, rest and shower and call when contractions were 3 to 4 minutes or closer for an hour, or if rupture of membranes. At 2230 hours, she requested a visit from the midwife as contractions were Q3 to 4 minutes, stronger (but length not indicated) and more show with wiping. At 0210 hours, she was 5 to 6 cm dilated, 60% effaced and vertex -1 spines, bulging membranes and recorded notes say "not in labour". It was reviewed with the patient when and why to call. Foetal heart rate auscultated twice - 136 and 152 bpm, respectively. Her vitals were normal and scant brown show.

July 9 - The midwife checked in by phone at 1100 hours to find the contractions spaced out, irregular and the patient had slept six hours in the night. The patient was advised to eat, sleep, drink and call if regular labour contractions or rupture of membranes.

At 2130 hours, the midwife again checked in to find contractions still irregular, and the patient was frustrated. She was reassured that this was a normal "labour" pattern and that the baby was active, so comfort measures were recommended. She was informed that a biophysical profile was booked for later in week.

July 10 - At 0030 hours, at 41 weeks and 1 day, the patient paged the midwife with Q5 to 20 minute contractions and wanting to do castor oil. She was discouraged from doing this by the midwife as the patient was now tired. Foetal movement was noted and she was advised when to page.

At 0550 hours, the patient called to report contractions all night, Q5 to 7 minutes, and lasting 60 seconds. The midwife recommended a shower and nipple stimulation and to call back when contractions were stronger after timing them.

At 0700 hours, the patient paged the midwife to report contractions at Q7 to 11 minutes apart. She was advised to eat, drink, rest and call back with stronger and more regular contractions. Foetal movement was noted.

At 1930 hours, the midwife checked in to find the contractions getting stronger after the patient took castor oil (no amount indicated in the chart) and evacuating her bowels. The patient and the midwife planned to check in over the next hour to ensure contractions kept coming and getting stronger. The patient was pleased that things were finally underway.

At 2100 hours, the patient paged the midwife to report active labour. The midwife advised that she would attend with the student and then she reported to the backup midwife.

On arrival at 2200 hours, the patient was noted to be laboring well and in active labour. The foetal heart was 152 bpm. At 2205 hours, there was a spontaneous rupture of membranes and the foetal heart was 150 bpm after rupture for clear fluids with vernix. Vaginal examination showed the cervix to be 9 cm dilated with the head well applied to the cervix. Foetal heart was normal with accelerations noted and it was being taken Q5 to 15 minutes apart. The birthing pool was filled at 2230 hours. The patient entered the pool at 2245 hours and the foetal heart was 140 bpm, with accelerations noted.

At 2315 hours, the foetal heart was 180 bpm, with acceleration. A vaginal examination revealed a fully dilated cervix with the head at +2 spines. The foetal heart was checked at Q2 to 5 minute intervals between auscultations with accelerations noted. The patient was labouring on the birth stool in the pool when the back up midwife arrived.

At 2340 hours, the head was visible. At 0002 hours, descent was noted; the foetal heart was normal and was last indicated at 0020 hours with birth following at 0026 hours.

The baby boy was born limp and non responsive. He was removed from the water and resuscitation commenced immediately. Meconium followed the birth.

At 0028 hours, Emergency Medical Services (EMS) were notified and the midwife making the call requested an intubation-capable paramedic.

The placenta was delivered at 0030 hours and appeared complete and normal on examination .

Resuscitation of the Newborn and Postpartum Care

Immediately after birth, the cord was clamped and cut and infant resuscitation commenced. There was no heart rate and no respiratory effort and the infant was limp with poor colour. Positive pressure ventilation and cardiac compressions were initiated immediately at 0027 hours and a heart rate was noted at one minute after birth. The infant was stimulated and suctioned and positive pressure ventilation continued until the EMS arrived at 0034 hours. Apgars at 1 minute was 2 (for heart rate) and at five and ten minutes was 3 (2 for heart rate and 1 for colour).

Cardiac compression commenced at 0027 hours and was halted at 0032 hours as heart rate > 100 bpm and the infant was pink. EMS arrived and after assessing the infant, decided not to intubate as there was a good heart rate, the air entry was good and the infant was pink. Positive pressure ventilation was continued until admission to the childrens' hospital at 0055 hours. The infant was intubated and stabilized and blood and diagnostic tests were undertaken. The infant had severe metabolic acidosis with no spontaneous respiration or movements noted until 0214 hours. Seizure activity had commenced at approximately 0150 hours.

Despite aggressive attempts at resuscitation, the infant did not respond. The infant was extubated at 1415 hours and died at 1450 hours on July 11, 2008.

The parents of the infant refused further contact with midwives at 0500 hours on July 11, 2008. The parents demanded that the birthing pool be removed from their home.

Post Mortem

Weight 3.74 kg, length 53cm, head 36 cm, chest 35 cm, abdomen girth 32 cm.

The infant died from cerebral hypoxic ischemic injury occurring around time of delivery.

The infant had a cerebral global hypoxic-ischemic injury with cerebral oedema and widespread petechial hemorrhages and systemic hypoxic ischemic injury – mild - with hemorrhagic infarction of adrenal glands, mucosal hemorrhage and central zone hepatocellular swelling of right lobe of liver.

As the child survived for several hours, it is not possible to determine from the autopsy results if fluid from the birthing pool was aspirated.

On examination, the placenta was term with mild villus dysmaturity, mild meconium exposure effects and excessively coiled cord with a false knot and an accessory lobe.

Discussion

The mother of the deceased was in communication with the midwives several times from the antenatal visit on July 8 until their arrival for the birth on July 10 at 2205 hours. Throughout July 9, the mother experienced irregular, painful contractions. The midwife felt that she was not in active labour and advised her to carry on. The numerous calls regarding the “irregular” labour pattern should have prompted an in-person visit early on July 10. The 0550 hours call to confirm foetal status should have been through heart tones, not just the report of foetal movements given over the phone. The patient should also have had a vaginal assessment and the labour status should have been addressed beyond, “rest, eat and await more regular contractions.” The labour may in fact have been prolonged and not just, “not active”.

Because of the mother’s GBS positive status, her cervical dilation, bulging membranes, gestation, primigravida status and her contraction pattern, she should have been augmented in her labour.

If the membranes had been ruptured as a form of augment on July 8 at midnight or July 10 at 0530 hours, the labour may have become more regular in its contraction pattern and could have brought on the active labour sooner and possibly affected the outcome

The record notes a discussion at the second visit on July 8, regarding the reluctance to rupture because of the GBS status. The mother was in the birth pool from 2245 to 0026 hours - a total of 1 hour and 41 minutes. Her membranes ruptured spontaneously at 2212 hours and the birth was at 0026 hours - 2 hours and 14 minutes later. The mother had a normal temperature on July 8 at midnight and July 10 at 2214 hours.

The foetal health surveillance commenced at 2205 hours on July 10, 2008 and appears to be in accord with the guidelines for intermittent auscultation in active labour and during active pushing of second stage. However, the length of the auscultations of the foetal heart is not indicated and there is no information regarding decelerations being present or absent.

The irregular contractions are used as the single measure of this patient's labour being active when, as a primigravida at 5 to 6 cm, she fits the criteria for a woman in active labour with respect to the openness of her cervix. On July 9, this patient should have been assessed at the hospital and a NST performed as a means of foetal surveillance. This woman was experiencing a very desultory and irregular labour pattern that was actually opening her cervix. The outcome might have been different if an augment was undertaken and the birth had occurred sooner. This condition persisted for 48 hours prior to the midwife saying she was finally in active labour. The patient should have had a cervical exam on the July 10 at 0530 hours when the contractions were Q5 to 7, lasting 60 seconds.

When the infant was born flat, no cord gases were taken.

Recommendations

1. Midwives should collect cord gases at all births.
2. Obstetrical care givers are cautioned from adhering to a strict definition of the latent phase of labour. Long, painful contractions "despite irregularity" that cause significant cervical change should be regarded as active labour. Considerations of care for a woman in active labour include assessment of progress, foetal surveillance and appropriate interventions provided in a timely manner.

Case: 2009-N-12

History

The mother of the deceased was a 32 year old G2P0 with an EDD of delivery of September 15, 2008 as determined by LMP and early ultrasound. Her routine prenatal laboratory investigations and second trimester ultrasound were normal. GCT was abnormal at 8.2 and a subsequent GTT was normal. A repeat ultrasound at 32 weeks to reassess placental location was normal.

GBS culture on August 18 at 36 weeks was not reported. Blood pressure on that visit was mildly elevated, but was subsequently normal. The GBS culture repeated on August 28 at 37 weeks 4 days was negative.

Past medical history included a pregnancy termination in 1997.

A home birth with midwives was planned.

Course in Labour and Delivery

The mother paged her midwife on September 7 at 0800 hours at 38 weeks 6 days, with query ruptured membranes and no contractions. The midwife advised her to call back with regular contractions and discussed consult timing of 24 hours after rupture of membranes and in light of GBS negative status.

The midwife visited at 1600 hours and assessed the mother. The maternal temperature was normal and foetal heart rate was in the 150's. The cervix was a fingertip dilated, 40% effaced with the vertex at sp-3. A discussion occurred as to when stimulation of labour should be considered. The plan was to check back in with the midwife before 2200 hours.

At 2100 hours, the mother contacted the midwife and updated that the contractions were every 7 minutes, lasting 45 seconds and she was coping well. She was instructed to call back when the contractions were every 5 minutes.

On September 8 at 0130 hours, the midwife attended after being paged and informed that the contractions were every 5 minutes. On assessment, the foetal heart was normal. The cervix was 2 cm dilated, 70% effaced with the vertex at sp-3. Contractions were getting stronger. At 0530 hours, the cervix was 2-3 cm and the plan was to reassess at 0730 hours and decide if consultation would be required for prolonged rupture of membranes.

At 0730 hours, the cervix was 3-4 cm and 90% effaced. An obstetrician was consulted by phone at 0800 hours. The mother was given the choice of staying at home for another 2-4 hours or transferring to hospital. The mother elected to continue at home in a birth tub. She remained afebrile on reassessment at 1000 hours. Contractions were every 2-3 minutes and the foetal heart rate was normal. At 1120 hours, the cervix was 4 cm dilated and contractions were strong. The decision was made to proceed to the hospital. The obstetrician on call and the Labour and Delivery Unit were notified.

The mother was admitted to hospital at 1220 hours. An obstetrical consultation was obtained and care was transferred. Contractions were q 2-3 minutes lasting 70 seconds. The cervix was 5 cm, dilated, 100% effaced with the vertex at spines -1. The foetal heart rate was in the 150's. Maternal temperature was 36.3 °C, haemoglobin 133 gm and WBC 30.1.

At 1255 hours, she was given Demerol 100 mg and Gravol 50 mg.

The mother was reassessed at 1530 hours and the cervix was 6-7 cm. The foetal heart showed occasional decelerations with good variability. A foetal scalp clip was applied. An epidural was placed at 1548 hours. At 1550 and 1559 hours, a deceleration to 90 occurred. At 1615 hours, the obstetrician was informed of late decelerations. On re-examination, the cervix was 8 cm dilated. At 1632 hours, she was thought to be fully dilated and was told to start pushing, but on recheck at 1640 hours, an anterior lip was felt and she was told to stop pushing. In light of the concerning foetal heart rate tracing and as delivery was not imminent, the decision was made at 1645 hours to proceed to Caesarean section.

The mother was taken to the Operating Room and administered a general anaesthetic. A female infant weighing 3585 gm was delivered at 1720 hours. Apgars were 1, 1 and 1 at one, five and ten minutes. Meconium was noted at delivery. The cord venous pH was 7.0 and BE -13.5.

The baby was unresponsive, with no spontaneous respirations and cyanotic. She was bradycardic with a heart rate of 40-60 bpm. She was given oxygen by bag and mask with no improvement. Cardiac compressions were started at 30 seconds and an oral airway was inserted to facilitate higher pressure ventilation. A "code pink" was not called since there were two nurses, one RT, an anaesthetist and a medical assistant in attendance.

Despite administering CPR, there was no response. The infant was intubated with a size 3.5 endotracheal tube at two minutes of age. There was good visualization of the cords and no CXR was done. The baby did not respond and pulses were not palpable, consistent with no cardiac output. An interosseous needle was placed in the right tibia and 3 ml of 1:10,000 Epinephrine was injected followed by 20 ml of normal saline. A nasogastric tube was inserted with no significant aspirate. Thick secretions were noted emanating from the ET tube requiring suctioning.

An umbilical vein catheter (UVC) was not in place, so 6 ml of 1:10,000 epinephrine was given via the ET tube. There was still no palpable pulse. Once a UVC was in place at 1750 hours, 3 ml of 1:10,000 epinephrine was given followed by 5 ml of NaHCO₃. A further 20 ml bolus of saline was given.

The infant was connected to a cardiac monitor and a profound bradycardia was noted. CPR was still in progress. The ET tube position was checked and 5 ml of surfactant was given. There was some improvement in the lung compliance, but no change in the heart rate. A second dose of NaHCO₃ and 5 ml of 10% dextrose were given, again with no response. After a fourth dose of epinephrine, there was a transient rise in the heart rate to 60 bpm. Pulses were still not palpable. The UVC was replaced and blood work and cultures obtained. At this point, CPR had been ongoing for 30 minutes. There were no spontaneous respirations, no spontaneous movements and profound bradycardia. The infant was unresponsive to epinephrine and NaHCO₃ infusions.

CPR was continued while the findings and failure of response to resuscitation was discussed with the family. At that point, CPR was discontinued and the baby pronounced dead at 1811 hours.

Post Mortem

Post mortem examination revealed severe diffuse acute bronchopneumonia in both lungs. Cultures from cord blood, cardiac blood and the lungs grew beta-hemolytic Streptococcus group B.

The cause of death was prolonged rupture of membranes with ascending infection, acute chorioamnionitis and foetal sepsis.

Discussion

This infant died from Group B Streptococcus sepsis. The mother had been screened at 36-37 weeks gestation in keeping with Society of Obstetricians and Gynaecologists of Canada guidelines for the management of Group B Streptococcus. The first culture done at 36 weeks had to be redone ten days later. It is not clear from the record why a second culture was necessary. The second culture was subsequently reported as negative. As such, this would be categorized as a false negative culture.

A recent study found that 61.4% of term infants with group B streptococcal disease were born to mothers who were GBS negative. The factors identified that contribute to false negative results were: screening more than 5 weeks before delivery, the collection of specimens, the processing of cultures and the recording and reporting of screening results.(1) It cannot be determined by this review if any of these factors were applicable in this case, although the fact that the culture had to be repeated suggests there may have originally been an issue with the collection of the specimen or the processing of the culture.

Prolonged rupture of membranes (>18 hours) is a significant risk factor for chorioamnionitis and neonatal sepsis. In this case, membranes had ruptured approximately 33.5 hours prior to delivery. The clinical diagnosis of chorioamnionitis is typically based upon the presence of an elevated maternal temperature (>38 degrees C) and the presence of at least two of the following: maternal tachycardia, foetal tachycardia, uterine tenderness, foul-smelling amniotic fluid or maternal leukocytosis. Maternal leukocytosis with a WBC of 30.1 at the time of admission to hospital was the only finding in this case. Based on these clinical criteria, there was no evidence of chorioamnionitis requiring antibiotic treatment. Prolonged rupture of membranes in the absence of clinical chorioamnionitis has been considered as an indication for prophylactic antibiotics.

The American College of Obstetricians and Gynaecologists Committee Opinion #279 (December 2002) recommends antibiotic prophylaxis when membranes are ruptured >18 hours. The Society of Obstetricians and Gynaecologists of Canada recommends treating prolonged rupture of membranes greater than 18 hours only if the Group B Streptococcus status is unknown.(2) Given the length of time that the membranes had been ruptured prior to her presentation to hospital, following the American College of Obstetricians and Gynaecologists guideline may not have changed the outcome in this case.

The management of term premature rupture of membranes remains controversial. The options are to either induce labour, or to monitor for chorioamnionitis while awaiting the spontaneous onset of labour. Recent evidence suggests that routine induction of labour is associated with a small reduction in maternal and neonatal infection without an increase in the Caesarean section rate.(3) In addition, the Term PROM study showed that neonates of women who were managed expectantly at home were more likely to be diagnosed with infection.(4) Women with term PROM planning a home birth should be

advised of this evidence and obstetrical consultation and assessment advised if membrane rupture is prolonged. Given that there are no studies specifically addressing the risk of GBS infection with prolonged PROM and a negative GBS culture, it cannot be concluded that obstetrical intervention would have changed the outcome in this case.

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Recommendations

1. Women planning a home birth should be informed of the risks of infection with prolonged rupture of membranes.
2. Obstetrical care providers are reminded that cultures for GBS may give false negative results which could be due to collection, transporting, processing and reporting results. Other possible causes are collection longer than 5 weeks before delivery, and transient colonisation.

Case: 2009-N-13

History

The mother of the deceased was a 37 year old G2P0. Her LMP was on December 23, 2006, but she had a history of irregular periods. An ultrasound on April 23, 2007 gave a gestational age of 18 weeks 6 days and the EDD was September 18, 2007. Routine prenatal laboratory investigations and GCT were normal. GBS culture on August 23 at 36 weeks, 2 days was positive.

The patient's past medical history was non-contributory and she was a non-smoker.

During her prenatal visit on July 24 at 30 weeks 1 day, her family doctor noted poor weight gain and concerns about growth based on SFH measurement. Prenatal care was transferred to an obstetrician. She was seen on August 2 at 33 weeks 3 days. Her blood pressure was normal and SFH was 33 cm.

Subsequent visits on August 23, August 30 and September 6 showed SFH measurements of 34 cm, 35 cm and 34 cm respectively. An ultrasound done on September 7 at 38 weeks 3 days gestation, gave an EFW of 2268 gm (<10th percentile by LMP), with an increased HC:AC ratio of 1.15 (normal 0.93-1.11). Subjectively, there was oligohydramnios. The report was faxed to the ordering physician on September 10. The results are recorded in the Antenatal Record 2 as “gestational age 35, vertex”, with no reference to the growth parameters or oligohydramnios. Subsequent visits on September 13 and 20 record SFH measurements of 35 cm and 35 cm. The cervix was closed. Induction was scheduled for September 24th.

Course in Labour and Delivery

The patient presented to Labour and Delivery at the health care centre on September 22 at 0545 hours at 40 weeks 4 days gestation, in active labour. Contractions had started on September 21 at 1300 hours and became regular at 0330 hours. Membranes spontaneously ruptured shortly after arrival at the hospital. There was thick meconium. On examination, the cervix was 9.5 cm dilated. Continuous electronic foetal monitoring showed deep decelerations to 60 bpm during contractions with recovery to the baseline after contractions. A decision was made to expedite delivery because of the non-reassuring tracing. The paediatrician was notified.

At 0555 hours, she was fully dilated. The vertex was at sp+2. With the paediatrician in attendance, the vacuum was applied and with the next contraction and the baby was delivered easily with one pull at 0606 hours. The cord was loosely around the neck X1. The birth weight was 2045 gm. Apgars were 3, 4 and 6 at one, five and ten minutes. Cord gases showed a venous pH 6.93 and BE -16mmol/L.

A “code pink” was called. The baby was intubated and suctioned for meconium. The baby was re-intubated and given positive pressure ventilation and chest compressions. The heart rate came up to 124 bpm, but the baby continued to have intermittent sustained gasps. An umbilical vein catheter was placed and the baby was given a bolus of 30cc normal saline then 6cc D10W. At approximately 30 minutes, the heart rate slowed. Chest compressions were re-instituted and three doses of epinephrine were given. The heart rate did not improve and at 46 minutes, the resuscitation was discontinued. The baby was pronounced dead at 0652 hours on September 22, 2007.

Post Mortem

At autopsy, there was evidence of intrauterine growth restriction with growth parameters below the 5th percentile. Examination of the lungs showed marked meconium aspiration. There was evidence of subacute/chronic intrauterine physiologic stress with increased extramedullary haematopoiesis and adrenal cortex lipid accumulation. CNS examination showed marked cerebral congestion and edema with swelling. There was minimal birth injury with hemorrhagic caput succedaneum and bilateral thin cephalohaematomas.

The placenta was small for gestational age with villus dysmaturity and hypervascularity and marked changes of meconium exposure. There was hypercoiling of the umbilical cord (coiling index 1.1 vs. normal 0.2) and the cord was mildly constricted at the insertion into the placenta.

The cause of death was perinatal asphyxia.

Discussion

This infant died shortly after birth as a result of perinatal asphyxia. There was evidence of placental insufficiency as the underlying cause given that the baby was small for dates. There was evidence of extramedullary haematopoiesis, the placenta was small and dysmature and there was chronic meconium staining. The degree of placental insufficiency was such that placental gas exchange would be severely compromised during the added stress of labour resulting in foetal and neonatal asphyxia.

Severe IUGR was identified on ultrasound on September 7 at 38 weeks 3 days. The Antenatal Record 2 does not indicate that this report triggered any concerns by the obstetrician. Normally delivery would be indicated given these findings in a term pregnancy. It cannot be determined by this review why the pregnancy was allowed to continue.

The autopsy report identified the hypercoiling of the umbilical cord as being associated with intrauterine growth restriction, foetal intolerance of labour, chorioamnionitis and intrauterine foetal demise. This, together with the cord constriction at the insertion to the placenta, could also be a cause of, or contribute to, the perinatal asphyxia. Birth injury was not a factor.

Recommendations

1. Obstetrical care providers are reminded that ultrasound findings of severe IUGR at term is an indication for urgent delivery.
2. The _____ Hospital should do a QCIPPA review of this case and inform the Regional Coroner of any changes to policy and procedures based on the results of the review. The Regional Coroner will update the committee on the report from the hospital review.

Note: The hospital reviewed this death and education directed specifically at the obstetrician involved and at the departmental level was completed.

History

This baby died approximately 10 hours after an emergency Caesarean section was performed for foetal bradycardia and vaginal bleeding after amniotomy. The operative report indicated a complete placental abruption.

The mother of the deceased was a 33 year old G5P2A2 with no medical problems. She had two previous uncomplicated vaginal deliveries in Argentina, and a spontaneous and therapeutic abortion. She had an uncomplicated pregnancy. Her prenatal care was consistent and documented. Her prenatal investigations were normal.

The mother's ultrasound revealed a large subchorionic haematoma inferior to the gestational sac measuring 13 x 2.5 x 1.1 cm. The morphology ultrasound showed normal foetal anatomy, but a low lying placenta and "periplacental vessels at the edge of the placenta extending to the internal cervical os." In addition, there was a "single venous structure passing across the internal os separate from the umbilical cord." At 32 weeks, the placenta was clear from the os by 5 cm. A single vessel with venous flow was seen 1.6 cm from the internal cervical os. At 35 weeks, the ultrasound appearance was the same. There was no antenatal bleeding. The presence of these ultrasound findings is noted on the antenatal forms, but the submitted antenatal forms do not record the visits after 33 weeks.

On September 8, 2007, at 39 weeks gestation, the mother presented to hospital in early labour. The foetal heart rate tracing was reactive. At 4 cm, following rupture of membranes, a large quantity of fresh blood was noted. A foetal scalp clip was applied and detected bradycardia. An emergency Caesarean Section was performed and a complete placental abruption noted. The baby was born 15 minutes after the amniotomy at a weight of 3.56 kg. Apgars were 0, and 0 at one and five minutes. The venous cord gas and hemoglobin were normal. There was not a sufficient sample for an arterial result.

Post Mortem

Autopsy findings were consistent with an acute perinatal hypoxic/ischemic injury. There was no morphologic evidence of abruption. There was a velamentous cord insertion and dilated intramembranous vessels with no evidence of injury to any of these vessels at the initial placental examination. There was a perivascular haematoma in the cord, but the timing of this iatrogenic injury could not be determined and may have occurred with delivery of the placenta.

Discussion

This is a case of a neonatal death following an acute hypoxic/ischemic event in early labour. Following what appears to be an uncomplicated amniotomy, there was considerable vaginal bleeding, and then foetal bradycardia. At Caesarean section, a

complete abruption was documented clinically. There were no pathologic features of abruption or placental pathology, but these are not always present despite clinical evidence of abruption.

This presentation is also consistent with a vasa previa and inadvertent rupture of a vessel free within the membrane. Foetal exsanguination quickly ensues. In this case, while the baby was born hypovolemic, the foetal Hgb after 50cc of blood (moderate transfusion volume) was 157g/l, which does not seem consistent with rupture of a foetal vessel and profound anemia leading to bradycardia and the hypoxic insult.

The nature of the discussion about the persistent vessel in close relation to the cervix is not known. It is unclear whether the on call physician was aware of the ultrasound findings. The ultrasound reports do not actually call the findings a vasa previa, but they certainly imply the potential risks of a vessel in the membrane close to the cervical os. Documentation of the cord insertion into the placenta and antenatal findings of a velamentous insertion would have increased the suspicion of a vasa previa. Elective Caesarean section should have been considered and documented given the ultrasound findings.

Recommendations

1. Obstetrical care providers are reminded of the new SOGC guidelines for the diagnosis and management of vasa previa (JSOGC 31:8, 2009).

Case: 2009-N-15

History

The medical history for the mother included a severe penicillin allergy and mild scoliosis. Her family history included a sister with a baby with neural tube defect and parents on both sides had diabetes and hypertension. The mother was a G2P0 with a history of miscarriage followed by a D & C in 2005, with no complications from the general anesthetic. Her gynecologic history included a normal PAP test in 2007.

Summary of Prenatal Care

The mother, age 28, was O positive and was 5'2" with a BMI of 29.8 at 16 weeks gestation. She gained 55 pounds in the pregnancy. She was planning a hospital birth and began care at 11 weeks gestation. Her LMP of April 21, 2006 was regular, but not certain and an ultra sound at 11 weeks 6 days and 18 weeks 1 day, all agreed by a few days with an EDD of January 25, 2007. The mother was HIV, Hepatitis B and syphilis negative and was rubella immune. She had a negative IPS screen. Her blood pressure and urinalysis were normal throughout the pregnancy and her SF measurements corresponded to the gestation.

At 11 weeks gestation, the record indicated normal hemoglobin. A 1 hour 50g OGCT was declined. She was GBS negative on swab at 36 weeks 6 days. She was seen on a routine prenatal schedule and at 40 weeks 3 days, she was examined and found to have a long posterior cervix that was a fingertip dilated and not able to stretch.

Summary of Labour, Delivery and Postpartum

On January 30, 2007 at 1140 hours, the patient paged the midwife to report contractions. She was assessed at the midwifery clinic at 1215 hours and contractions were noted to be Q5 to 10 minutes, lasting 30 seconds, cervix 2 cm, 40 to 50% effaced, vertex -2 spines, membranes intact with mucous and show were noted. The patient's vital signs were normal, foetal heart was normal and there were foetal movements, so she returned home to await active labour. At 1515 hours, the patient paged the midwife with contractions every 3 minutes, lasting one minute and was seen for triage assessment at the clinic at 1545 hours with normal vital signs. Her cervix was 5 cm dilated, 50% effaced, vertex -1 spines and membranes intact. Foetal movements were present and foetal heart rate was normal.

The patient was admitted to hospital at 1600 hours in active labour. At 1845 hours, she was 7 cm dilated, followed by ARM for a small amount of clear fluid. She was fully dilated at 2050 hours and spontaneously pushing at 2110 hours. She used nitrous oxide for pain relief in active labour. The foetal heart rate was normal from admission to delivery with a baseline rate change occurring at approximately 1715 hours, from 140 to 120 bpm, then again at 2135 hours from 120 to 155 bpm. At 2222 hours, a non-responsive 3300 gram male infant was born at 40 weeks and 5 days gestation. The infant was described as flaccid, with no respiratory effort and there was no detectable heart rate.

The tight nuchal cord was clamped and cut on the perineum to facilitate the birth. Resuscitation with bag and mask ventilation and cardiac compressions was commenced. The delivery suite nurses were alerted and the respiratory therapist and paediatrician were paged. The nurses came to aid with the resuscitation and the respiratory therapist arrived six minutes after delivery. The placenta delivered at 2228 hours and was noted as meconium stained. The baby was intubated and his heartbeat became detectable. The paediatrician arrived when the infant was 13 minutes of age and noted that the baby was being ventilated through the endotracheal tube, but was still blue in colour with an arterial oxygen saturation of only 80%. Apgar scores reported by the paediatrician were 0, 0 and 2 at one, five and ten minutes. A cord blood pH of 6.64 was reported in the final note. A chest x-ray was taken showing clear lungs and a normal cardiac silhouette. The endotracheal tube position was adjusted. Vascular access was obtained and a bolus of normal saline was administered.

Although the notes provided for review are incomplete, it appears that the baby's colour improved, but he did not exhibit spontaneous respirations until after 60 minutes of age. A capillary blood gas taken at approximately one hour of age revealed pH=7.04, HCO₃=10 mmol/L with a base deficit of 21 mmol/L. Ampicillin and gentamicin were

administered. A call was placed to the tertiary hospital and a transport team was dispatched.

The transport team arrived when the baby was approximately four hours of age. The baby was poorly responsive and some seizure activity was noted. An infusion of dopamine was started to support the blood pressure. Anticonvulsant treatment was commenced, but the seizures were difficult to control. The infant required treatment with phenobarb, Dilantin, lorazepam and midazolam before the seizures ceased. The infant was ventilated and transported at approximately ten hours of age.

The baby was assessed by members of the critical care team and the consultant neurologist. They all concurred that he demonstrated evidence of severe hypoxic-ischaemic encephalopathy. There was also evidence of myocardial, renal and hepatic injury. Neuro-imaging and electrophysiological studies supported the diagnosis of severe HIE with poor prognosis. Although there was gradual improvement in end-organ function, the infant remained neurologically obtunded. Over a period of several days, multiple discussions were held with the family to explain the findings and prognosis. This resulted in a decision to withdraw life-sustaining medical therapy. The infant was extubated on February 5, 2007 and passed away a few hours later.

Post Mortem

No postmortem examination was done.

Death was attributed to hypoxic-ischaemic encephalopathy as a consequence of cardiac arrest during delivery with the umbilical cord around the neck.

Discussion

This baby died at six days of age after withdrawal of life-sustaining medical therapy. The infant had suffered a severe hypoxic-ischaemic insult in the perinatal period and showed evidence of profound CNS injury. The cause of the hypoxic-ischaemia is not certain, but cord occlusion may have played an important role. It appears that the foetal assessments in the hours before delivery were reassuring and the depressed condition of the infant at birth was a surprise to all in attendance. The cord was found to be tightly wrapped around the baby's neck when the head was being delivered and it was necessary to clamp and cut the cord before further descent could occur. Although this is not an uncommon finding in normal deliveries, it is possible that, with descent of the head, umbilical blood flow to the baby was compromised resulting in acute, total asphyxia.

Based on the limited documentation, the midwife and other members of the team who attended the baby at birth appeared to have responded appropriately to the unanticipated neonatal arrest. It is indicated in the Coroner's Investigation Statement that ventilation and cardiac compressions may not have commenced until four minutes after birth, but this could not be confirmed in the record.

This review was limited by the incomplete record supplied by the midwives and the hospital where the birth occurred.

Recommendations

1. The hospital and mid-wifery clinic should review their procedures for documenting and recording the actions of all members of the medical care team.

Case: 2009-N-16

History

The mother of the deceased was a 14 year old G1P0 who presented at the midwifery practice at 24 weeks and 5 days gestation. Her estimated date of delivery was June 15, 2008, based on a 13-week ultrasound. Her blood work and 18-week ultrasound were normal. Of note was a history of depression with antidepressant medication used until mid-pregnancy. She reportedly had poor social support, a history of family violence and increasing depression following discontinuing medication. She appeared to be underweight with a BMI of 16.9 and she reportedly smoked five cigarettes a day.

The mother was initially seen by her family doctor, then went into midwifery care. All scheduled visits were attended. She gained a total 51 pounds. At approximately 33 weeks gestation, she had a hospital triage visit for possible preterm labour.

Fundal heights were equivalent to gestational age until 35 weeks when the foetus was 33 cm. At 40 weeks, the foetus was 35cm. This represented a 2.5 cm growth over approximately seven weeks.

At 1245 hours on June 22, 2008, at 41 weeks gestation, the mother was assessed to be in labour with her cervix 80% effaced, 6-7 cm dilated, membranes intact and the pp at spines +1. Her labour reportedly began at 0200 hours. She was admitted to labour and delivery, but her labour was non-progressive. Foetal heart tones remained normal with accelerations and periodic decelerations noted. ARM was done with no fluid revealed. A consult was done for epidural and augmentation of labour at 1730 hours. An epidural was given at 1800 hours. Care was finally transferred to the obstetrician on call at 2125 hours. At 2150 hours, oxytocin augmentation was commenced and at 2130 and 2155 hours, there were prolonged decelerations down to 60 bpm for three minutes. Intrauterine resuscitative measures were carried out with good effect. The obstetrician was notified and ordered augmentation to be discontinued. At 2300 hours, the mother's cervix was unchanged and a decision was made to do a Caesarean section.

Summary of Postnatal Course

The baby was delivered by emergency Caesarean section at 0009 hours on June 23, 2008. The paediatrician had been summoned and was in attendance. The baby was

somewhat hypotonic with poor respiratory effort, so bag and mask ventilation was started. The paediatrician described the baby as having a “stoned appearance” and indicated that he thought the depressed state was likely due to maternal sedation. The baby’s condition improved after about 30 seconds of positive pressure ventilation. Apgar scores awarded were 5 and 9 at one and five minutes. Cord blood gases revealed a normal pH of 7.36.

The examination by the paediatrician indicated that apart from significant growth restriction and features of postmaturity, the infant appeared normal. The weight was 2190 g which is below the third percentile for gestational age. The length and head circumference were also less than the third percentile, but proportionally less so than the weight. There was no clinical evidence of congenital infection or syndrome. The baby was transferred to the special care nursery for observation.

In the special care nursery, vital signs were normal and the infant was placed in an isolette. There was a brief period during which oxygen saturation appeared low, but this was attributed to poor signal acquisition by the pulse oximeter. The infant quickly settled in room air with good oxygen saturation. The infant was given a small bottle feed at 0110 hours and settled well. Blood glucose was checked at 0230 hours and was slightly low at 2.5 mmol/L. At 0315 hours, the reading was 2.2 mmol/L, so the infant was given an additional feed of 15 ml by bottle. Over the next few hours, the blood glucose was borderline and the infant was not bottling well, so a nasogastric tube was inserted for gavage feeding. This was successful in bringing up the blood glucose which was consistently acceptable after a slightly low reading of 2.5 mmol/L at 0745 hours.

The infant seemed to be doing well until there was a large regurgitation during a gavage feed at 1813 hours. The nurse who took over care of the baby at 1945 hours noted that the respiratory rate had increased to 72 bpm, although the infant was crying. The infant was described as jaundiced and “greenish”. An hour later at 2040 hours, the infant was unsettled and it was difficult for the nursing staff to sooth him. He was making a moaning sound, had a tracheal tug and was tachypnoeic at 110 breaths per minute. Capillary refill was decreased and a pulse oximeter indicated a reading of 55%. Supplemental oxygen was administered and the RT and paediatrician were paged. Oxygen saturation remained low in 100% oxygen.

The paediatrician and RT arrived at 2110 hours. Examination revealed a dusky baby in respiratory distress with poor capillary refill. The heart rate, blood pressure and temperature were normal. Air entry was equal bilaterally, pulses were palpable and there was no murmur. A chest x-ray was done, blood work drawn and the baby placed on nasal CPAP. There was prolonged bleeding from the puncture site. Although the time of the study is not shown on the report, it appears that the first x-ray taken was normal, showing no evidence of parenchymal lung disease, air leak or cardiac abnormality. The capillary blood gas came back showing pH=7.01, pCO₂=74 mmHg, HCO₃=18 mmol/L. When this result was received, the paediatrician contacted the NICU at a larger hospital to arrange for transport. A team was not immediately

available and the paediatrician was advised to give a normal saline bolus and start antibiotics.

The infant's oxygen saturation remained low. A decision was made to intubate, so the anaesthetist on call was summoned. At 2308 hours, the infant was intubated orally and the tube secured. The infant's condition did not improve. A chest x-ray was ordered to check tube placement. The x-ray revealed a large left pneumothorax. The chest was needled with an angiocath and a chest tube placed with the assistance of a second paediatrician who had been called in. Despite successful positioning of the tube and evacuation of air, the infant steadily deteriorated and was pronounced dead at 0037 hours on June 24, 2008.

Prior to the infant's death, a CBC had revealed a very low platelet count of 25 and a platelet transfusion had been ordered. In addition, upon the suggestion of the larger hospital where the infant was to be transferred, PGE1 had been prepared for infusion in case the baby had a ductal dependent cardiac lesion. Neither had been instituted before the infant passed away.

Post Mortem

Post mortem examination revealed:

1. Severe intrauterine growth restriction
2. Evidence of left pneumothorax and chest tube placement
3. Lungs
4. Variable degrees of alveolar expansion
5. Generalized parenchymal congestion
6. Hyaline membrane formation
7. Many blood vessels filled with thrombi
8. Extramedullary hematopoiesis in liver
9. Negative bacterial and virological studies
10. Normal male karyotype
11. Negative toxicology

Death was attributed to "acute severe respiratory distress syndrome following a left pneumothorax in a newborn" due to "severe intrauterine growth restriction". An additional report received from a pathologist with special expertise in the neonatal pulmonary system indicated that there was "lung hypoplasia with superimposed diffuse alveolar damage" and that "this explains best the clinical presentation and rapid demise of this infant."

Discussion

The mother of the deceased infant was a young primip with many personal and social issues. In addition, her nutritional status was poor and she smoked. These factors put her at risk during pregnancy. Contrary to the College of Midwives Indications document, there was no mention of a level 1 consultation being done with other midwives regarding the care plan. Although there were initially no clear indicators for a level 2 consultation, it might have been prudent given the many issues (e.g. young age of the mother, nutritional status, history of depression and poor social support, etc.), that were present at the time.

The fundal heights were inaccurately plotted on the growth chart. The growth actually drops off the 75-percentile curve at 33 weeks and ends up in the 25 percentile at 40 weeks. The chart shows the growth curve remaining on the 75-percentile dropping to the 50-percentile at 40 weeks. Given the risk factors present in this case and the drop off in growth, an ultrasound would have been helpful in determining whether the growth and fluid levels were adequate and consultation with an obstetrician for inadequate growth would have been required.

Evidence shows that fundal height measurements can be useful in determining whether there is adequate growth in pregnancy, but may be affected by measurements done by different healthcare providers, habitus of the woman and descent of the presenting part at term. Maternal weight is not an accurate determinant. However, given that there was a 51 lb weight gain, it may have been difficult to assess the size of the baby by palpation. Oligohydramnios is also difficult to determine clinically, but could have been picked up with ultrasound.

This infant died at just over 24 hours of age from intractable hypoxemia following the development of respiratory distress and a tension pneumothorax. The root cause of the primary respiratory disorder remains obscure. Although the infant was growth restricted and was born by emergency Caesarean Section after an abnormal foetal heart tracing, he was born in relatively good condition with normal cord gases. The cause of the growth restriction is also not clear, although it was most likely due to placental insufficiency attributed to cigarette smoking.

The original pathologist performing the post mortem suggested that the cause of death was respiratory distress syndrome (RDS). Classic RDS is due to surfactant deficiency, occurring in preterm infants who demonstrate characteristic clinical and radiological findings not seen in this case. Although hyaline membranes were seen on microscopic examination of the lungs, this can be a non-specific finding in any baby receiving positive pressure ventilation. Rather than RDS, it is more likely that the infant suffered from persistent pulmonary hypertension of the newborn (PPHN) leading to profound oxygenation failure. PPHN can be primary or secondary to several associated neonatal conditions. There are a number of possibilities for the root cause of the baby's problem, but none is fully supported by the clinical course and results of investigations. These possibilities include:

- Sepsis with persistent pulmonary hypertension.

Babies with congenital sepsis will often present with increasing respiratory distress and may develop refractory hypoxemia due to PPHN. As possible supportive evidence, this infant had a high white blood cell count and low platelets that could result from bacterial sepsis. However, the mother was GBS negative and had no evidence of chorioamnionitis. Blood cultures taken from the baby were negative and no bacteria were identified on post-mortem examination.

- Chronic intrauterine hypoxia with PPHN

Placental insufficiency results in chronic intrauterine hypoxia which causes IUGR and other collateral effects. Among these is a tendency for normal postnatal pulmonary vascular relaxation to be impaired, especially if there is a superimposed acute perinatal asphyxial stress. This baby was certainly very undergrown and post-mortem examination revealed extensive extramedullary hematopoiesis consistent with chronic intrauterine hypoxia. However, Apgar scores were good and the cord blood gas was normal indicating that there was no acute impairment of gas exchange between placenta and foetus during the immediate perinatal period. The consultant pathologist indicated that there was some degree of pulmonary hypoplasia, however the baby required no respiratory support until his sudden deterioration at 20 hours of age. He clearly had sufficient lung tissue to support adequate gas exchange for a period of time, but the pulmonary hypoplasia that presumably resulted from placental insufficiency would have been associated with an increased tendency to develop PPHN.

- Cyanotic Congenital Heart Disease

Several forms of cyanotic congenital heart disease can present with refractory hypoxemia presenting several hours after birth as the ductus arteriosus begins to constrict. These include transposition of the great vessels, tricuspid and pulmonary atresia. The normal post-mortem cardiac examination excludes this possibility.

- PPHN resulting from foetal exposure to SSRIs.

There is a report of an association between intrauterine exposure of the foetus to SSRIs and subsequent development of PPHN (N Engl J Med 2006;354:579-87). However, this association seems to be confined to exposure in the latter part of pregnancy and it appears that the baby's mother discontinued her use of antidepressants prior to the 20th week of gestation.

- Other causes of severe IUGR that might be associated with poor postnatal adaptation have also been excluded by the post-mortem examination. These include congenital viral infections and chromosomal abnormalities.

It should be mentioned that there are no apparent errors or deficiencies in the care provided to the baby by the members of the paediatric care team. The baby was monitored in the SCN before his deterioration which appeared to have come rather quickly and unexpectedly. When it became apparent that the infant was quite ill,

support was quickly mobilized and each of the members of the team took the appropriate steps to investigate and support the infant. It is unfortunate that the transport team from the larger hospital was unavailable to help, but it is unlikely that earlier mobilization would have improved the outcome. However, it is possible that, if the infant had been born in a tertiary perinatal centre with in-house neonatology coverage and the ability to provide high-level intensive care, he might have survived. In particular, the availability of inhaled nitric oxide, might have been helpful.

Recommendations

1. Midwives are reminded of the College Of Midwives of Ontario requirement for level 1 consultations to be completed and documented.
2. Obstetrical healthcare providers are reminded of the importance of accurate assessment and documentation of prenatal visits.
3. Obstetrical healthcare providers are reminded of the importance of ordering ultrasounds when there are deviations in the growth rate of the baby.

Case Summaries: Stillbirths

Case: 2009-S-1

History

The mother was a 39 year old primigravida. Prenatal care was performed by her family doctor until 31 weeks, and then by an obstetrician. Her dates were certain and were confirmed with 12 and 19 week ultrasound scans. She had IPS screening which was positive for Down's syndrome, but subsequent amniocentesis was normal. Routine antenatal blood tests were all normal. She was a healthy woman and a non smoker.

At 32 weeks gestation, she had a two hour, 75 gram oral glucose tolerance test because her obstetrician found her to be large for dates. At 34 weeks gestation, an ultrasound was done to follow up the false positive IPS test. The oral glucose tolerance test was normal, but the scan did confirm a large baby with measurements advanced 2-3 weeks. The ultrasound also demonstrated a possible foetal heart arrhythmia. After the arrhythmia was noted, the mother had weekly NSTs for three weeks, which were all normal. A TSH was also done at 35 weeks and was normal. Her GBS swab was negative. Her blood pressure in the obstetrician's office was 124/66 at 31 weeks and 130/92 and 140/86 at 39 weeks. Urine testing for protein was always negative.

The mother presented to the Labour Room at 40 weeks and 4 days gestation with irregular contractions and spontaneous rupture of membranes for 30 minutes. The nurse placed an external foetal monitor which showed a foetal heart rate of 160-180 bpm in the first 10 minutes. The mother's pulse was 74 bpm, but her blood pressure was 175/104 and 168/94 at admission and five minutes later. The nurse assessed her cervix and found it to be 3 cm dilated, 100% effaced and the presenting part was very low. Her pad was noted to contain thick meconium. She was contracting every 2-3 minutes.

The nurse monitored the mother for the next hour. Initially the tracing showed tachycardia to 170-180 bpm with minimal variability and no decelerations or accelerations. By 30 minutes after admission, the foetal heart had settled to 160 - 165 bpm with moderate variability. At 49 minutes, the tracing showed some irregularities (possible skipped beats), but the nurse's notes do not indicate that an arrhythmia was heard. Nitrous oxide was given. At 55 minutes, the baseline heart rate fell to 140-150 bpm. The variability was moderate, but difficult to interpret, due to the irregular tracing at this point. At 60 minutes after admission, the mother's blood pressure was 173/91 with a pulse of 71 bpm. The obstetrician on call that night was notified of the patient's admission. Routine orders were obtained. The obstetrician also ordered an IV to be started for rehydration. A CBC (group and screen) and coagulation screen showed normal Hb, platelet count and INR and PTT. Urine tested trace for protein.

At 75 minutes after admission, the mother was up to the washroom and a possible deceleration was heard on her return to the monitor. At 85 minutes, her IV was started and the foetal heart rate baseline was 135-140 with minimal variability. Two more

decelerations occurred at 100 and 110 minutes after admission. The toco was not recording well during this period and no note is made of the timing of these decelerations to contractions by palpation.

At 117 minutes after admission, there was a deceleration to 70 bpm and the return to baseline heart rate was slow and incomplete. The nurse examined her one minute later and found the cervix to be 4 cm, with again a very low presenting part and no fluid was noted. After the exam, the foetal heart returned to 115 - 120 bpm for one minute, but soon there were recurrent deeper and wider decelerations. Oxygen was given by mask and the obstetrician was called at 129 minutes after the admission. The abnormal foetal heart activity continued with many large fluctuations of the foetal heart between 110 and 80 bpm until 139 minutes after admission, when no foetal heart could be detected.

The obstetrician arrived at 143 minutes (14 minutes after being called) and attempted to detect the foetal heart by applying a scalp clip. When this was unsuccessful, a real time ultrasound scan was performed and again no heart activity could be detected. The obstetrician discussed with the parents that it appeared their baby had died. The parents requested an urgent Caesarean. General anaesthesia and a midline incision were used and the baby was delivered 30 minutes after the decision for Caesarean was made. Thick meconium stained fluid and oligohydramnios were noted at the surgery. The baby had poor tone and no spontaneous movement. Apgar scores were 0, 0 and 0 at one, five and ten minutes. The baby, a female, weighed 3910 grams.

The birth was attended by the pediatrician on call, two pediatric nurses and a respiratory technologist. The pediatrician suctioned meconium with an endotracheal tube, then ventilated the baby with endotracheal tube in place while chest compressions were applied. An umbilical vein catheter was placed and epinephrine was given. The baby was described as limp and unresponsive. No attempts at respirations and no heartbeat or movement were seen at any time during the resuscitation. The resuscitation was halted 15 minutes after the birth.

Postmortem

Autopsy showed no congenital abnormalities. Microscopic exam of the placenta showed multiple infarcts. Microscopic exam of the lungs showed the presence of amniotic contents. Cause of stillbirth: perinatal asphyxia.

Discussion

When this woman presented in labour, she had an elevated blood pressure and a foetal tachycardia was detected with minimal variability. Thick meconium stained fluid was noted. However, after 30 minutes the tachycardia was improved, as was the variability. The nurse observed her for one hour prior to notifying the obstetrician of her admission. When the obstetrician was made aware of the patient, her blood pressure was still elevated, but lab tests (including platelets and urine protein), were normal. The foetal tachycardia had resolved, but the variability was difficult to assess due to what appears

to be an irregular heartbeat. It is unclear if this was detected by the nurse and if the obstetrician was informed. The foetal heart monitor strip continued to be difficult to interpret with a changing (i.e. dropping) baseline heart rate, periods of minimal variability and three decelerations over 35 minutes, with no indication of the contraction activity at the time. The baby died about 20 minutes after the sudden onset of a series of decelerations or agonal bradycardia, which started about two hours after the patient's admission. The reason for the abnormal foetal heart rate and the intrapartum death are unclear. Findings of meconium aspiration on autopsy do not define the process leading to the death.

The evidence of placental infarcts and elevated blood pressure in a 39 year old pregnant woman suggest placental insufficiency may have played a role in the foetal demise.

Recommendations:

1. Obstetrical caregivers are reminded that interpretation of the foetal heart rate monitor strip includes assessment and documentation of the variability of accelerations and decelerations. The interpretation of decelerations also requires the documentation of uterine contraction activity with a monitor or by palpation.
2. The hospital should ensure that the labour records have a place to document each of these aspects of foetal health surveillance as well as the nursing interpretation of these findings and the action plan.

Case: 2009-S-2

History

The mother was a 26 year old primigravida who received care from her family doctor until 14 weeks gestation, at which time she transferred to a registered midwife. Initial laboratory investigations, including PAP smear and IPS tests, were all within normal limits. The family physician identified an increased blood pressure of 130/90 at 10 weeks, however it remained normal through all subsequent visits. The mother had a history of asthma for which she was using Ventolin and Symbicort. She had an elevated BMI of 47 and weighed over 300 pounds. The mother had ultrasound exams at 12, 19, 27, 36 and 39 weeks gestation. These scans noted that the anatomy review was limited due to maternal habitus and the last two identified that the baby was cephalic and large for gestational age. At this point, the midwife advised moving to a hospital birth and the parents agreed. Doppler studies done six days prior to labour were normal and the estimated foetal weight at that time was 4131 grams. The swab for GBS was negative at 37 weeks gestation. The mother's chart indicates that there were multiple informed choice discussions with the midwife relating to risk factors and place of birth.

At 2000 hours on June 11, 2007, at 39 weeks and 4 days gestation, the mother began labour while at home. She called the midwife at 0140 hours and was admitted to the

hospital by the midwife at 0230 hours on June 12, 2007. She was in early labour with contractions every 3 to 5 minutes, lasting for 60 seconds. Her blood pressure was 127/71 and pulse was 101. The cervix was found by the midwife to be 3 cm dilated, 1 cm long and posterior. The midwife noted that it was difficult to palpate contractions and auscultate the foetal heart due to the maternal size. At 0250 hours, the midwife recommended and performed an ARM. Thin meconium staining was revealed at this time. The midwife informed the labour room nurses of the presence of meconium and a large for gestational age infant. The midwife intended to ask the emergency room physician or the anaesthesiologist on call to attend the birth. The midwife also inquired into the hospital's policy for continuous EFM with meconium stained amniotic fluid. The nurse responded that there was no policy, but if the initial monitor strip was reactive, intermittent auscultation would be acceptable. The EFM appears to have been used to make intermittent assessments of the foetal heart between 0300 hours and 0500 hours, with a few small segments of baseline for about one minute, recorded ranging from 120 to 150 beats per minute. There is no contraction recording in this time period. The midwife recorded foetal heart rates ranging from 118 to 150 bpm in the progress notes, every 15 to 25 minutes, from 0230 hours to 0600 hours.

By 0500 hours, the pains were much stronger and the mother was requesting pain medication. She was given acetaminophen and dimenhydrinate by mouth by the midwife. At 0540 hours, the midwife contacted the nurses to request a consultation with the anaesthesiologist for an epidural and a consultation with the obstetrician on call for BMI and possible scalp electrode placement due to her difficulties tracing foetal heart. The nurses placed an IV line for the mother and contacted the anaesthesiologist who commenced the epidural at 0555 hours. The nurses also contacted the obstetrician on call at 0600 hours. Continuous foetal monitoring was implemented by the midwife after the epidural at 0613 hours, about 18 minutes after the epidural was started. The monitor showed a baseline of 135 to 145 bpm, with moderate variability. A change in the foetal heart rate from 140 to 90 bpm for about 2 minutes, was noted at 0622 hours and was coincident with a contraction. The baseline returned to 145 bpm at 0624 hours.

The obstetrician assessed the mother at 0624 hours and reviewed the history and progress with the midwife. The obstetrician noted that she reviewed a short EFM trace (from 0613 hours) which "appeared reactive" and noted the one possible early deceleration. She discussed this with the midwife who explained that this was artifact as she was adjusting the monitor at that time. The obstetrician found the mother's cervix to be 3 to 4 cm dilated, 70 to 80% effaced and not very well applied at station -1 to 0 with membranes absent. She noted some early caput formation, but applied a scalp clip with ease. The foetal heart rate was picked up at 130 to 140 bpm. The obstetrician ordered oxytocin to augment the labour and left for the operating room to perform a Caesarean delivery on another patient.

The internal EFM tracing showed a baseline rate of 138 to 145 bpm with minimal/moderate variability and no decelerations or accelerations between 0627 and 0654 hours. The toco was showing uterine contractions every 1.5 to 3 minutes during

this time. The midwife inserted a Foley catheter at 0632 hours and hung the oxytocin drip at 0653 hours.

At 0654 hours, the foetal heart rate fell and the oxytocin was discontinued only 1 to 2 minutes after it was commenced. The foetal heart rate dropped to 60 bpm by 0656 hours and never recovered. The midwife contacted the nursing staff who came to her aid. The scalp clip came off during the midwife's exam and she proceeded to place a new clip, confirming persistent bradycardia. Intrauterine resuscitation was commenced with IV fluids and oxygen. The nurses contacted the obstetrician who was still in the operating room with the other Caesarean patient. She instructed the staff to transfer the mother to the operating room and prepare for an emergency Caesarean section. The mother was transported to the operating room at 0706 hours and was seen by the obstetrician at 0712 hours. The foetal heart rate tracing was reviewed and was noted to be very erratic, with large swings between 130 and 200 bpm. Another clip was placed as there was some concern that the other may have been dislodged. The tracing continued to show what was likely to be artifact.

As soon as an operating room was ready, a Caesarean was performed with an epidural anaesthetic and a Pfannenstiel incision. As the Caesarean was starting, the nurses paged a second anaesthesiologist and a paediatrician to come to the operating room. The surgery was commenced at 0725 hours and the baby was delivered at 0730 hours. The baby was female, 4375 grams, with no gross abnormalities. The infant had Apgar scores of 0, 0, and 0 at one, five and ten minutes. Cord blood gases showed 6.749/93.4/30.3/12.2/-29.2 and 7.095/61.6/37.6/18.1/-13.3 for arterial and venous pH/pCO₂/pO₂/bicarb/base excess, respectively.

There was never any sign of life or a heartbeat. The baby was given resuscitation initially by the anaesthetist, obstetrical nurses and the midwives. Suctioning revealed thick particulate meconium. Positive pressure ventilation and chest compressions began immediately. The anaesthesiologist intubated the baby and adrenaline was given twice. A respiratory technician and second anaesthetist also assisted. The paediatrician arrived at about 15 minutes and confirmed the ET tube placement and attempted an IV. At 25 minutes, efforts were discontinued and death pronounced.

The midwives continued supportive care into the postpartum period. The midwives did in-person and phone consultations at Day 1, 2, 3, 4, 5 and 8. The midwives also informed the family physician of the stillbirth and the history of the patient's obstetric care outcome.

Postmortem

Placenta: early acute phlebitis and mild acute chorioamnionitis

Cause of Stillbirth: Intrapartum death secondary to perinatal asphyxia.

Discussion

The risks of shoulder dystocia were a reality in light of this woman's BMI of 47 and the estimated foetal size, both being risk factors. The plan to birth in a hospital was appropriate. In addition however, it would be prudent for midwives and family physicians caring for morbidly obese woman to consider referring to an obstetrician and an anaesthesiologist in the intended birth centre prior to labour, though it is unlikely such consultation would have changed the eventual outcome.

In the presence of meconium and slow labour progress, the move to EFM should have been considered earlier in the labour. The monitoring was clearly difficult due to this mother's obesity, however if continuous EFM was felt to be indicated earlier, referral to an obstetrician would have occurred with internal scalp clip placement and a more accurate assessment of the foetal condition. It is possible that the outcome would still be the same, even with earlier invasive monitoring.

The obstetrician ordered, and the midwife commenced, oxytocin to augment labour with meconium stained amniotic fluid and with a tracing with minimal variability and a baseline rate which appears to be elevated over the early labour auscultated rates. The obstetrician should have requested a more thorough assessment of the foetal status with longer EFM before making this decision. The obstetrician was not able to stay in the labour room to follow the foetal heart as she was required to perform another Caesarean. The midwife should have reassessed the EFM prior to commencing the oxytocin drip and should have showed the tracing to the obstetrician for reassessment. However, the oxytocin is unlikely to have played a significant role in this outcome as it was running for such a short time as to have had little effect on the labour.

When the foetal heart rate dropped, the nurses acted in aid of the midwife and all appropriate intrauterine resuscitative measures appear to have been attempted. The obstetrician and anaesthesiologist were both close at hand, but busy with another Caesarean birth. Still, the Caesarean was commenced within 30 minutes of the bradycardia and the baby was delivered after only five minutes of surgery. The foetus had likely succumbed prior to the operation, but this was impossible to discern with confidence.

The resuscitation efforts were appropriate with the use of positive pressure ventilation, cardiac compressions, endotracheal intubation and adrenaline. The anaesthesiologist had to leave the mother to aid with the efforts for the baby until another anaesthesiologist came at five minutes and the pediatrician arrived at 15 minutes after the birth. Given that the bradycardia initially occurred about 30 minute prior to the birth, these backup personal should have been notified earlier to enable their attendance at the birth.

Recommendations:

1. Family doctors, nurse practitioners and midwives should consider consultation with a specialist physician for morbidly obese women at intake into their maternity care.

Obstetricians should refer such women to the anaesthesiologists in the intended birth hospital.

2. The hospital should review current foetal surveillance protocol and indications for electronic foetal monitoring and ensure it is referenced, edited and updated accordingly with current guidelines such as “Foetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline”, SOGC 2008.
3. Caregivers are reminded that use of an electronic foetal monitor for intermittent auscultation protocol is not appropriate.
4. If a caregiver feels EFM is indicated and a suitable tracing cannot be obtained, referral for more invasive monitoring should be considered.
5. The hospital should review the requirements for labour induction or augmentation with oxytocin, including assessment of foetal status, with this midwife and ensure that policies and procedures are written to guide such actions.
6. It is recommended that the College of Midwives of Ontario include the practice of placement of scalp electrodes within their scope and training.
7. When abnormal foetal status is diagnosed and delivery is expected, the nursing staff should routinely notify the pediatrician on call to attend.

Case: 2009-S-3

History

The mother of the deceased was a 28 year old G3P2 with a LMP of April 26, 2007. Her doctor gave an EDC of January 30th 2008. The patient's EDC (by LMP given to ultrasound) of April 19, 2007 was January 23 2008.

The patient had non insulin dependent diabetes mellitus (NIDDM) for five years prior to this pregnancy and she was treated with Glyburide. Her past obstetrical history was significant for a pregnancy in 1993 complicated by preeclampsia and intrauterine growth retardation (IUGR) necessitating induction at 36 weeks for a 3 lb 9 oz baby girl which she delivered vaginally. A second pregnancy in 2004 was complicated by preexisting NIDDM, preeclampsia with failed induction, and Caesarean section at 41 weeks. This baby was an 8 lb 8.5 oz baby boy.

During this pregnancy, the patient seemingly presented once on June 18, 2007, but there are no notes documenting what was discussed or planned other than weight, blood pressure and urinalysis. She was not seen again till October 3, 2007 at 20 weeks gestation and again, there were no notes charted on the antenatal sheets. The only blood work on record from the office chart was dated October 30, 2007. The patient was A positive, HepB, VDRL and HIV negative. She had a normal hemoglobin, decreased B12 at 103 and elevated blood sugar at 10.1.

The first ultrasound was done October 18, 2007, presumably ordered at the October 3, 2007 visit. The ultrasound showed an intrauterine pregnancy at 24 weeks, with no gross abnormalities, but visualization was noted to be poor, secondary to body habitus. The placenta was noted to be thickened. The radiologist recommended a repeat ultrasound. A second ultrasound was done on December 6, 2007. The growth was noted to be appropriate, but at 94th percentile. There was no further blood work in the chart and no notes suggesting consultation obtained with any other physicians or allied health care providers.

GBS screening was done and was positive. She had a repeat Caesarean section arranged for 39 weeks at her 36 week visit. She was hypertensive as of her January 19, 2008 visit. Her BP was 136/96 with trace protein. On January 22, 2008 at her last office visit, her BP was 156/105 with trace protein. There is no documentation of any blood work being drawn, any history or physical, other than weight, blood pressure and urinalysis being done. There is no evidence of any foetal surveillance being done in light of her diabetes and hypertension.

The patient presented to hospital on January 27, 2008 for admission prior to her booked Caesarean section on January 28, 2008. Her vitals at 1340 hours were significant for BP 143/102 and foetal heart rate tracing was non-reassuring. The patient's doctor was contacted at 1345 hours and indicated that he would attend. The foetal heart rate deteriorated with decelerations noted at 1355 hours. The foetal heart rate was noted to be 66 at 1410 hours, then unable to be detected. The patient's doctor was attending at this point and was unable to obtain ultrasound or Caesarean Section in timely fashion. The Caesarean Section was obtained later in the day and a stillborn baby girl weighing 3.82 kg, was born at 1709 hours. The reason for Caesarean Section three hours after the FHR was lost is unclear from the notes.

Post Mortem

Stillbirth - No Specific Etiology

Discussion

The mother of the deceased had a high risk pregnancy complicated by pre-existing NIDDM. It appears that this was not recognized by her doctor. The mother should have been followed by a multidisciplinary team to appropriately manage her diabetes and pregnancy. She should have had increased foetal surveillance and planning around the appropriate timing of her planned Caesarean Section. Her prenatal care fell below the standard of care expected of an obstetrical care provider. Once arriving in hospital, a non-reassuring foetal heart tracing was noted and urgent Caesarean Section was not able to be performed in a timely fashion. She subsequently had a Caesarean Section three hours after the foetal heart was last heard. The reason for the Caesarean section at this late stage, versus induction of labour, was not discussed in the hospital notes.

Recommendations

1. The doctor involved in this case should be referred to the CPSO for review of both his record keeping and care as both fell below the standard expected of an obstetrical care provider in Ontario in 2007/2008.

Case: 2009-S-6

History

The mother of the stillborn was a 28 year old G3P0 with an EDD of November 11, 2008 based on LMP and early second trimester ultrasound. Ultrasound on June 21 at 18 weeks 5 days was normal except for the presence of placenta previa. Follow up ultrasounds in the third trimester showed resolution of the placenta previa. Routine prenatal laboratory investigations were normal. Her blood type was A negative with a negative antibody screen. She received Rh immune globulin at 28 weeks. IPS and second trimester GCT were normal. She was GBS negative.

The mother's medical history included a pregnancy termination in 2001 and a spontaneous abortion in 2007. Her antenatal care was initially provided by her family doctor and obstetrician, but she subsequently chose midwifery care at 35 weeks gestation.

Course in Labour and Delivery

Labour onset at 1600 hours on November 1 at 38 weeks 4 days gestation. She was admitted to the hospital at 1758 hours under the care of her midwives. Her cervix was 6 cm dilated and foetal heart was normal. An ARM was carried out at 2010 hours for clear fluid. An epidural was started at 2100 hours and thereafter foetal monitoring was by EFM. She was fully dilated at 2400 hours with the vertex at spines. The foetal heart was normal during the first stage. During the second stage, contractions were noted to be further apart and not as strong. Pushing was ineffectual. An obstetrical consultation was obtained and oxytocin augmentation was initiated at 0300 hours. The contraction pattern improved by 0345 hours. At 0425 hours, there was no progress with pushing and the obstetrician was notified. Variable decelerations were noted at 0440 hours with an increase in the baseline foetal heart rate to 160-170. The vertex was at spines +1 - +2 with pushing. After the options were discussed with the patient, it was decided to proceed with a trial of vacuum extraction. The procedure was abandoned when a gentle pull for a couple of minutes produced no descent. The operating room staff was called in urgently for Caesarean section at 0500 hours. The baseline foetal heart rate just before commencement of the operation at 0537 hours was 170-180.

The mother was delivered of a 7 lb 5 oz male infant at 0547 hours. The amniotic fluid was clear. The baby was flat with Apgars of 0, 0 and 0. Arterial cord pH was 7.20 and venous cord pH was 7.25.

Post Mortem

The baby's growth parameters were appropriate for gestational age and there were no congenital anomalies.

Reflection of the scalp revealed extensive haemorrhage across the back of the head with about 10cc of liquid blood drained upon incision into the scalp hematoma. There was molding of the skull bones with depression of the frontal and occipital bones relative to the parietal bones. There were symmetrical rectangular fractures of the outer table of the right and left parietal bones measuring 2.4 cm X 2 cm and 2.3 cm X 2 cm respectively. There was a thin layer of subdural haemorrhage bilaterally with a total volume of about 10cc. There was extensive subarachnoid blood over the base of the brain within the sulci and particularly over the left frontal and temporal lobes.

The cause of death could not be determined.

Discussion

This infant was stillborn at Caesarean section following a failed vacuum procedure. Cord gases showed no evidence of asphyxia and although there was evidence of cranial trauma at autopsy, the injuries were not felt to be the cause of death.

The birth injuries identified at autopsy can occur spontaneously, but are more commonly related to operative vaginal procedures. Subdural and subarachnoid haemorrhage have been reported to occur in 8 and 2.3 per 10,000 vacuum deliveries respectively as compared to 2.8 and 1.3 per 10,000 unassisted deliveries. Depressed skull fractures occur in 3.7 per 100,000 deliveries. While the majority are associated with forceps delivery, they have also been reported in unassisted and Caesarean deliveries. Such fractures can occur during Caesarean delivery with elevation of the deeply impacted foetal head.

Vacuum delivery has also been associated with an increased occurrence of subgaleal haemorrhage. The volume of blood that can potentially be lost into this space can result in hemorrhagic shock and death, but the volume of blood present at autopsy in this case did not appear to be large enough to explain the outcome.

It is likely that given the amount of molding identified at autopsy, the station of the presenting part at the time of the trial vacuum procedure was overestimated. The position of the vertex is not recorded in the record. As such, this may have been a high mid-pelvic operative procedure. Operative procedures from this station are associated with a significant chance of failure and an increased risk of birth trauma. In such a setting, it is advisable to have the Caesarean section team mobilized prior to the trial to minimize the time interval between the trial and delivery. It cannot be determined from this review if such preparations, or going straight to Caesarean section without the trial vacuum extraction, would have altered the outcome.

Recommendations

None

Case: 2009-S-7

History

The mother of the deceased was a 36 year old G5P3 with an EDD of May 21, 2008 based on her LMP and 1st trimester dating ultrasound. Routine prenatal laboratory investigations were normal, but she was positive for sickle cell trait. Serum creatinine was normal. Prenatal genetic testing was declined. Ultrasounds at 20 and 29 weeks were normal. GCT was normal and GBS testing was negative.

The patient's family doctor consulted an obstetrician at 11 weeks gestation with the plan to do shared care until the third trimester.

The mother's obstetrical history included three previous term pregnancies and a spontaneous abortion. The term pregnancies were complicated by hypertension and were all delivered at term. The first baby was delivered in 1988 weighing 2.64 kg; the second in 1994 weighing 2.89 kg and the third in 2006, weighing 3.26 kg. Following her last pregnancy, she was started on hydrochlorothiazide, which was continued until being changed over to labetalol 200 mg, twice daily, early in the second trimester of this pregnancy. Her blood pressure was well-maintained, running from 110/74 to 134/88 and at no time was there proteinuria. SFH measurements were consistent with appropriate growth. Her prenatal care was transferred to an obstetrician at 35 weeks. Labour was induced at 38 weeks because of chronic hypertension.

Course in Labour and Delivery

Three applications of Prostin gel occurred from May 8 to May 9. Labour did not ensue and the patient was discharged home. She returned on May 10 at 1005 hours and was admitted at 1140 hours. She was assessed by the obstetrician on call. Her blood pressure was 143/84. She had occasional contractions and the foetal heart was normal. The cervix was 3-4 cm dilated, 70% effaced and the presenting part documented at spines -2. An artificial rupture of membranes was performed for clear fluid and oxytocin was ordered.

The following timeline is reconstructed from a combination of the medical record and interviews with the care providers outlined in the Coroner's Investigation Statement.

1145 hours – Oxytocin was started as per protocol. The baseline foetal heart rate (FHR) was 135-145 bpm, minimal to moderate variability and variable decelerations noted.

1200 hours – Contractions were q6m lasting 40 seconds. By 1215 hours, the patient was experiencing strong contractions and feeling rectal pressure. No vaginal

examination was performed at this time. The monitor was frequently readjusted as the FHR was lost with patient's movements.

1230 hours – Contractions were q2-6m lasting 40s. The baseline FHR was now 150-155 bpm, minimal variability and deepening decelerations. Oxytocin infusion was increased to 4 mu/min.

1250 hours – Severe variable deceleration occurred. The obstetrician was informed while attending a delivery. Other than a change of position at 1245 hours, there is no documentation of the use of intrauterine resuscitation techniques despite a rising baseline, minimal variability and severe variable decelerations. The oxytocin continued at 4 mu/min.

1310 hours – The foetal heart rate tracing was reviewed by the obstetrician and the oxytocin was ordered stopped. No vaginal examination was performed at this time. IV fluids were increased. At 1320 hours, the variable decelerations were down to 60 bpm, lasting 80 s.

1345 hours – The patient was repositioned because of loss of contact with the foetal heart. The cervix was 3-4 cm dilated and the presenting part was noted to be ballotable. The patient requested an epidural. She was allowed up to the washroom to urinate and returned to bed 5 minutes later. O2 was commenced.

1400 hours – The obstetrician was requested to attend to reassess the patient and apply a foetal scalp clip for better monitoring of the FHR.

1415 hours– The patient was repositioned on her left side with the FHR tracing showing minimal variability and decelerations.

1425 hours – The monitor alarmed (possibly indicating lack of contact). Two nurses responded and on entering the room, found there was no FHR on the monitor. The patient was repositioned, but the foetal heart could not be found. It was then noted that the umbilical cord was protruding from the vagina.

1430 hours – One of the nurses placed a hand in the vagina to elevate the presenting part and keep pressure off the cord. The other nurse wheeled the bed down to the operating room. The obstetrician was met in the hallway. A stat call was placed for anaesthesia and paediatrics. In the operating room, the monitor was reapplied. A heart rate of 108 bpm was recorded, but the nurse noted there was no pulsation in the cord.

1440 hours – Anaesthetist arrived.

1444 hours – Caesarean section was commenced under general anaesthesia and the baby was delivered. Apgars were 0, 0 and 0. The arterial cord blood gas pH was 7.40. Bag and mask ventilation was commenced.

1445 hours – Cardiac compressions started.

1448 hours – Intubation attempted x2. Bag and mask ventilation resumed.

1451 hours – Intubation again attempted. Bag and mask ventilation resumed.

1455 hours – Umbilical vein catheter inserted. 10cc NS given along with 3cc of 1:10,000 epinephrine.

1448 hours – Second bolus of epinephrine given.

1502 hours– Intubation successful.

1525 hours– Childrens' hospital consulted and advised to stop resuscitation.

1536 hours – The baby was pronounced dead.

Post Mortem

There were no congenital anomalies. Body weight was 3.16 kg (25th percentile for gestational age).

There was evidence of severe hypoxic-ischemic stress compatible with cord compression. There was no significant placental pathology.

The cause of death was peri-partum asphyxia.

Discussion

This infant died as a result of acute asphyxia due to umbilical cord prolapse and occlusion. This occurred in the setting of induction of labour for chronic hypertension at 38 weeks gestation. At the time the induction was started, the presenting part was noted to be high. This is a recognized risk factor for cord prolapse. Although not an absolute contraindication for an artificial rupture of membranes, obstetrical care providers must be cognizant of the risk, not only at the time of membrane rupture, but also until such time that the presenting part becomes fixed in the pelvis. Allowing the patient to ambulate in this setting would be ill-advised. Furthermore, obstetrical care providers should have a heightened index of suspicion that variable decelerations may be due to cord prolapse and a vaginal examination should be carried out promptly.

Review of the heart rate tracing indicates that a vaginal examination should have been considered at 1310 hours to rule out cord prolapse. A second opportunity for this consideration occurred at 1405 hours. Although the obstetrician was notified, a vaginal examination was not performed. It cannot be determined if this would have changed the outcome. Further review of the FHR tracing revealed an atypical and then abnormal pattern. When an atypical tracing is apparent, intrauterine resuscitation should be commenced. While there was some indication that O₂ was given, maternal position was changed and hydration was increased at different times. Performance of a vaginal examination and stopping or decreasing the oxytocin before 1310 hours should have been carried out.

The cord blood gas result is not consistent with the outcome.

Recommendations:

1. Obstetrical care providers are reminded that the possibility of umbilical cord prolapse should be considered in the presence of variable decelerations, particularly when the presenting part is not in the pelvis.
2. Obstetrical care providers are reminded that ambulation in the setting of ruptured membranes and a high presenting part increases the risk of cord prolapse.
3. Obstetrical care providers are reminded of the importance of effective interpretation of EFM tracings, consideration of potential causes and additional clinical actions required based on the SOGC Clinical Practice Guideline for Foetal Health Surveillance, September 2007.
4. Obstetrical care providers are reminded of the importance of full and accurate documentation.

Case: 2009-S-9

History

The mother of the deceased was a 32 year old G5T3P0A1L3. In 2001, at 42 weeks gestation, she had a spontaneous vaginal delivery of a 3.6 kg baby induced with artificial rupture of membranes.

In 2003, at 40 weeks and 6 days gestation, the mother delivered a 3.46 kg baby with spontaneous labour.

In 2004, at 42 weeks gestation, labour was induced with prostaglandin. There was an ARM followed by spontaneous delivery.

In 2005, the mother had a spontaneous abortion at 7 weeks 4 days gestation that required no attendance.

The mother had a medical history that included varicosities of the right calf and thigh and obesity.

Summary of Prenatal Care

This patient entered midwifery care at approximately 7 weeks' gestation. At 5'2", she had a BMI of 29 and gained 23 pounds (10.43 kg) in the pregnancy. Her blood pressure, urinalysis and fundal height measurements were all charted and were within normal. She did not have any known drug allergies, but environmental sensitivities were noted. She was Hepatitis B, HIV and syphilis negative and rubella immune. The patient's first ultrasound (8 weeks 2 days) gave an EDD of October 10, 2006. The second ultrasound (12 weeks 6 days) gave an EDD of October 6 and a third ultrasound (19 weeks 1 day) gave an EDD of October 8 with normal foetal morphology, anterior placental location and normal amniotic fluid volume. The EDD used on the record was October 8, 2006.

The IPS was negative. WinRho was administered at 25 weeks 3 days and glucose screening was offered, but declined. GBS culture was negative at 36 weeks 3 days. Further ultrasounds were done at 32 weeks 5 days for growth and are noted as "normal".

When the pregnancy was postdates, two BPPs were done and both scored 8/8. Two ultrasounds for postdates indicated that the foetus appeared to be four weeks smaller than for dates. Cervical exams were done at 39 weeks 4 days and 40 weeks 4 days. On October 16, at 41 weeks 1 day, the cervix was soft stretchy posterior and 1-2 cm dilated and the head was not well applied. The patient was given homeopathic medications that were not identified on the record. On October 19, 2006 at 41 weeks 4 days, the cervix was examined, but not described until a narrative at 2030 hours that night. An illegible antenatal note may have indicated that there was a plan for artificial rupture of membranes.

Summary of Labour, and Delivery and Postpartum

On October 19, 2006 (41 weeks 4 days) at about 2030 hours, the patient called the midwife after she suspected that her membranes had ruptured. She reported bloody show and clots which was said to be the result of a stretch and sweep done at 1600 hours when the cervix was 2-3 cm soft, stretched to 4 cm and was posterior. The membranes were felt and it appeared to be a cephalic presentation. The patient was unsure about the frequency of foetal movements, so the midwife advised her to drink juice, focus on the movements and call back if no movements were detected. The midwife also instructed the patient to call if bleeding increased and to check in before she went to bed.

On October 20, 2006 (41 weeks 5 days), the midwife was paged by the patient at 0615 hours. The patient reported that she had experienced irregular contractions that ended at approximately 0100 hours. At 0400, the pains commenced Q10, mild to moderate. The patient reported foetal movements during the night, but that she had not felt any since waking in labour.

The plan by the midwife was to visit the patient at home at 1000 hours to conduct an assessment. At 0955 hours, the midwife was paged by the patient who indicated that she was in intense pain and wanted to move to her mother's home where the birth was planned to take place. The patient and the midwife both arrived at the patient's mother's home at 1005 hours. At 1010 hours, they were unable to get a foetal heart, so the patient was asked to move to a more comfortable chair located in the basement where it would have been easier to monitor the foetal heart rate.

At 1017 hours, a foetal heart rate could not be detected and the patient was requesting Demerol for pain relief. At 1023 hours, an assessment by the midwife found the woman to be 7cm, 80% effaced at 0 station membranes, intact and show. The midwife informed the patient that she was too dilated with two minute strong contractions, to go to the

hospital. The midwife recommended that the patient take a shower for pain relief and to “slow” the contractions. The midwife indicated that this would give her time to find the foetal heart and set up equipment.

At 1023 hours, the first midwife reported to a second midwife who was en route to the residence, that she could not find the foetal heart rate.

By 1028 hours, the patient was downstairs in the shower with very hard contractions. While in the shower, she was pushing and indicating that the baby was coming. The baby was born at 1035 hours with a heart rate of zero, no respirations, no blood in the cord and two loops wrapped tightly around the infant’s neck. The midwife untied the cord and tried chest compressions and suctioning for one minute, then records indicate that the midwife said she “quit.” The midwife did not call for an ambulance and no cord gases were drawn.

At 1055 hours, the second midwife arrived to a visibly upset family. The placenta arrived at 1105 hours. The baby was reported as being stillborn and non responsive to resuscitative efforts by the first midwife. The parents declined third stage management. At 1115 hours, vaginal bleeding was reported and Oxytocin was given. The midwives helped the mother upstairs to bed for monitoring of bleeding and to warm up.

At 1130 hours, the second midwife called Hospital A to report that they were transporting in with a stillborn. She then called 911 indicating that she needed transport to hospital for a stillborn and mother. At 1140 hours, the first EMS crew arrived. Two other EMS crews subsequently followed. The first crew assumed care of the baby and sought permission to commence resuscitative efforts. The other EMS crew members assessed the mother.

The midwife engaged in an argument with the EMS crews about where to transfer the patient and child. There was significant discussion about seeking authority to cease resuscitative efforts on the baby once it was learned how long the infant had actually been without signs of life. The midwife was demanding that the ambulance transfer the patients to Hospital A, while the EMS crews were being directed by a doctor to transport the patients to Hospital B.

The EMS crew was given permission to cease resuscitation efforts on the infant and the mother and stillborn were transported to Hospital B at 1230 hours.

The woman was treated as an outpatient at Hospital B and received her WinRho. She was then discharged to the care of the midwife. The woman remained in midwifery care until discharge at six weeks postpartum.

Postmortem

No post mortem was conducted.

Discussion

An ultrasound at 41 plus weeks may have shown greater than a three week variance in the calculated gestational age and estimated due date, however there is no indication that this was ever discussed with the woman, midwife or physician. A post dates pregnancy and a suspected small for gestation age foetus may have increased this woman's risks for a planned homebirth.

The question of whether this woman would have experienced a stillbirth if she had been induced cannot be addressed. The Society of Obstetricians and Gynaecologists of Canada (SOGC) Guideline for the Management of Pregnancy (September 2008), indicates that at 41+0 to 42+0 weeks, increased antenatal foetal surveillance that includes at least a non stress test and an assessment of amniotic fluid volume, is recommended. It further recommends that women should be offered induction at 41+0 to 42+ 0 as research indicates a decrease in perinatal mortality without increased risk of Caesarean section. In addition, women should be offered sweeping of membranes at 38 to 41 weeks following a risk-benefit discussion. This mother did have increased antenatal foetal surveillance and she had stretch and sweeps commencing 39 weeks 4 days.

The Society of Obstetricians and Gynaecologists of Canada (SOGC) guidelines state that counting up to six movements in a two-hour period offers short test duration, a proven track record, and a relatively low rate of alarm. Women should be informed that in most foetuses with a positive test (fewer than 6 movements in 2 hours), the result is often a false positive, and a good outcome ensues.

Women who report decreased foetal movements (< 6 distinct movements within 2 hours) should have a complete evaluation of maternal and foetal status, including non-stress test and/or biophysical profile. Prior to considering an intervention for foetal well-being, an anatomical scan to rule out a foetal malformation should be done, if one has not already been done.

In this case, contrary to the guideline, the mother was advised to drink juice, lie down and call back if no movement was detected.

The first midwife had many opportunities and clinical reasons to call an ambulance (e.g. no foetal heart detected, no second midwife at birth, baby not responsive at birth and inability to set up bag and mask for neonatal resuscitation). The second midwife also chose not to call an ambulance immediately upon her arrival at the scene 30 minutes after the birth. She waited another 30 minutes (now one hour after actual birth), to call the EMS.

The resuscitation performed by the attending midwife appears to be totally inadequate. The record indicates that the midwife unwrapped the nuchal cord that was void of blood. She then suctioned, although there was no notation of how the suctioning was done. The record indicates that the midwife did one minute of cardiac compressions, then "quit".

The midwife was obligated to perform neonatal resuscitation until the EMS arrived to assist and transport to a hospital. The midwife was certified to provide neonatal resuscitation and did not in any way offer the accepted resuscitative efforts for which she was trained.

Further notes in the clinical record of the resuscitation state that it was, “precipitous birth, no time for positive pressure ventilation equipment to be set up.” Suction was said to have been performed for one minute followed by one minute of cardiac compressions. The midwife had her bags in the house, yet never provided positive pressure ventilation. The time to locate and unpack and set up bag and mask, connect oxygen and open flow valve to perform Neonatal Resuscitation is less than 60 seconds. There were two other adults in the house at the birth that could have followed directions or assisted with the cardio pulmonary resuscitation while positive pressure ventilation was undertaken. It is not clear why the midwife felt she did not have time to set up the necessary equipment.

There was no notation on the record of the parents ever asking the midwife to cease attempts at resuscitation. Upon arrival of the EMS crews one hour after the birth, the parents gave permission for resuscitative efforts to be performed. The midwife did not communicate with the EMS that the infant had been deceased for almost an hour.

The argumentative behaviour between the midwives and the EMS was inappropriate. Midwives in Ontario, when dealing with an emergency situation, may direct an ambulance to the most appropriate, prepared and aware centre. This often happens in rural locations where the closest transfer point offers no obstetrical or neonatal services. In this case, the midwife did not require emergency transfer when she finally called an ambulance. Therefore, the need to direct the ambulance to a waiting and appropriate emergency service did not exist. The midwife may have wanted to direct the ambulance to a hospital where she had privileges, although this is not noted in the record.

At home deliveries, midwives should set up all resuscitative equipment and medications exactly as would be present in a birth room. The midwife in this case failed to do so.

The midwives in this case acted outside the scope of practice and standards and regulations of the profession of midwifery in Ontario by not providing the necessities of life when skilled and trained to do so, inappropriate declaration of a death and failure to transfer in a timely manner to a hospital in an emergency situation.

Recommendations

1. The midwives involved in this case should be referred to College of Midwives of Ontario for further investigation.

Summary of Recommendations – Maternal

Case	Cause of Death	Recommendations
M-1	Staphylococcus aureus endometritis and sepsis.	None
M-2	Pulmonary Embolus	None
M-3	Haemoperitoneum following Caesarean section	<ol style="list-style-type: none"> 1. The _____ obstetrical unit should review its PACU protocols, particularly as pertains to notification of unstable vital signs to both the anaesthetist and obstetrician. 2. Obstetricians are reminded of the utility of applying manual compression to the descending aorta to control pelvic bleeding as an aid in the establishment of a circulating blood volume during resuscitation of hemorrhagic shock and Disseminated Intravascular Coagulopathy.
M-4	Blood loss due to ruptured right ectopic tubal pregnancy.	None
M-5	Multi-organ failure secondary to DIC due to postpartum hemorrhage from placenta previa and placenta accreta.	None
M-6	Septicemia due to infective endocarditis	<ol style="list-style-type: none"> 1. Caregivers are reminded of the importance of properly educating patients on steroids to seeking care for fevers or other signs of infection. Caregivers are reminded that chronic steroid usage can mask the usual signs of infection. Caregivers are directed to review the “Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008” at www.survivingsepsis.org.
M-7	Complications of intra-abdominal	None

Case	Cause of Death	Recommendations
	hemorrhage.	
M-8	Delayed hanging, with hypoxic ischemic encephalopathy	1. Obstetrical Care Providers are reminded that psychiatric disease may complicate pregnancy and may be a cause of maternal mortality.
M-9	<ol style="list-style-type: none"> 1. Severe myometritis with inflammation of the cervix 2. Necrotizing fasciitis involving the vulva, right thigh and upper inner thigh 3. DIC with micro-thrombi in various organs 4. Intravascular bacterial dissemination 5. Acute tubular necrosis 6. Early centrilobular necrosis of the liver 7. Premortem blood cultures positive for strep A pyogenes 8. Generalized edema and effusions in pleural cavities, peritoneal cavity and pericardium 9. Bilateral pulmonary edema 	None
M-10	Bilateral central and peripheral pulmonary emboli and thrombosis of the pelvic veins.	None
M-11	Intra-abdominal bleeding and DIC due complications of Caesarean section.	<ol style="list-style-type: none"> 1. All health care providers are reminded to review the patients' vital signs for symptoms of postoperative blood loss and respond to the nurses concerns with other investigations to confirm the problem or reassure the other caregivers. 2. The nurses of this hospital should be

Case	Cause of Death	Recommendations
		<p>aware of the options they have for calling the department chief or chief of staff if they are concerned about a patient and feel the attending physician is not acting appropriately.</p> <p>3. The hospital is requested to do a Quality of care review of his case.</p>
M-12	Severe chest injuries due to blunt force trauma to the chest and abdomen.	None
M-13	Hypertensive Heart Disease.	None
M-14	Sepsis due to infective endocarditis involving both prosthetic mitral and aortic valves.	None
M-15	Aneurysm of the left ventricle of the heart.	None
M-16	Massive ischemic stroke of the right hemisphere 16 days post partum.	1. The Chief Coroner's office should investigate the circumstances relating to the delayed reporting of this maternal death and the reason why an autopsy
M-17	Hypoxic ischemic encephalopathy due to submassive hepatic necrosis of unknown etiology after a recent Caesarean section at	None
M-18	Acute cardiovascular collapse, probably from amniotic fluid	None
M-19	Massive post partum hemorrhage secondary to uterine atony.	<p>1. Obstetrical care providers are reminded that post partum hemorrhage complicates up to 5% of deliveries and remains a cause of maternal morbidity and mortality. All obstetrical care providers at the hospital should consider review of the ALARM course and develop protocols dealing with obstetrical emergencies.</p> <p>2. Obstetrical care providers are reminded</p>

Case	Cause of Death	Recommendations
		that otherwise healthy women can tolerate a significant blood loss prior to becoming symptomatic and aggressive attention to the ABC's is required.
M-20	Suspected Peripartum Cardiomyopathy	<ol style="list-style-type: none"> 1. Health care providers are reminded that peripartum cardiomyopathy can occur up to five months post partum. 2. The Chief of Emergency Medicine at Hospital B should review this case particularly with regard to triaging and timelines for assessment.
M-21	Blunt impact head injuries.	None

Summary of Recommendations – Neonatal

Case	Cause of Death	Recommendations
N-1	Hypoxic-ischemic injury.	<ol style="list-style-type: none"> 1. Obstetrical care providers are advised to become familiar with the current classification of intrapartum electronic foetal monitoring and the recommended actions in the setting of atypical and abnormal tracings. 2. Care providers are reminded that augmentation of labour in the presence of concerns regarding foetal well-being should be commenced with extreme caution.
N-2	Perinatal Asphyxia With Meconium Aspiration	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded of the importance of clear documentation. 2. Obstetrical care providers are reminded of need to identify early at risk pregnancy and offer as timely as possible consultation to help plan further management. This is especially important in smaller centres which do not have on site access to more specialized care. 3. Obstetrical care providers are reminded of the importance of identifying and acting on abnormalities in foetal heart monitoring. 4. Paediatric health care providers are reminded that early blood gas estimation is essential in dealing with compromised babies as well as close monitoring of vital signs.

Case	Cause of Death	Recommendations
N-3	Acute hypoxic ischaemic encephalopathy due to severe prematurity and placental abruption	<ol style="list-style-type: none"> 1. Obstetrical health care providers are reminded of their duty to assess, treat and document in a timely fashion. 2. Nursing staff are reminded that if the doctor fails to respond appropriately they may seek other care for the patient. 3. The local hospital should establish guidelines for nurses to use when they are unable to get a satisfactory response from an attending physician.
N-4	Hypoxic-ischemic encephalopathy due to abruption placenta.	<ol style="list-style-type: none"> 1. The obstetrical care providers in this case should be required to demonstrate competence in the interpretation and management of foetal heart rate tracings. 2. Obstetrical care providers are reminded that cervical ripening agents are contraindicated in the absence of normal tests of foetal well-being.
N-5	Hypoxic-ischemic complications of perinatal asphyxia of undetermined etiology.	None

Case	Cause of Death	Recommendations
N-6	Complications of perinatal asphyxia due to placental chorioamnionitis and cord vasculitis with E. coli septicaemia and preterm premature rupture of the membranes	<ol style="list-style-type: none"> 1. The obstetrician should review Foetal Health Surveillance to ensure that they are able to identify abnormal tracings and proceed with appropriate interventions. 2. The hospital should review Foetal Health Surveillance with the labour room staff and with the obstetrician to ensure that staff are able to identify abnormal tracings and proceed with appropriate interventions. 3. The hospital should review their policy for foetal heart rate monitoring to be sure that continuous Electronic Foetal Monitoring is used when preterm labour is suspected or confirmed and that documentation and terminology is consistent with current standards and begins with admission to the labour room. 4. The hospital should review their policy for medication administration in labour to ensure that a foetal heart rate assessment is done prior to the administration of narcotics. 5. Caregivers are reminded that all notes and orders should be dated and timed.
N-7	Perinatal asphyxia due to undetermined consequences.	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded that foetal well-being should be assured in the form of a normal foetal heart rate tracing prior to the commencement of Oxytocin. 2. Obstetrical care providers are advised to be familiar with the current guidelines for action in the setting of atypical and abnormal EFM tracings. 3. Obstetrical providers should, to the degree possible given the resources within their hospital, coordinate activities

Case	Cause of Death	Recommendations
		<p>with anaesthesia and Paediatrics in advance if there is a high risk situation in the delivery room.</p> <ol style="list-style-type: none"> 4. Obstetrical care providers are reminded of the importance of determining foetal position prior to performing an instrumental delivery. 5. The Hospital should ensure that health care providers who may be called upon to resuscitate newborns should have up to date NRP provider status.
N-8	Septic complications of Acute Gastric Perforation	<ol style="list-style-type: none"> 1. Paediatric care providers are reminded that infants with refractory sepsis may be infected with an antibiotic resistant organism and standard antibiotic regimens may be ineffective. Empirical treatment with higher-level antibiotics should be considered under these circumstances. 2. Blood cultures and other appropriate clinical samples should be take prior to institution of antibiotic treatment in neonates with suspected sepsis and repeated if the baby's clinical condition fails to improve as expected. A revision to the antibiotic regimen should be considered after samples are collected.
N-9	Congenital toxoplasmosis with necrotizing pneumonia	<ol style="list-style-type: none"> 1. Obstetrical caregivers are reminded of the importance of acting on atypical and abnormal foetal status in preterm infants even while transfer is arranged. 2. The Ministry of Health and Long Term Care should urgently review the bed capacity for severely sick neonates needing transfer to neonatal units throughout the province. A review should also be conducted on the availability of transport teams that service neonatal units.

Case	Cause of Death	Recommendations
N-10	Complications of prematurity.	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded to have a high index of suspicion for premature labour, particularly in the presence of risk factors. 2. The hospital should review its guidelines as to which pregnant patients presenting to the emergency department would best be assessed in a dedicated obstetrical triage unit. 3. Obstetrical care providers are reminded of the utility of foetal Fibronectin testing in the assessment of possible preterm labour. 4. Paediatric care providers are reminded to consider the possibility of pneumothorax in any ventilated neonate who experiences an acute cardiorespiratory deterioration. 5. This report should be forwarded to the Chair of the Paediatric Death Review Committee.
N-11	Cerebral hypoxic ischemic injury.	<ol style="list-style-type: none"> 1. Midwives should collect cord gases at all births. 2. Obstetrical care givers are cautioned from adhering to a strict definition of the latent phase of labour. Long, painful contractions “despite irregularity” that cause significant cervical change should be regarded as active labour. Considerations of care for a woman in active labour include assessment of progress, foetal surveillance and appropriate interventions provided in a timely manner.

Case	Cause of Death	Recommendations
N-12	Prolonged rupture of membranes with ascending infection, acute chorioamnionitis and foetal sepsis.	<ol style="list-style-type: none"> 1. Women planning a home birth should be informed of the risks of infection with prolonged rupture of membranes. 2. Obstetrical care providers are reminded that cultures for GBS may give false negative results which could be due to collection, transporting, processing and reporting results. Other possible causes are collection longer than 5 weeks before delivery, and transient colonisation.
N-13	Perinatal asphyxia.	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded that ultrasound findings of severe IUGR at term is an indication for urgent delivery. 2. The Hospital should do a QCIPPA review of this case and inform the Regional coroner of any changes to policy and procedures based on the results of the review. The Regional coroner will update the committee on the report from the hospital review. <p>Note: The hospital reviewed this death and education directed specifically at the obstetrician involved and at the departmental level was completed.</p>
N-14	Acute peri-natal hypoxic/ischemic injury.	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded of the new SOGC guidelines for the diagnosis and management of Vasa Previa (JSOGC 31:8, 2009).
N-15	Hypoxic-ischaemic encephalopathy.	<ol style="list-style-type: none"> 1. The hospital and mid-wifery clinic should review their procedures for documenting and recording the actions of all members of the medical care team.

Case	Cause of Death	Recommendations
N-16	Acute severe respiratory distress syndrome following a left pneumothorax in a newborn” due to “severe intrauterine growth restriction.	<ol style="list-style-type: none"> <li data-bbox="802 281 1430 422">1. Midwives are reminded of the College Of Midwives of Ontario requirement for level 1 consultations to be completed and documented. <li data-bbox="802 443 1430 583">2. Obstetrical healthcare providers are reminded of the importance of accurate assessment and documentation of prenatal visits. <li data-bbox="802 604 1430 745">3. Obstetrical healthcare providers are reminded of the importance of ordering ultrasounds when there are deviations in the growth rate of the baby.

Summary of Recommendations – Stillbirths

Case	Cause of Death	Recommendations
S-1	Perinatal asphyxia.	<ol style="list-style-type: none"> 1. Obstetrical caregivers are reminded that interpretation of the foetal heart rate monitor strip includes assessment and documentation of the variability of accelerations and decelerations. The interpretation of decelerations also requires the documentation of uterine contraction activity with a monitor or by palpation. 2. The hospital should ensure that the labour records have place to document each of these aspects of foetal health surveillance as well as the nursing interpretation of these findings and the action plan.
S-2	Intrapartum death secondary to perinatal asphyxia.	<ol style="list-style-type: none"> 1. Family doctors, nurse practitioners and midwives should consider consultation with a specialist physician for morbidly obese women at intake into their maternity care. Obstetricians should refer such woman to the anaesthesiologists in the intended birth hospital. 2. The hospital should review current foetal surveillance protocol and indications for Electronic Foetal Monitoring and ensure it is referenced and edited and updated accordingly with current guidelines such as “Foetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline”, SOGC 2008. 3. Caregivers are reminded that use of an electronic foetal monitor for intermittent auscultation protocol is not appropriate. 4. If a caregiver feels EFM is indicated and a suitable tracing cannot be obtained, referral for more invasive monitoring should be considered.

Case	Cause of Death	Recommendations
		<p>5. The hospital should review the requirements for labour induction or augmentation with oxytocin, including assessment of foetal status, with this midwife and ensure that policies and procedures are written to guide such actions.</p> <p>6. It is recommended that the College of Midwives of Ontario include the practice of placement of scalp electrodes within their scope and training.</p> <p>7. When abnormal foetal status is diagnosed and delivery is expected, the nursing staff should routinely notify the pediatrician on call to attend.</p>
S-3	Stillbirth – No Specific Etiology	<p>1. The doctor involved in this case should be referred to the CPSO for review of both his record keeping and care as both fell below the standard expected of an obstetrical care provider in Ontario in 2007/2008.</p>
S-4	Foetal Haemorrhage due to Velamentous insertion of umbilical cord.	<p>1. The hospital should review the system for contacting the on call anaesthetist and the expectations for their availability.</p>
S-5	Intrauterine Asphyxia.	None
S-6	Undetermined.	None
S-7	Peri-partum asphyxia.	<p>1. Obstetrical care providers are reminded that the possibility of umbilical cord prolapse should be considered in the presence of variable decelerations, particularly when the presenting part is not in the pelvis.</p> <p>2. Obstetrical care providers are reminded that ambulation in the setting of ruptured membranes and a high presenting part increases the risk of cord prolapsed.</p> <p>3. Obstetrical care providers are reminded</p>

Case	Cause of Death	Recommendations
		<p>of the importance of effective interpretation of EFM tracings, consideration of potential causes and additional clinical actions required based on the SOGC Clinical Practice Guideline for Foetal Health Surveillance, September 2007.</p> <p>4. Obstetrical care providers are reminded of the importance of full and accurate documentation.</p>
S-8	Undetermined.	None
S-9	Asphyxia.	<p>1. The midwives involved in this case should be referred to College of Midwives of Ontario for further investigation.</p>

Glossary of Terms

AFE	amniotic fluid embolism
AFP	alpha -fetoprotein
ANA	antinuclear antibody
APTT	activated partial thromboplastin time
ARM	artificial rupture of membranes
AST	Aspartate aminotransferase
AVM	arteriovenous malformation
BID	two times a day
BMI	body mass index
BP	blood pressure
BPP	biophysical profile
CBC	complete blood count
CK	Creatine Kinase
CNS	central nervous system
CPAP	Continuous positive airways pressures
CPR	cardio pulmonary resuscitation
CT (CAT)	computerized axial tomography
CXR	chest x-ray
DIC	disseminated intravascular coagulation
ECG	electrocardiogram
EDC	estimated date of confinement
EDD	estimated date of delivery

EFW	estimated foetal weight
EMR	electronic medical record
ETT	endotracheal tube
FFP	fresh frozen plasma
GAS	Group A streptococcus
GBS	group B streptococcus
GCT	glucose challenge test
GTT	glucose tolerance test
GSC	Glasgow Coma Scale
HCG	human chorionic gonadotropin
HELLP	haemolysis elevated liver enzymes low platelets
HIV	Human Immunodeficiency Virus
ICH	intracerebral haemorrhage
ICU	intensive care unit
INR	International Normalization Ratio
IPS	integrated pregnancy screening
IUGR	intra uterine growth retardation
LDH	Lactate dehydrogenase
LMP	last menstrual period
MCAD	medium chain acyl CoA dehydrogenase
MFM	maternal foetal medicine
MRI	magnetic resonance imaging
NST	non-stress test

OGTT	oral glucose tolerance test
OR	operating room
PIH	pregnancy induced hypertension
PPROM	pre-term premature rupture of membranes
PPV	positive pressure ventilation
PTT	partial thromboplastin time
QID	four times a day
RR	respiratory rate
SC	serum creatinine
SFH	symphysis fundal height
SLE	systemic lupus erythematosus
SOGC	Society of Obstetricians and Gynaecologists of Canada
STSS	Streptococcal Toxic Shock Syndrome
TID	three times a day
TSS	Toxic Shock Syndrome
UAL	umbilical arterial line
UVC	umbilical venous catheter
VBAC	Vaginal birth after Caesarean
V/Q	ventilation-perfusion
VSA	vital signs absent
VSD	ventricular septal defect
WBC	white blood cell
WNL	within normal limits

