



Maternal and Perinatal Death Review Committee

2011 Annual Report

Office of the Chief Coroner
Province of Ontario
December 2012

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

Message from the Chair

The Maternal and Perinatal Death Review Committee (MPDRC), together with its predecessor, the Obstetrical Care Review Committee, has been providing expert advice to coroners' investigations in Ontario since 1994.

Through an agreement with Health Canada to assist in the identification and prevention of maternal deaths in Canada, the Office of the Chief Coroner for Ontario has established a policy to investigate and review all maternal deaths that occur after 20 weeks gestation, during delivery or immediately following delivery, and up to 42 days postpartum. Any deaths after 42 days and up to 365 days post delivery are reviewed if the cause of death is directly related to the pregnancy or a complication of the pregnancy.

Each year, a small percentage of stillbirths and perinatal deaths investigated by the Office of the Chief Coroner (OCC), have issues identified by Regional Supervising Coroners that bring them to the attention of the MPDRC. In many cases, the initial concerns about the care the mother and/or child received are raised by investigating coroners and families.

The MPDRC is comprised of well respected and experienced experts representing the fields of obstetrics, maternal-fetal medicine, midwifery, perinatal nursing, obstetrical anaesthesiology, pathology, paediatrics and family medicine.

Since its inception, the Committee has reviewed a total of 259 cases and generated 466 recommendations towards the prevention of stillbirths and deaths involving mothers and neonates. In 2011, 30 cases were reviewed and 47 recommendations were made.

The top areas of concern identified in recommendations made from 2004-2011 relate to: medical and nursing issues; policy and procedures; communications/documentation; and diagnosis and testing involving electronic fetal monitoring. As we strive towards reducing similar deaths and improving the quality of care provided to mothers and infants, the identification of these trends will help guide the direction of future recommendations and initiatives of the MPDRC and increase awareness and prompt action by stakeholders within the obstetrical care community.

It is an honour to participate in the work of the MPDRC and I am grateful for the commitment of its members to the people of Ontario. I would also like to acknowledge Ms. Kathy Kerr, Executive Lead. Without her efforts, the work of the committee and the production of this report would not be possible.

I would like to acknowledge Dr. Catherine MacKinnon, an obstetrician from a level II facility in Brantford, who retired from the MPDRC in 2011 after many years on the committee. The Office of the Chief Coroner has benefited immensely from her down to earth and expert reviews, and we wish her all the best in her retirement.

It is my privilege to present to you the 2011 Annual Report of the Maternal and Perinatal Death Review Committee (MPDRC).

A handwritten signature in black ink, appearing to read "Rick Mann". The signature is fluid and cursive, with a long horizontal stroke at the end.

Rick Mann, MD, CCFP, FCFP
Chair, Maternal and Perinatal Death Review Committee

Committee Membership (2011)

Dr. Michael Dunn	Neonatologist (Level 3)
Dr. Karen Fleming	Family Physician (Level 3)
Dr. Robert Gratton	Maternal Fetal Medicine
Dr. Steven Halmo	Obstetrician (Level 2)
Ms. Susan Heideman	Perinatal Nurse
Dr. Robert Hutchison	Obstetrician (Level 3)
Dr. Sandra Katsiris	Anesthesiologist
Ms. Michelle Kryzanasuskas	Midwife (Rural)
Dr. Catherine MacKinnon	Obstetrician (Level 2)
Dr. Dilipkumar Mehta	Paediatrician (Level 2)
Ms. Linda Moscovitch	Midwife (Urban)
Dr. Toby Rose	Forensic Pathologist
Dr. Gillian Yeates	Obstetrician (Level 1)
Dr. Rick Mann	Chairperson Regional Supervising Coroner
Ms. Kathy Kerr	Executive Lead

Executive Summary

- In 1994, the Office of the Chief Coroner established the Obstetrical Care Review Committee. In 2004, the name of the committee was changed to the Maternal and Perinatal Death Review Committee (MPDRC).
- The purpose of the MPDRC is to assist the Office of the Chief Coroner in the investigation, review and development of recommendations directed towards the prevention of future similar deaths relating to all maternal deaths (irrespective of cause) and stillbirths and neonatal deaths where the family, coroner or Regional Supervising Coroner have concerns about the care that the mother or child received.
- Since 2004, the MPDRC has reviewed 259 cases and generated 466 recommendations aimed towards the prevention of future similar deaths.
- Each year, an average of 32 cases are reviewed and 58 recommendations are made.
- The top areas of concern identified in recommendations made from 2004-2011 relate to: medical and nursing issues; policy and procedures; communications/documentation; and diagnosis and testing involving electronic fetal monitoring.
- In 2011, 30 cases were reviewed and 47 recommendations were made.
- Of the 30 cases reviewed in 2011, 3 were maternal, 14 were neonatal and 13 were stillborn.

Chapter One: Introduction

Purpose

In 1994, the Office of the Chief Coroner established the Obstetrical Care Review Committee. In 2004, the name of the committee was changed to the Maternal and Perinatal Death Review Committee (MPDRC).

The purpose of the MPDRC is to assist the Office of the Chief Coroner in the investigation, review and development of recommendations directed towards the prevention of future similar deaths relating to all maternal deaths irrespective of cause. This includes all deaths during pregnancy and the post-natal period (which is considered to be up to 42 days after delivery). Any deaths after 42 days and up to 365 days post delivery are reviewed if the cause of death is directly related to the pregnancy or a complication of the pregnancy.

The Committee reviews stillbirths and neonatal deaths where the family, coroner or Regional Supervising Coroner have concerns about the care that the mother or child received.

Findings of legal responsibility or conclusions of law are not permitted under the *Coroners Act*.

Definition of Maternal, Stillborn and Neonate

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.” Deaths involving women who are pregnant, but where the death was not attributed to pregnancy are noted for statistical purposes only and no formal review is conducted.

Maternal deaths are classified by the following criteria:

- Antepartum - >20 weeks gestation

- Intrapartum - during delivery or immediately following delivery
- Postpartum - < 42 days after delivery

This Committee does *not* review late maternal deaths occurring >42 days unless the cause of death is directly related to the pregnancy or a complication of the pregnancy..

Stillbirth is defined as the complete expulsion or extraction from the mother of a product of conception either after the 20th week of pregnancy or after the product of conception has attained the weight of 500 grams or more, and where after such expulsion or extraction there is no breathing, beating of the heart, pulsation of the umbilical cord or movement of voluntary muscle.

Perinatal/Neonatal deaths are defined as death during, at the time of, or shortly after birth, including home birth (perinatal) and deaths within the first seven days after birth (neonatal).

Aims and Objectives

1. To assist coroners in the Province of Ontario to investigate maternal and perinatal deaths and to make recommendations that may prevent similar deaths.
2. To provide expert review of the care provided to women during pregnancy, labour and delivery, and 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.¹
4. To inform doctors, midwives, nurses, institutions providing care to pregnant and

¹ Special Report on Maternal Mortality and Severe Morbidity in Canada, Health Canada, 2004.

postpartum women and newborns, and relevant agencies and Ministries of Government about hazardous practices and products identified during case reviews.

5. 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.
6. To help identify the presence or absence of systemic issues, problems, gaps, or shortcomings of each case to facilitate appropriate recommendations for prevention.
7. To help identify trends, risk factors, and patterns from the cases reviewed to make recommendations for effective intervention and prevention strategies.
8. To conduct and promote research where appropriate.
9. To stimulate educational activities through the recognition of systemic issues or problems and/or referral to appropriate agencies for action.
10. Where appropriate, to assist in the development of protocols with a view to prevention.
11. Where appropriate, to disseminate educational information.

Note: All of the above described objectives and attendant committee activities are subject to the limitations imposed by the Coroners Act of Ontario and the Freedom of Information and Protection of Privacy Act.

Structure and Size

The Committee membership consists of respected practitioners in the fields of specialty including: obstetrics, family practice, specialty neonatology, community pediatrics, pediatric and maternal pathology, anesthesiology, midwifery and obstetrical nursing. The membership is balanced to reflect wide and practicable geographical representation as well as representation from all levels of institutions providing obstetrical care including teaching centers to the extent possible. The Chairperson will be a Deputy

Chief Coroner or Regional Supervising Coroner or other person designated by the Chief Coroner.

Other individuals are invited to the Committee meetings as necessary on a case by case basis (e.g. investigating coroner, Regional Supervising Coroner, other specialty practitioner relevant to the facts of the case, etc.).

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 other members. 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 then sent to the referring Regional Supervising Coroner who may conduct further investigation (if necessary). Recommendations are distributed by the Committee Chair to agencies and organizations who may be in a position to effect the implementation of such recommendations. Organizations are asked to respond back within one year with the status of implementation of recommendations.

Where 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.

Limitations

This Committee is advisory to the Coroner system and will make recommendations to the Chief Coroner through the Chairperson.

The consensus report of the Committee is limited by the data provided. Efforts are made to obtain all relevant data.

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*, the *Freedom of Information and Protection of Privacy Act* and the *Personal Health 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 written 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 a 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 are made following 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.

Chapter Two: Statistical Overview (2004-2011)

The MPDRC (and previously the Obstetrical Care Review Committee) has generated recommendations since being established in 1994. Over time, not only has the Committee evolved, but so too have medical technologies, policies, procedures and public and professional attitudes towards maternal and perinatal care in the province. In order to provide an analysis that is reflective of more current values and attitudes, the statistical analysis contained within this annual report will focus on cases reviewed and recommendations made since 2004.

From 2004-2011, the MPDRC has reviewed a total of 259 cases. Of these cases, 84 (32%) were maternal, 109 (42%) were neonatal and 66 (25%) were stillbirths. These numbers reflect the policy of the Office of the Chief Coroner to review all maternal deaths. Deaths involving women who are pregnant, but where the pregnancy did not cause or contribute to the death, are noted, but do not undergo formal review (and thus are not reflected in these statistics). Neonatal and stillbirth reviews are conducted only when the family, coroner or Regional Supervising Coroner have concerns about the care that the mother or child received.

The number of cases noted in Chart One is based on the year the case was reviewed, which, in many cases, is not the same year in which the death actually occurred.

Chart One: MPDRC - # of Cases Reviewed (2004-2011)

	2004	2005	2006	2007	2008	2009	2010	2011	Total
Total # of cases reviewed	30	30	25	27	30	46	41	30	259
Maternal	10	12	4	15	8	21	11	3	84
Neonatal	12	11	13	12	12	16	19	14	109
Stillbirth	8	7	8	0	10	9	11	13	66

Chart One indicates that the number of total cases reviewed from 2004-2011 has varied from a low of 25 cases in 2006, to a high of 46 cases in 2009. This variance is likely reflective of committee administrative practices (e.g. time required for processing of review materials and compilation of final reports). On average, 32 cases are reviewed per year.

Chart Two: MPDRC - # of Recommendations (2004-2011)

	2004	2005	2006	2007	2008	2009	2010	2011	Total
Total # of Recommendations made	56	71	58	36	46	69	83	47	466
Maternal	11	19	5	16	3	12	15	2	83
Neonatal	30	31	31	20	24	41	48	26	251
Stillbirth	15	21	22	0	19	16	20	19	132

Chart Two indicates that the MPDRC has generated a total of 466 recommendations from 2004-2011. From this total, 83 (18%) were related to maternal cases, 251 (54%) from neonatal cases and 132 (28%) from stillbirth cases. Consistently over the years, the majority of cases and recommendations relate to reviews of neonatal deaths. On average, 58 recommendations are made per year.

Upon reviewing the recommendations that have been made, it is evident that certain areas of concern or themes have consistently emerged over time. The following general areas of concern have been identified:

- medical (e.g. medical or nursing decisions)
- policy and procedure (e.g. adherence or development of policy and procedures)
- communication/documentation (e.g. sharing and documenting information)
- quality (e.g. quality of care reviews)
- diagnosis and testing (e.g. interpretation of laboratory results)
- diagnosis and testing – specifically Electronic Fetal Monitoring (e.g. interpretation of EFM results)
- education/training (e.g. continuing education)
- resources (e.g. access and allocation of resources)
- transfer (e.g. movement of patients)
- other (e.g. referral to another committee for review)

Chart Three: MPDRC – Number and percentage of recommendations based on area of concern/theme (2004-2011)

	Maternal	Neonatal	Stillborn	Total	% of Total
Medical	36	47	29	112	24%
Policy and procedure	20	44	27	91	20%
Communications/documentation	7	42	25	74	16%
Quality	11	18	7	36	8%
Diagnosis and testing	1	30	13	44	9%
Diagnosis and testing - EFM	0	39	23	62	13%
Education/Training	1	15	4	20	4%
Resources	2	11	2	15	3%
Transfer	4	5	2	11	2%
Other	0	1	0	1	0%
				466	

The % of recommendations based on area of concern or theme is demonstrated in the following pie chart:

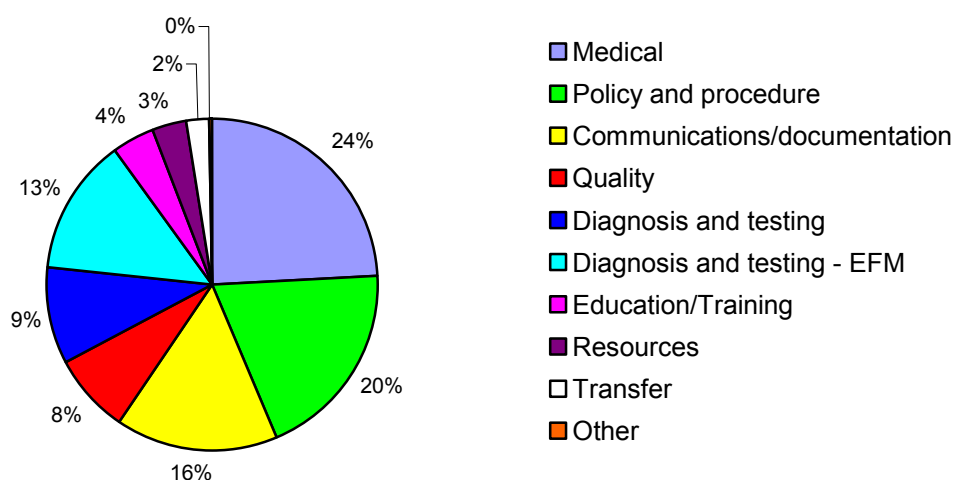


Chart Three demonstrates that 24% of all recommendations made by the MPDRC from 2004-2011 relate to improving or addressing medical/nursing issues. An additional 20% of the recommendations pertain to the development of, or adherence to, policies and procedures and 16% to communication and/or documentation and in particular, the timely and accurate sharing of information between healthcare providers and with the patient.

One area of specific concern that has been identified over the past few years relates to the use of Electronic Fetal Monitoring (EFM) technology, how EFM results are interpreted by obstetrical care providers and what follow-up actions are taken in response to the findings. From 2004-2011, there have been 62 recommendations made specifically pertaining to EFM. Further discussion on EFM is included in **Chapter Four: Lessons Learned from MPDRC Reviews**.

Chapter Three: Summary of Cases Reviewed in 2011

This annual report includes summaries of reviews conducted by the Maternal and Perinatal Death Review Committee in 2011. Cases reviewed may involve deaths that occurred in previous years.

Total number of cases reviewed:	30
Total number of recommendations:	47
Number of maternal cases reviewed:	3
Number of maternal cases noted for statistical purposes only*:	7
Total number of maternal deaths:	10
Number of recommendations from the maternal deaths reviewed:	2
Number of neonatal cases reviewed:	14
Number of recommendations from the neonatal deaths:	26
Number of stillborn cases reviewed:	13
Number of recommendations from the stillborn cases:	19

* 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.” Deaths involving women who are pregnant, but where the pregnancy did not attribute to the death, are noted for statistical purposes only and a formal review is not conducted.

The following summaries include a detailed description of all cases – maternal, neonatal and stillbirth - reviewed by the MPDRC in 2011, together with recommendations made towards the prevention of future similar deaths. All personal identifiers including the names of patients, hospitals, practices and healthcare providers have been redacted for privacy.

Case Summaries: Maternal Deaths

Reporting of Maternal Deaths in Ontario

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” (see Section 10 (1) (c) of the Coroners Act). Deaths involving women who are pregnant, but where the pregnancy did not cause or contribute to the death, are noted for statistical purposes only and no formal review is conducted.

The following maternal deaths are noted for statistical purposes only - no formal review was conducted:

1. OCC file: 2010-3911

The deceased was a 28-year-old P₂G₁ with a history of Hepatitis B. She had gestational diabetes.

Cause of death: Hepatocellular carcinoma (Hep B)

2. OCC file: 2009-14617

The deceased was a 23-year-old previously healthy woman who presented to hospital complaining of a three hour severe headache. She was five days post-partum, having delivered a normal, full-term child vaginally under epidural.

Cause of death: venous sinus thrombosis due to acute streptococcus pneumoniae meningitis

3. OCC file: 2011-2047

The deceased was a 28-year-old woman who presented to hospital with blue feet and hands and with abdominal pain.

Cause of death: Septic shock secondary to Streptococcus pyogenes Group A infection in a pregnant woman at 39 weeks.

4. OCC file: 2011-5319

The deceased was a 43-year-old primiparous woman who was being followed by an obstetrician as she was considered a high risk pregnancy. She suffered from being overweight, gestational diabetes on insulin and hypertension. She was brought to hospital by ambulance complaining of a cough and shortness of breath.

Cause of death: Pulmonary embolus

5. OCC file: 2011-3694

The deceased was a 36-year-old primigravida who conceived through a fertility drug. At 15 weeks gestation, one of the twins had intrauterine demise without expulsion or bleeding.

The deceased had a spontaneous rupture of membranes with clear fluid. She was allowed to continue into labour, her condition being stable. Post Caesarean section, there was persistent uterine atony with persistent bleeding despite intensive attempts to 'firm' up the uterus. A hysterectomy was performed. It was noted that there was persistent bleeding with hypotension post hysterectomy. A vascular surgeon was requested to attend. It was noted that the iliac vessel was 'nicked'. The deceased received blood and fluid replacement during this period. She went on to develop disseminated intravascular coagulopathy and was transferred to the intensive care unit for further aggressive resuscitation, despite which she succumbed.

Cause of death: amniotic fluid embolism

6. OCC file: 2010-9114

The deceased was a 39-year-old female who was in her 8th month of pregnancy. This was her third pregnancy. She lived in a home with her two children (ages 14 years and 6 years), a cousin, and another relative who was trained as a nurse. EMS was called

when the deceased was short of breath and collapsed.

The deceased was originally from Grenada and did not have any prenatal care in Canada.

Cause of death: hypertensive heart disease

7. OCC file: 2011-3326

The deceased was a 20-year-old female who was found dead beside a roadway. She had been reported missing several weeks prior. The deceased was pregnant and it was believed that she planned on terminating the pregnancy.

Cause of death: unascertained

The following cases received full reviews by the Maternal and Perinatal Death Review Committee:

Case: 2011-M-1
OCC file: 2010-500

History

The deceased was a 22-year-old woman who died five days postpartum. Her past medical history included a history of asthma as a child, recurrent urinary tract infections and irritable bowel syndrome. She was one of a set of twins born prematurely and was of slight stature (4 feet 11 inches tall and weight 85 pounds at her first prenatal visit). She had seen dieticians regarding attempts to gain weight. She had bunions and metatarsal primus varus deformity of her feet and had surgery for left forefoot reconstruction at the age of 16. Post operatively, she continued to have disabling foot pain. She had many specialist consultations. She was found to have evidence of Reynaud's on testing by the vascular surgeon and referral to a rheumatologist was made, but never attended. Her family physician ordered a rheumatology screen which was negative. She had a colposcopy for abnormal pap smears.

The woman was under the care of a midwife during her pregnancy. All routine prenatal investigations were normal except for a swab for bacterial

vaginosis which was treated in the first trimester. She was non-immune to rubella. She was referred to an obstetrician at 31 weeks when an ultrasound revealed bilateral club feet in the fetus. She declined referral to a genetics clinic.

At 37 weeks gestation, she presented to the emergency department at her local general hospital with an acute spontaneous tension pneumothorax requiring urgent chest tube placement. She was transferred to a neighbouring community with a larger hospital for care by a cardiovascular thoracic surgeon. The obstetrician on call was also consulted. The patient was stable and the plan was to await spontaneous labour. Her water broke five days later and arrangements were made for Caesarean section. A healthy male infant was delivered with bilateral club feet.

Postpartum, the mother's chest tube was clamped and subsequently removed on the second postoperative day. Chest x-rays the next day were normal. The patient was discharged home on the fourth day.

On the fifth day post partum, the woman developed flank and lower abdominal discomfort. She collapsed and became unresponsive. Paramedics found her to have a thready pulse, no respirations and no response to pain stimulus. She was transported to the Emergency Room by ambulance and CPR was commenced. She was intubated, but vascular access proved very difficult. Intraosseous access was established in both lower extremities. Aggressive resuscitation continued in the Emergency Department. The Emergency Department physician felt the patient was hypovolemic, but no source of blood loss could be determined. An ultrasound exam showed no evidence of free fluid in the abdomen or pericardial effusion. There was no evidence of pneumothorax. Attempts at resuscitation were halted after 53 minutes.

Post mortem

Post mortem findings: Acute aortic dissection at the aorto-iliac bifurcation with massive retroperitoneal haemorrhage.

Cause of Death: Exsanguination due to Acute Dissection of the Aorta

Discussion

This patient died as a result of aortic dissection and retroperitoneal blood loss. This condition often happens suddenly without warning. There was little that could be done for the patient by the time the EMS team arrived at the scene.

Recommendations

No recommendations.

Case: 2011-M-2
OCC file: 2008-5178

Antenatal History

The deceased was a 35-year-old G₄P₂ with an Estimated Date of Delivery (EDD) of August 7, 2008. Her past medical history consisted of a first trimester spontaneous abortion, a term pregnancy delivered vaginally for a 7 lb infant in 1999 and a second term pregnancy delivered vaginally of a 7.5 lb infant in 2005. The pregnancies were uncomplicated. Other past medical history was unremarkable. Family history was positive for deep vein thrombosis (DVT) in her mother associated with a hospitalization.

On April 5, 2008 at 22 weeks gestation, the woman experienced two episodes of fainting while at home. The second episode was witnessed by her husband and was associated with seizure-like activity. 911 was called and upon arrival, the paramedics observed a third episode of fainting. She was taken to the Emergency Department (ED) of the local hospital.

On arrival in the ED, she was hypotensive with a BP 95/68, pulse 107 bpm and diaphoretic. Fetal heart rate (FHR) was 150 bpm. She denied chest pain or shortness of breath. Investigations revealed a sinus tachycardia on EKG. O₂ saturation was 100% on room air. Troponin was elevated at 0.15. A CT scan of the head was normal. Internal medicine was consulted. It was felt that the elevated troponin was not cardiac related. A repeat test was elevated at

1.15. Neurology was consulted. It was felt that she had experienced convulsive syncopal episodes. She was admitted to hospital and placed on telemetry. An obstetrical ultrasound on April 7 showed a 22 week 4 day viable pregnancy.

On the morning of April 7, while in the shower, the woman felt unwell and pulled the alarm bell. She was found to be agitated and not responding to verbal commands. She was transferred back to bed and was noted to be bradycardic. A "code blue" was called. Full resuscitation was commenced and after 40 minutes, a pulse was re-established. During the code, pH fell to 6.8. Post arrest intubation and sedation were maintained and she was transferred to the Critical Care Unit (CCU) at a tertiary hospital for post-arrest care.

In the CCU at the tertiary hospital, she was hemodynamically stable, but with a persistent tachycardia. Bedside echocardiogram showed normal LV function with severe RV dilation and hypokinesia consistent with pulmonary embolus. Lower extremity Dopplers were negative. She was started on anticoagulation.

An obstetrical ultrasound confirmed fetal demise.

On April 8, 2008, a CT angiogram of the chest showed multiple subsegmental emboli and an obstructing embolus in the lingula.

On April 9, 2008, she was weaned from sedation in order to better assess neurologic status. That evening, she had a possible seizure episode and she was started on IV dilantin. Nasogastric tube feedings were started.

Gradual weaning off sedation was again started on April 15 and was completed by 0600 hours on April 17. An MRI on April 18 showed diffuse white matter changes. The plan was to observe for any clinical signs of neurologic recovery.

On April 23, 2008, labour was induced with the administration of misoprostol 400mcg intravaginally. The heparin was stopped and she went on to deliver a stillborn infant without complication.

On April 25, 2008, a tracheostomy was performed.

Clinically, there was no evidence of neurologic recovery. A repeat MRI on April 28 showed extensive anoxic changes with laminar necrosis. The prognosis for good neurologic recovery was extremely guarded. She was transferred to the Medical/Surgical ward at the local hospital on May 1, 2008 for palliative care.

On May 18, 2008, a percutaneous endoscopic gastronomy (PEG) was inserted and nasogastric (NG) tube was removed.

On June 2, 2008, she was transferred to the Complex Continuing Care site.

On July 23, 2008, O₂ saturation dropped and she was suctioned for food content. She was transferred to the Intensive Care Unit (ICU) for management of aspiration. She deteriorated while in the ICU with worsening oxygen desaturation. The family agreed to a "Do Not Resuscitate" status and death was pronounced at 1242 hours on July 24, 2008.

Post mortem

A post mortem examination was not performed.

Discussion

The deceased died from aspiration as a complication of anoxic brain injury resulting from pulmonary embolus at 22 weeks gestation. She presented with syncopal episodes on April 5 which, in retrospect, were likely due to microemboli. Her care providers appropriately considered cardiac and neurologic etiologies. This was an atypical presentation for pulmonary embolus and this diagnosis was not considered. Microemboli may not have been detected on imaging studies even if the diagnosis had been considered. If imaging studies had been done and confirmed a diagnosis of pulmonary embolus on April 6, instituting anticoagulation therapy may not have prevented the massive embolus of April 7.

Recommendations

No recommendations.

Case: 2011-M-3
OCC file: 2010-9901

History:

The deceased was a 39-year-old G₃P₁ with an EDD of September 1, 2010. Initial laboratory investigations were normal. An Integrated Prenatal Screening (IPS) was positive for Down syndrome. The records provided do not indicate if she was referred for genetic counseling. A second trimester ultrasound on April 1 showed an 18 week 3 day fetus with no fetal anomalies and no placenta previa. Several uterine fibroids were identified, the largest measuring 3.8 cm. A glucose challenge test (GCT) at 28 weeks was normal.

Her past obstetrical history included a term pregnancy delivered by forceps at 40 weeks for a 6 lb 11 oz female in 2007. She had a dilation and curettage (D & C) for a spontaneous abortion in 2009. Her past medical history and family history were non-contributory. She was allergic to Latex.

She was admitted to Hospital A with spontaneous rupture of membranes (SRM) on August 3, 2010 at 0130 hours at 35 weeks 6 days gestation. Hemoglobin at the time of admission was 135 g/L. Augmentation was started at 0930 hours. On reaching full dilation and the presenting part coming down, she was found to have a breech presentation. In her first pregnancy, she was found to have a narrow pubic arch and forceps were required. For the present pregnancy, the decision was made to proceed with delivery by Caesarean section under general anaesthesia. At 1400 hours, she was delivered of a 5lb 9oz female infant with Apgars of 8 and 10 at one and five minutes. The placenta was delivered without difficulty and there were no intra-operative complications.

She was transferred to the Recovery Room where she was noted to have increased vaginal blood loss. She initially responded to massage and was administered Hemabate®. Vital signs showed a tachycardia of 120 bpm at 1427 hours and 148 at 1548 hours. At 1630 hours, her pulse was 136 and blood pressure was 75/48. At 1720 hours, her blood pressure was 92/72, pulse 141 and hemoglobin was

90g/L. She was attended by the obstetrician at 1730 hours. A clot was expelled. At 2000 hours, the obstetrician was notified of steady vaginal bleeding and a drop in blood pressure. The decision was made to take her back to the operating room. The possible need for laparotomy and hysterectomy were discussed. Packed red cell transfusion was started.

Under general anaesthesia, the cervix was inspected and what appeared to be a laceration on the left side was sutured. Oozing was noted from other areas of the cervix and sutures were placed to achieve hemostasis. During exploration of the cervix, the uterus became atonic and Hemabate® was given in addition to the oxytocin. A D & C was performed for several clots, but no significant tissue was obtained. The bleeding subsided and after a further 20 minutes of observation and no active bleeding, the procedure was completed and she was returned to the Recovery Room. During the procedure, blood pressure ranged from 90-100/50-60. She received a total of four units of packed cells, two litres of plasma, one litre of hydroxyethyl starch and two units of fresh frozen plasma (FFP).

In the Recovery Room, the uterus was boggy and the bleeding resumed. She was given Hemabate® intramyometrially in addition to intravenous oxytocin. Blood pressure was 80-100/50-60, hemoglobin 60 g/L. It was questioned as to whether the low hemoglobin was dilutional or due to an intra-abdominal bleed. The decision was made to return to the operating room for probable hysterectomy.

She was returned to the operating room at 0004 hours on August 4, 2010. Assistance from the Intensive Care Unit (ICU) and Emergency Room were called. The anaesthetist noted the heart rate was 115 bpm and thready. Blood pressure and oxygen saturation could not be measured. The patient was rousable to voice. At laparotomy under general anaesthesia, there was a small amount of blood in the peritoneal cavity. The uterus was soft and boggy. Note was made of several fibroids, the largest estimated to be approximately 6 cm. Sponge sticks were placed down each side of the uterus to occlude the blood supply. A hysterectomy was performed. On completion of the procedure, several areas of

bleeding were noted along the pelvic side walls. These were oversewn. Ligaclips were also used on the left pelvic side wall and the angle of the vagina. Ultimately, hemostasis appeared to be satisfactory. Gelfoam® was placed retroperitoneally on the left at the level of the angle of the vagina. A Jackson-Pratt drain was placed and brought out retroperitoneally on the right side.

At 0440 hours, during the procedure, the patient required dopamine and norepinephrine for blood pressure support. Following the initiation of norepinephrine, the patient was noted to be in ventricular tachycardia. A "code blue" was called and chest compressions were commenced. Epinephrine and atropine were given. After a shock of 150J and then a second shock at 200J, sinus rhythm was restored. Blood pressure could not be obtained and phenylephrine was started at 0058 hours. The heart rate slowed to 31. A second "code blue" was called and she was again resuscitated and sinus rhythm was restored. The time interval from the onset of the procedure when a blood pressure could not be obtained until an effective circulation was restored after the second code was over one hour.

A right internal jugular line was placed at 0053 hours. An attempt to place an arterial line was unsuccessful. The patient received a total of nine units of packed cells, 1.5L of hydroxyethyl starch, four units of FFP and one unit of platelets. At 0145 hours the hemoglobin was 50 g/L and platelet count 97,000. The INR was 2.2, ALT was 723 and AST was 1176. The procedure was completed at 0255 hours. Chest x-ray showed a whiteout on the left and the endotracheal tube (ETT) was withdrawn 2 cm. The internist on call was consulted and the patient was transferred to the ICU.

In the ICU, vital signs were stable. She was weaned off of pressor support, but did not regain consciousness. Pupils were fixed and dilated. A CT scan of the head at 1103 hours showed diffuse intracerebral edema. On August 5, 2010, an apnea test revealed no spontaneous respirations. After the family was consulted and informed, the woman was extubated on the morning of August 6 and died at 1057 hours.

Post mortem

Autopsy findings showed the cause of death to be extensive hypoxic encephalopathy secondary to post partum hemorrhage requiring massive transfusion.

Discussion

The deceased died from hemorrhagic shock resulting from postpartum hemorrhage. Although there was concern that bleeding was as a result of a cervical laceration, the principal cause was uterine atony. Uterine fibroids may have been a predisposing factor. The bleeding is mostly described as a steady flow, rather than large gushes. It is possible that this type of steady flow lead to an under-estimate of the total amount of blood lost and thus the questioning as to the low hemoglobin immediately after the D & C being dilutional or due to intra-abdominal bleeding.

There was a period of time when the woman was tachycardic and hypotensive before interventions were initiated. Often, young healthy patients are

capable of a significant compensatory response to severe degrees of hypovolemia until the point where the compensatory mechanisms become overwhelmed and the blood pressure suddenly crashes. Aggressive volume and blood product replacement, preferably with the aid of a central line, is needed if this compensatory phase can be recognized in order to avoid such an outcome.

Recommendations

1. Obstetrical care providers are reminded that blood loss from postpartum hemorrhage is difficult to estimate and therefore clinical markers should be used as outlined in the SOGC Clinical Practice Guideline "*Active Management of the Third Stage of Labour: Prevention and Treatment of Postpartum Hemorrhage*" No. 235 October 2009.
2. Hospital A should review its Post Anaesthetic Care Unit (PACU) policies regarding post delivery care.

Case Summaries: Neonatal Deaths

Case: 2011-N-1
OCC File: 2010-5852

History

The mother of the deceased infant was a 29 –year-old gravida 1 with an EDD of July 14, 2010. There was no indication of previous health concerns. The prenatal 2 record was available up until 20 weeks gestation and at that time, the blood pressure was normal, as was the clinical examination. All appropriate antenatal testing had been done and was normal. IPS showed low risk of Down Syndrome.

She presented to Hospital A on the afternoon of May 12, 2010 at 30 weeks and 4 days gestation with a history of intermittent abdominal pain since the previous evening and also passing small “streaks of blood.” She was examined by a physician shortly after admission when the cervix was closed, though 60 to 70% effaced. The first dose of betamethasone was given. For the next couple of hours, she continued to have cramps and at 1900 hours, the attending physician contacted Criticall to arrange transfer to a perinatal centre.

At that time, all Level 3 centres in the province were closed and arrangements were subsequently made for transfer to an “advanced Level 2” centre. At 2145 hours, the patient was transferred to Hospital B. During the five hour stay in Hospital A, vital signs were normal, with a blood pressure of 130/69. Medications given prior to transfer included the first dose of betamethasone and first dose of intravenous ampicillin for Streptococcus prophylaxis.

She was admitted to Hospital B at 2235 hours. At that time, vital signs were stable. She gave a history of leaking per vagina during the time since leaving Hospital A. At that time, she was felt to be in threatened preterm labour with preterm premature rupture of membranes (PPROM). She was continued

on IV ampicillin and continuous fetal monitoring with a plan for the second dose of betamethasone.

A biophysical profile done on May 13 showed decreased amniotic fluid volume (AFV) with amniotic fluid index (AFI) of 8, normal doppler and an estimated fetal weight (EFW) of 1358 g. No abnormality was reported of the placenta.

From the evening of May 13 and over the next couple of days, she had intermittent chest pain. Her elevated troponins were investigated and consulted with cardiology. Her blood pressure was only mildly elevated with the highest recording of 140/80. Despite increased troponins, there was no other evidence of cardiac disease and it was felt the levels may possibly have been associated with preeclampsia, which subsequently evolved. During the time in hospital, prolonged continuous fetal monitoring was normal.

During the day on May 15, her blood pressure gradually increased and was sustained at 150 to 160 over 90 to 100 with normal PIH blood work, with new onset of 2 + urine protein. At this point, she was felt to have preeclampsia with the background of PPRM and threatened preterm labour.

In the early morning of May 16, induction of labour with oxytocin, along with magnesium sulphate and penicillin G Streptococcus prophylaxis, were initiated with continuous monitoring. At 0627 hours, the nurse contacted the resident to see the patient because of the first onset of two decelerations. The patient was fully dilated at spines +2 and feeling pressure. The resident went to call the staff and upon return, the fetal heart could not be detected by external Doppler. A scalp clip was applied confirming the fetal heart to be in the 90’s.

At 0630 hours, the attending physician, pediatric and neonatal staffpersons were present. With active pushing at 0634 hours, and with an episiotomy, the

infant was delivered at 0638 hours. The newborn was a male infant weighing 1476 g. There was considerable bleeding and clots at the time of delivery of the placenta, compatible with an acute abruption.

The newborn was “flat, pale and cyanotic.” As there was no sign of respiratory effort, positive ventilation was initiated; the heart rate was less than 60 bpm. The baby was intubated at two minutes of age, but did not respond well to positive pressure ventilation (PPV) and 100 % oxygen. At four minutes of age, because of absence of improvement, the endotracheal tube (ET) tube was pulled and a piece of mucous was found to be stuck at the tip. Re-intubation was successful.

Initial epinephrine was given by ET tube and subsequently by umbilical venous catheter (UVC) line, along with saline boluses. There was no response to the resuscitation during these efforts and the baby was pronounced dead at 0725 hours.

Cord blood gases revealed an arterial PH of 7.16 and a base deficit of 6.3 and a venous PH of 7.31 with a base deficit of 3.3.

Post mortem

An autopsy was not conducted as the circumstances of the death were well documented.

The placenta was examined and found to be 332 g. There was an area of placental infarction, though the size was not measured. There was velamentous insertion of the umbilical cord and mild funisitis. Membranes revealed early chorioamnionitis.

Discussion

This was a case of a primiparous patient who, at 30 weeks and 4 days gestation, developed threatened preterm labour and subsequently PPROM. She initially went to hospital and was transferred to an “advanced Level 2” hospital. She was admitted for observation and there were prolonged times of continuous external monitoring, which were normal.

During the hospital stay, she developed mild preeclampsia. Because of the recurrent problems of maternal chest pain, new onset of preeclampsia, PPROM and threatened preterm labour, induction of labour was recommended. Until the time of full dilatation, continuous monitoring was normal, then there was the first evidence of fetal heart deceleration. Shortly after these initial decelerations, there was persistent bradycardia. Spontaneous vaginal delivery was facilitated with pushing and episiotomy. Appropriate staff was in attendance, including the attending obstetrician and neonatal staff.

There was evidence of an acute abruption at the time of delivery. The neonate underwent resuscitation immediately, however did not respond to resuscitation efforts and was pronounced dead at approximately one hour of age.

The cord gases were within the normal range. During the resuscitation, there were times where thick mucous had to be removed from the ET tube with re-intubation. Apart from this, it is unclear why this newborn could not be resuscitated.

This case was discussed with the Chief of Obstetrics at the hospital involved and the case was reviewed at a meeting of the hospital’s perinatal death review committee.

Recommendations

No recommendations.

Case: 2011-N-2
OCC File: 2010-6303

History

The mother was a 22-year-old primigravida who was cared for in her pregnancy by a registered midwife from 9 weeks. She had a medical history that included severe burns as a child with extensive skin grafting. She quit smoking when she found out she was pregnant. She was 5’ 5” and 285 lbs at 9 weeks. She had a BMI of 52 at the time of labour. Ultrasounds were done at 12.5, 19.5, 21.5 and 30.5 weeks. The only finding on the ultrasounds was a

two vessel umbilical cord. No fetal heart abnormalities were seen and growth was appropriate to 30 weeks. IPS was negative and an oral glucose challenge test (OGCT) at 27 weeks was normal. Routine prenatal labs were all normal. Group B Streptococcus (GBS) swab was positive at 35 weeks.

She was admitted to the labour room at the hospital at 1030 hours on March 20, 2010 at 39 weeks and 4 days gestation. She had spontaneous rupture of membranes (SROM) of clear fluid at 0830 hours. Her midwife provided care and consulted with the obstetrician verbally for GBS prophylaxis orders. She received 2g of ampicillin at 1140 hours, 1g at 1540 hours and 1g at 1940 hours per the medication administrations record.

There were no midwifery or nursing notes about her admission assessment and no maternal vital signs were recorded throughout the labour except on the anaesthesia record and the post anaesthetic care unit (PACU) record. The only narrative notes were those typed onto the electronic fetal monitoring (EFM) strip.

It appeared that the midwife auscultated the fetal heart using the EFM machine. There were small fetal heart recordings of 150-160 bpm for one minute at 1013 hours, 160-170 bpm for ten seconds at 1125 hours, 130-140 bpm for ten seconds at 1219 hours, 150 bpm for thirty seconds at 1310 hours and 140 bpm for twenty seconds at 1400 hours. At 1429 hours, there was a note that the patient was in the shower and anaesthesia was consulted regarding an epidural. At 1436 hours, continuous EFM was recorded for two minutes and showed a baseline heart rate of 150-170 bpm, with average variability and accelerations. The tocodynamometer (toco) did not record well.

The anaesthesia records indicated that an epidural was started at 1445 hours. EFM was restarted at 1510 hours and the baseline was recorded as 150 bpm with minimal variability. The tracing was flat for thirty minutes, but then there were accelerations and average variability. The toco did not appear to be working. At 1617 hours, there was a prolonged deceleration lasting ten minutes. The tracing

afterwards was again flat with minimal variability and a baseline of 140 bpm. Notation on the EFM states "fhr 60-90 bpm x 4 min. pt repositioned on her right side. ve 4-5 cm dilated. fhr returned to baseline. pt comfortable with epidural." After thirty minutes, the variability improved to average and there were accelerations.

The fetal heart was normal until 1715 hours when several gaps in the recording occurred, suggesting possible decelerations. At 1735 hours, EFM notes indicated that the obstetrician was in the room and that he examined the mother and placed a fetal scalp clip at 1740 hours. Between 1740 and 1800 hours, the baseline was 150 bpm with minimal variability and accelerations present.

The obstetrician did not write or dictate a note at this time, but noted in the dictated Caesarean section note several hours later that he was asked to see the mother "for possible augmentation of labour" as she had been "4-5 cm dilated for at least 5-6 hours" prior to his assessment. He indicated that he ordered oxytocin augmentation at that time. The EFM notes stated that care was transferred to the obstetrician at 1804 hours. There was no indication from midwifery or nursing notes, or from the medication administration record, when oxytocin was started or what dosage was used.

Another prolonged deceleration to around 100 bpm occurred from 1802 to 1808 hours just following the placement of an in-and-out bladder catheter. The baseline returned to 150 bpm with minimal to average variability and accelerations present. At 1830 hours, the anaesthesiologist was contacted as the mother was feeling her contractions.

At around 1900 hours, short variable-type decelerations began. The toco was not picking up contractions, but there were two EFM notes saying, "pt is having a contraction." Between 1900 and 2000 hours, the decelerations became more repetitive (every 2-3 minutes), wider and more complicated. At 1952 hours, a note on the EFM read, "pt states she feels like she is constantly cramping, oxytocin drip turned down." This was the only indication on the hospital chart that oxytocin was started.

At 2000 hours, the baseline was 160 bpm with minimal variability. At 2004 hours, there were four complicated variable decelerations in four minutes. By 2030 hours, the fetal heart rate was failing to return to baseline between decelerations. The delivery record and a notation on the EFM showed the mother to have been fully dilated at this time.

The EFM tracing continued to deteriorate and at 2053 hours, the mother was transferred to the delivery room. When the monitor was restarted at 2104 hours, there was absent variability. A bradycardia occurred to 90 bpm at 2108 hours and less than 60 bpm by 2115 hours. The anaesthesiologist was called at 2110 hours and arrived and topped up the epidural at 2116 hours. At 2118 hours, forceps were applied, but abandoned as the epidural was insufficient. General anaesthesia was given at 2120 hours. A midline skin incision was made and low transverse Caesarean was completed. The baby was born at 2132 hours.

The male infant weighed 3.53 kg and was flat at birth with no heart rate or spontaneous respirations. Apgar scores were 0 initially and 2 at one, five and ten minutes. Venous cord gases were 7.19/46/32/17.6/19 with base excess of -10.4. Full resuscitation was required including two doses of epinephrine via the endotracheal (ET) tube and a bolus of saline through an umbilical line. A heart rate was obtained at ten minutes and spontaneous respirations after thirty minutes of age. Seizure activity was noted at one hour of life.

The baby was transferred to the intensive care unit (ICU) and given intravenous antibiotics, phenobarbital and midazolam. A pneumothorax was identified and treated. There was evidence of end organ damage with elevated cardiac and liver enzymes, creatinine and decreased urinary output. On the second day of life, the baby was sent for an EEG.

Later that day, the infant had a sudden change in perfusion with cyanosis and mottling from the neck down. The pediatrician on call contacted the children's hospital transport team for advice and it was felt that the degree of hypoxic ischemic encephalopathy was very significant from the time

of birth and neurologic outcome in all likelihood would be exceedingly poor. It was felt by all that the baby's prognosis was so poor that withdrawal of care, rather than transfer, should be discussed with the parents. After discussion with the family, care was withdrawn at 1530 hours. The baby died at 0424 hours the next day, March 23, 2010.

Post mortem

No post mortem was conducted. Placental pathology showed a two vessel cord and peripheral mild old ischemic changes.

Discussion

This baby died of severe intrapartum asphyxia. The pregnancy was uncomplicated except for a finding of a two vessel umbilical cord and the mother's obesity. Due to her size, there appear to have been some difficulties in monitoring the fetal heart and contraction activity. However, there was evidence of atypical EFM from two hours prior to the delivery, and then abnormal fetal EFM from one hour prior to the delivery. These changes did not appear to have been identified or acted upon until forty minutes prior to birth when forceps delivery was organized, but abandoned due to poor pain control.

It is almost impossible to understand what, or when, a diagnosis was made, and what actions were made to improve the fetal status as there are insufficient notes by the midwife, obstetrician and nursing staff during the labour. The only notes are those typed onto the EFM tracing. It is possible that aggressive intrauterine resuscitation and earlier delivery could have improved the outcome. It is also recognized that due to the mother's morbid obesity, it is difficult to achieve a Caesarean section in the usual time. In this situation, from the time of abandoning the forceps to inducing general anaesthesia, was two minutes. The baby was delivered twelve minutes after anaesthesia was administered.

Recommendations

1. All obstetrical care providers at Hospital "A" should review the Fetal Health Surveillance in

Labour Guidelines as per SOGC Guidelines September 2007.

2. Hospital "A" should review its policies regarding its intrapartum documentation.
3. The Regional Supervising Coroner should consider a Regional Coroner's Review of this case specifically addressing documentation by all obstetrical care providers involved.

Case: 2011-N-3
OCC File: 2009-11355

Clinical History

The mother of the deceased was a 45-year-old G₅P₃ who presented to the nursing station in a remote First Nation community on May 15, 2009 complaining of dysuria and suprapubic pain over the previous four days. She had no prior prenatal care. When the pregnancy was confirmed, the registered nurse on duty initiated the first prenatal visit. Her weight was 66 kg, blood pressure 150/90 and there was trace proteinuria. Symphysis-fundus height (SFH) was noted to be 22 – 23 cm. Routine prenatal laboratory investigations were ordered and the results were normal.

An EDD was set for October 5, 2009 based on a dating ultrasound arranged on her first visit on May 15. The ultrasound was performed on June 17. Ultrasound showed a singleton fetus with measurements consistent with 24 weeks 2 days, normal morphology and no placenta previa.

The mother's past obstetrical history revealed three previous vaginal deliveries. Details of these pregnancies were uncertain, but it appeared that at least one was premature at 36 weeks. There was a questionable history of hypertension.

The mother smoked 6+ cigarettes per day and abused alcohol in the form of "homebrew" every weekend. She was in an abusive relationship and was occasionally assaulted by her partner. She was on a prenatal vitamin - ferrous gluconate 300mg (twice daily) - and was given a prescription for

metronidazole 500mg twice daily for bacterial vaginosis.

The documentation provided for review was confusing. A physician note dated May 31 indicated numerous risk factors. An entry on the Antenatal Record 2 for June 17 indicated an ultrasound was being done that day and a 50 g Glucose Challenge Test (GCT) was done and reported as normal.

The mother could not be contacted for prenatal visits on July 16 and August 17. Documentation by a physician on the Zone Hospital Physical Field Visit Form was done on August 18 and noted that the patient was non-compliant. Ultrasound results were documented and mention was made that some results were still outstanding. Physical findings (i.e. BP, SFH and FH) were not recorded, leading to the possibility that the patient was not actually seen, but her records reviewed by the visiting physician.

Course in Labour and Delivery

The mother presented with contractions to the Nursing Station in the remote First Nation Community at 0030 hours on September 5, at 35 weeks 5 days gestation. Contractions were every seven minutes, lasting 1.5 minutes. BP was 175/115 with 2+ proteinuria. She was asymptomatic. Fetal heart rate was 148. The physician was notified and orders were given for hydralazine 5mg IV, magnesium sulphate 4g IV, ampicillin 2g IV and Indomethacin 100 mg per rectum. BP at 0200 hours was 160/109. Hydralazine 20mg (by mouth) was ordered.

At 0245 hours, contractions were five minutes apart, lasting 35 seconds. The air ambulance service was called and made aware of the change in contractions; they were estimated to land in twenty minutes. The cervix was 3-4 cm dilated. The physician was notified and subsequent hydralazine orders received 5-10mg IV every twenty minutes, when necessary.

On arrival of the air ambulance team, vaginal examination disclosed a breech presentation. Membranes ruptured for meconium. Fetal heart rate was 132 bpm. Extra staff was called in to assist and

preparations were made for delivery. The cord prolapsed and with delivery to the head, the cord was noted to be around the body and neck twice. After the cord was unwrapped, the head delivered spontaneously at 0615 hours. Cord gases were not obtained. Resuscitation was initiated by the air ambulance medic. Apgar scores were 2 and 5 at one and five minutes. The airway was suctioned with a 2.5 mm endo-tracheal tube (ETT) for no meconium return. The baby began spontaneous respirations with stimulation and the ETT was withdrawn.

Respirations became irregular and the baby was re-intubated. Apgar score at ten minutes was 1. After auscultation of the chest, the ETT was pulled back 1 cm. Attempts at umbilical vessel cannulation were unsuccessful. The baby's abdomen was noted to become distended and rigid. Auscultation of the chest revealed absence of breath and heart sounds. The right chest was needled for no air return. At forty minutes into the code, the physician was notified and the paramedic was eventually able to get a field pronouncement at 0710 hours. The baby weighed 1.5 kg.

Post mortem

Pathology of the placenta showed a central area of chronic infarction measuring 4 cm and microscopic findings in keeping with hypertensive injury. Placental weight was low for gestational age at the mean for 31-32 weeks gestation.

No autopsy was performed.

Discussion

This infant died shortly after delivery at approximately 36 weeks by spontaneous frank breech complicated by cord accident and meconium. The baby was small for dates and there were placental changes consistent with chronic hypertension. There were a number of significant risk factors for intrapartum asphyxia. The initial plan was to transfer the mother from the remote First Nation community where she lived, to the base hospital, but the labour progressed too quickly. Fetal heart monitoring was by intermittent auscultation on a sporadic basis. Normal heart rates were recorded.

The baby was flat at birth with poor Apgar scores, but cord gases were not done and therefore the acid-base status at birth is not known. Resuscitation efforts by the paramedics and nurses in the remote community were unsuccessful.

Although this pregnancy had been identified as high risk on May 31 due to the patient's age and lifestyle, the plan to write a letter of referral once all test results were back never materialized. The challenges of providing care for a non-compliant patient in a remote community are recognized.

Recommendations

1. All nurses working in remote nursing stations and remote health centres where unplanned births may occur, should be certified in the Neonatal Resuscitation Program (NRP) on an annual basis.
2. Nurses working in remote nursing stations and health centres where unplanned births may occur should be required to spend a period of time on the labour and delivery unit of the hospital they transfer to and consult with.
3. Air transport crews, Emergency Medical Services (EMS) attendants and nurses working in remote locations should complete an emergency obstetrical skills course annually in collaboration with their transport/receiving consulting centre.
4. The provincial and federal governments should consider providing remote nursing stations with the capacity for electronic fetal monitoring and distant telemetry.
5. Health Canada should conduct research to explore barriers to obstetrical care in remote First Nations communities with the goal of achieving a consistent standard of obstetrical care for all women in Ontario.

Case: 2011-N-4
OCC File: 2009-10271

History

The mother was a 24-year-old G₁P₀ with an EDD of July 27, 2009. Routine prenatal laboratory investigations were normal and an IPS was negative. An ultrasound on February 28 at 18 weeks 4 days, showed multiple choroid plexus cysts. Amniocentesis showed normal female karyotype. A glucose challenge test at 28 weeks was normal. Blood pressure and growth by symphysis fundal height were normal throughout the third trimester. The last visit recorded on the Antenatal Record 2 was on June 29, at 36 weeks gestation.

Her past medical history was unremarkable and her family history was positive for diabetes. She was a non-smoker.

She presented to the triage unit at the local hospital on the morning of July 31 because of a two day history of decreased fetal movement. A non-stress test (NST) was performed and was interpreted as "borderline." She was asked to return later that day for a repeat NST. She presented at 1900 hours and the repeat NST showed "improved variability, but no accelerations." An ultrasound showed adequate amniotic fluid, breathing movements and fetal tone, but no gross fetal body movements. The cervix was 1cm dilated, soft and thick and the presenting part was high. A decision was made to induce labour.

She reached the maximum oxytocin infusion rate during the night, but failed to go into labour. She received intravenous penicillin G prophylaxis for group B Streptococcus (GBS).

After sign-out the following morning, the new obstetrician on call assessed the patient at 0845 hours. There was no change in the cervix. The fetal heart rate was in the 140's with no decelerations. The oxytocin induction was continued and she was reassessed at 1125 hours. Labour had not been established and there was no change in the cervix. Operative delivery was discussed, but the final decision was not made until the patient's husband returned at 1240 hours. The couple agreed to

proceed to Caesarean section, but were advised by the anaesthetist to wait six hours because the patient had eaten lunch.

On August 1, 2009, at 1829 hours, the patient, under spinal anaesthesia, was delivered by Caesarean section of a 3650 g female infant. Thick meconium was encountered on entry into the amniotic sac and the paediatrician on call was called to attend. The head was delivered with assistance of the vacuum extractor. The baby was suctioned prior to delivery of the trunk. Apgars were 2, 4 and 5 at one, five and ten minutes. Cord blood gases were arterial pH 7.06, BE -11.2 and venous pH 7.21 and BE -9.2. The heart rate was initially 60 bpm. Positive pressure ventilation (PPV) was initiated by the respiratory therapist and the heart rate increased to 120 bpm. There was no respiratory effort. The anaesthetist attempted to intubate the baby with a 3.5 endotracheal tube (ETT), but was unsuccessful. The tube was removed and after the stomach was suctioned, the baby was re-intubated with a 3.0 ETT. The O₂ saturation climbed from 54 to 94 and heart rate was 163. The baby was pink, but remained flaccid. The paediatrician arrived at 1856 hours and took over the resuscitation and an umbilical artery line was placed. The initial laboratory investigations showed a hemoglobin of 139 g/L, WBC 51.4 and blood sugar 0.6. The baby was given intravenous dextrose and antibiotics were started.

The children's hospital was contacted at 2006 hours and the transfer team arrived at 2125 hours. The baby was transferred and arrived at the children's hospital at 0045 hours on August 2. Seizure activity was noted at six hours. Subsequent imaging studies and EEG confirmed diffuse encephalopathy. After discussion with the parents, the baby was extubated at 2000 hours on August 11 and was pronounced at 0744 hours on August 12.

Post mortem

Autopsy revealed growth parameters appropriate for age. There were no congenital abnormalities. There was diffuse hypoxic-ischemic injury.

The placenta was excessively large and weighed 620 g (after fixation in formalin). This, together with the

finding of villous dysmaturity, raised the consideration of maternal diabetes mellitus. The umbilical cord was over-coiled (coiling index 0.7 vs. 0.2 normal).

Although this can be associated with vascular occlusion, there was no morphologic evidence of this. Meconium exposure effects were noted. The cause of death was global cerebral hypoxic-ischemic injury.

Discussion

This infant died as a result of perinatal asphyxia following delivery by Caesarean section approximately 34 hours after the mother presented to the obstetrical triage because of decreased fetal movement. The NST done at the time of initial presentation at 0810 on July 31 was interpreted as "borderline." This term is not recognized in the 2007 SOGC publication "Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline."

On review, the tracing ran from 0822 hours to 0945 hours. During that time, there were no fetal heart rate accelerations. Based on the consensus guideline, the tracing is abnormal and requires urgent action such as a biophysical profile or consideration for delivery. A note in the medical record indicated that the unit was busy and the plan was to repeat the test in the evening. A second NST done between 1904 hours and 1954 was atypical (i.e. < 80 minutes without accelerations) and was interpreted as "improved." What appears to have been a bedside ultrasound was done at 2000 hours and was reported as showing adequate amniotic fluid, breathing movements and tone, but no movement. The biophysical profile was noted to be 6/8. The score of 6/8 reassured the caregivers and met the requirement of the guideline for urgent assessment of an abnormal NST.

Oxytocin induction was started despite the cervix being unfavourable. The induction ran from 2130 hours until 1000 hours on August 1 without labour being established. During that time, the fetal heart rate tracing was for the most part unchanged from the NST's. There does not appear to have been any

further fetal surveillance after the induction was stopped.

Other than the ultrasound assessment at 2000 hours on July 31, from the time the patient first presented at 0800 hours on July 31 because of decreased fetal movement, until delivery at 1839 hours on August 1, fetal surveillance testing was not normal. The accuracy of the ultrasound surveillance test cannot be determined by this review. The clinical setting of decreased fetal movement together with the fetal heart rate tracings, indicate that earlier intervention was indicated. It cannot be determined from this review whether earlier intervention would have changed the outcome.

Recommendations

1. Obstetrical care providers are reminded of the SOGC guidelines on antepartum and intrapartum fetal surveillance.
2. Obstetrical care providers are reminded of the indications for, and the SOGC guidelines on, Induction of Labour at Term. (Aug. 2001)

Case: 2011-N-5
OCC File: 2009-15214

History:

The mother of the deceased was a 25-year-old G₁P₀ who presented to a midwifery practice on October 20, 2009 at 26 weeks gestation. All investigative testing done to date were normal. Her blood type was A negative and Rhogam had been administered by her family physician prior to coming into midwifery care.

On November 5, 2009 at 28 weeks gestation, the woman advised the midwife that she had independently gone for a 3D ultrasound. She was told by the private company that performed the ultrasound that there appeared to be something wrong with the baby's hand and foot, although the nature of the alleged abnormality was not clear. A previous ultrasound done at 17 weeks gestation had

revealed normal morphology. No further follow-up was done.

On the day of the 3D ultrasound, the mother's blood pressure was 124/92. She was sent to triage at the hospital for a consult and work-up for eclampsia. She was sent home as everything appeared normal.

On November 15, 2009 at 30 weeks gestation, the woman called her midwife reporting cramping over the last 24 hours. An assessment was done in triage at the local hospital. The cervix was closed, however within three hours she progressed to 3-4 cm dilated and fully effaced. A transfer of care was initiated at 2235 hours. A fetal fibronectin test was positive and dexamethasone 6mg IM along with IV antibiotics were given. The FHR was 130-140 bpm and reassuring. The baby was in a breech position.

An urgent transfer to a level 3 hospital was arranged. Following spontaneous rupture of membranes (SROM) and abnormal FHR, an emergency Caesarean section was done at 0510 hours on November 16, 2009. A 1407 g baby boy was delivered with Apgars of 1, 0 and 0 at one, five and ten minutes.

Efforts to intubate the infant were initially difficult. The infant was pronounced at 0533 hours in the Neonatal Intensive Care Unit (NICU).

Upon examination, the infant had obvious facial dysmorphic features and limb anomalies. There was dark reddish discoloration on his buttocks, although the delivery was atraumatic.

Post mortem

The cause of death was undetermined. Karotype was normal as were all cultures.

The infant had pronounced hypoplasia of the lungs and absent musculature of the diaphragm. Neuropathology exam was normal.

There were multiple congenital abnormalities including: bilateral club feet, bilateral flexion contracture at wrists, deep set base of nose, folded ear helices, bilateral palmer creases, hypoplastic lungs, small nephroblastic remnant in right kidney,

absent musculature in diaphragm, moderate lymphoplasmacytic infiltrate in portal areas with multifocal mild effacement of the limiting plate and extensive recent hemorrhage in dermis and subcutis in skin sampled from lower back.

All cardiac blood and lung cultures were negative and the placenta and cord were unremarkable.

Discussion

This baby was born with multiple anomalies. The initial 17 week ultrasound showed no abnormalities, while a 3D ultrasound performed by a non-medical facility at 28 weeks, reportedly showed anomalies of the hands and feet. At this stage of the pregnancy, it is not believed that an alternative course of action would have resulted in a different outcome. Appropriate follow-up was done for suspected preterm labour. Unfortunately, as a result of extreme anomalies and prematurity, this baby did not survive.

Recommendation

1. [Obstetrical care providers are reminded that abnormalities noted on ultrasound examinations require further assessment.](#)

Case: 2011-N-6

OCC File: 2009-10610

History

The mother of the deceased was a 30-year-old G₂P₁L₁ in her second pregnancy. She had previously had an uncomplicated preterm vaginal delivery at 33 weeks gestation in 2006. She was overweight at 193.5 pounds at 15 weeks, increasing to 208.5 pounds at 34 weeks. Her symphysis fundus height (SFH) was consistently elevated: 34 cm at 30 weeks and 39 cm at 34 weeks. An ultrasound done at 34 weeks showed an estimated fetal weight of 2507 grams (88th percentile). There was a suggestion on the ultrasound report of potential for macrosomia at birth. There was no record of glucose testing on the chart. All other blood screening was normal and she was GBS positive.

The pregnancy was normal and uncomplicated prior to labour at 39 weeks and 1 day. She had ruptured membranes at eight hours prior to delivery. She had two doses of cefazolin for Streptococcus prophylaxis in labour. The first stage of labour was 16 hours and the second was 43 minutes. Nursing notes and monitor strips in labour were not available for review.

There is no dictated operative note, but the delivery record indicates a shoulder dystocia that lasted a total of twelve minutes. In an attempt to resolve this, they used the McRoberts maneuver and attempted to rotate the shoulder in either direction with supra pubic pressure. The Woods maneuver in both directions was also attempted in order to remove the posterior arm.

The baby was born at 0328 hours on July 16. The birth weight was 4125 g . Apgars were 0, 2, 3 and 4 at ten, twenty, thirty and sixty minutes. The cord was tight nuchally and it was clamped and cut prior to the subsequent delayed delivery. The cord PH was 7.25, though it is not clear whether this was an arterial or venous sample.

Resuscitation was initiated with positive pressure ventilation and chest compressions. Multiple attempts at intubation were unsuccessful and at 19 minutes of age, the baby was successfully intubated.

The first palpable pulse of 70 was noted. The venous gas from the umbilical cord catheter at 37 minutes of age revealed a PH of 6.67, increasing to 6.84 at approximately 90 minutes of age.

The neonatal team from the children's hospital was contacted and arrived at the delivery hospital (located in an adjacent province) at 0610 hours when the infant was approximately 2 hours and 40 minutes of age. On arrival, the baby was in an open care bed. An endotracheal tube was in situ. A UVC was in place and the infant was receiving fluids, gentamycin and an ampicillin infusion. The arterial PH was around 7.46 with a bicarb of 12, showing a significant improvement since the resuscitation efforts prior to arrival of the team. They departed the referral nursery at 0823 hours and arrived at the NICU in the children's hospital at 0905 hours.

From the time of admission to the NICU, there was no spontaneous movement. The infant developed seizures, required continuous ventilation and showed very little breathing above the ventilator. Investigations by EEG showed suppressed activity with seizures and an MRI on July 20 showed evidence of acute profound hypoxic ischemic injury.

Based on the delivery history, physical examination, early onset of seizure activity, plus MRI and EEG results, a multidisciplinary meeting that included NICU staff, Neurology and a social worker, was held. Withdrawal of care was recommended to the parents. The infant died approximately 24 hours later, on July 22, 2009, at six days of age.

Post mortem

The autopsy report confirmed severe anoxic ischemic changes in the brain. There were no other significant findings. The clavicles did not appear to have been fractured during the delivery.

Discussion

This infant was born at term after an uncomplicated pregnancy and spontaneous labour. At 34 weeks gestation, there was evidence that the baby was large for dates. No further clinical notes after that date were available for review.

There were no nursing notes or monitoring strips from the time of labour provided for review. The written summaries do not suggest that there were any concerns until the delivery of the head. The cord gas that was taken was normal and this represented the status at the time of delivery of the head, but not the full delivery.

The baby was born after an extremely difficult delivery where shoulder dystocia was experienced. It is evident by the notes that all reasonable efforts were made to deliver the baby.

Early blood gases on the newborn showed extreme acidosis and together with poor Apgar scores, suggested a poor prognosis from the time of delivery. Despite efforts at resuscitating this infant,

there was early evidence of ischemic brain injury from which recovery was not possible. It appeared that the staff in the first hospital and subsequently at the children's hospital, made every effort possible at the time of delivery and afterwards.

Recommendations

No recommendations.

Case: 2011-N-7

OCC File: 2010-3878

History

The infant was born on March 29, 2010 to a 34-year-old primigravida woman at 37 weeks and five days gestation. The mother's general health was good and she had excellent prenatal care. IPS and other antenatal screening were negative. She had gestational diabetes that was managed with diet. Swab for Group B Streptococcus was negative. During the latter part of the pregnancy, it was found that she was developing mild hypertension and had reduced amniotic fluid. This prompted a planned induction of labour which resulted in the development of regular contractions on the morning of March 29, 2010. She progressed rapidly to full cervical dilatation, but the head failed to descend after 3.5 hours of pushing. A Caesarean section was subsequently performed under spinal anaesthesia.

The baby was born in good condition at 1206 hours. She required no resuscitation and was awarded Apgar scores of 9 at one minute and 9 at five minutes. Cord arterial gases showed a pH of 7.25 and base deficit of 4.9 mmol/L. Birth weight was 2898 g. The infant was transferred to the postpartum ward with her parents. An initial glucometer reading at forty minutes of age was normal at 3.1 mmol/L. Vital signs were normal. Breastfeeding was commenced and she did well through the night, feeding every 1-3 hours. In the morning, she was examined by the attending physician and was deemed to be a healthy newborn.

At 0820 hours, the baby was described as being "fussy at breast." The nurse suggested that the mother try to calm the baby by placing her on her

chest. The baby appeared to have settled when the nurse checked ten minutes later. At 0941 hours, there was an urgent call from a visitor indicating that the baby was in distress. The father brought the infant to the nurse in the hallway outside the room. The infant was described as limp, unresponsive and grey in colour. She was taken to an assessment area and the emergency bell was activated. The infant had no audible heart rate. Positive pressure ventilation and chest compressions were commenced. The Code team arrived from the Special Care Nursery at 0945 hours and continued with the resuscitative efforts. Cardiac leads were connected and showed asystole. Interventions included endotracheal intubation, ongoing positive pressure ventilation, chest compressions and four doses of endotracheal epinephrine. There was no response to any of these measures and the baby was pronounced dead at 1005 hours on March 30, 2010.

Post mortem

A very detailed examination of the body and investigation to determine the cause of death were performed. In spite of this, no clear cause of death was determined. The only positive autopsy findings were:

1. Hyaline membranes present in the alveoli (etiology unclear)
2. Mild anoxic-ischemic encephalopathy changes (likely agonal)
3. Small right pneumothorax (likely due to resuscitative ventilation)

Microbiology and toxicology results were negative. Biochemical testing revealed an elevated level of bile glutarylcarnitine suggestive of a metabolic disorder, but this was ruled out when definitive testing was performed.

Death was attributed to "anoxic-ischemic encephalopathy due to acute lung injury with hyaline membrane of undetermined etiology."

Discussion

This infant died after suffering an unexpected cardiac and respiratory arrest at approximately 21 hours of age. The pregnancy had been uneventful

and there was no indication of fetal compromise. The infant emerged in good condition after delivery by Caesarean section for failure to progress. She had normal vital signs and appeared to be breastfeeding well. The first sign of a problem was when she exhibited some irritability at the breast, but she settled after the mother was advised to hold her to her chest. The parents noticed that something was wrong with the baby an hour later and, when handed to the nurse, she was suffering from a full cardiorespiratory arrest. A Code was called and staff was quickly mobilized. In spite of aggressive resuscitative measures, a heart beat could not be established and the baby was pronounced dead shortly thereafter.

Sudden unexpected collapse in an apparently healthy newborn is a rare, but devastating event. It is estimated to occur in approximately 0.05/1000 live births. In one of the largest series from the UK, no underlying abnormality could be detected in two thirds of cases although there was considerable variability in the approach to investigation. While some of these infants could be successfully revived and had good outcomes, one third had a poor outcome with either death or significant neurological sequelae. Possibly pertinent to this case, mothers were commonly primiparous and unattended by clinical staff before the collapse was recognized. Many of the infants were being held skin-to-skin, having been placed prone on the mother's chest. The clinical/pathological diagnosis in many of these infants was airway obstruction during breastfeeding or prone positioning.

It is essential that a rigorous investigation take place before the cause of a case of unexpected collapse is deemed to be "unknown." In this case, a full anatomic autopsy was performed along with appropriate metabolic, toxicologic and microbiologic investigations. None of these investigations revealed a plausible explanation for the baby's collapse. No infectious agents were isolated from specimens taken from the baby's lungs, blood or liver and no foci of infection were revealed in the microscopic sections. The hyaline membranes in the lung and small pneumothorax were most likely due to aggressive positive pressure ventilation provided during resuscitation. The other positive finding of

mild anoxic-ischemic encephalopathy is likely to be a consequence of the arrest, rather than the cause.

Skin-to-skin care between mothers and infants in the period after delivery has been shown to promote normal mother-baby interactions and effective breastfeeding and is recommended as a normal, early care practice in most hospital guidelines. However, it appears that this practice is associated with the rare occurrence of unexpected collapse in an otherwise healthy newborn. It has been suggested that some cases may be due to unrecognized, accidental asphyxia. It is possible that some infants with an underlying vulnerability, with positioning that provides an additional challenge to their respiratory function, may suffer a respiratory arrest.

In this case, the baby was placed on the mother's chest after a short period of fussiness. She appeared to settle and remained in this position for some time. The nurse in attendance assisted with the original positioning, but there were no additional observations recorded. Appropriate standards of care were provided and, when the baby's collapse was recognized, a prompt and effective response was mounted. Appropriate measures failed to revive the baby suggesting that she may have suffered the arrest some time before a serious problem was apparent to the family. It is not known why this baby died. It is possible that the baby died as a result of airway occlusion associated with her positioning on the chest. It is also possible that she died from some unrecognized problem with her respiratory control or an undetected infectious, cardiac or metabolic condition.

Recommendations

1. The causative association between skin-to-skin care and sudden unexpected collapse of newborns remains unproven.

Obstetrical care providers charged with the management of new mother-baby diads in the immediate post-partum period should, when initiating and supporting skin-to-skin care and breastfeeding, ensure that the infant's position is safe, the nose and mouth are not occluded

and that parents are properly instructed. Intermittent, frequent observation should occur when skin-to-skin care and breastfeeding are being practiced, especially with primiparous mothers.

2. To the Canadian Paediatric Society (Canadian Paediatric Surveillance Program):

Further study into the association between skin-to-skin care and sudden unexpected collapse of newborns and possible preventive strategies should be undertaken.

References

1. Becher J-C, Bhushan SS, Lyon AJ. Unexpected collapse in apparently healthy newborns – a prospective national study of a missing cohort of neonatal deaths and near-death events. Arch Dis Child Fetal Neonatal Ed; June 28, 2011 [Epub ahead of print].
2. Fleming PJ. Unexpected collapse of apparently healthy newborn infants: the benefits and potential risks of skin-to-skin contact. Arch Dis Child Fetal Neonatal Ed; September 12, 2011 [Epub ahead of print].

Case: 2011-N-8
OCC File: 2009-15213

History

The mother was a 20-year-old G₂P₀ with a history of substance misuse and no prenatal care. The estimated date of delivery was unknown.

The mother presented to Hospital A in preterm labour at 2030 hours on November 16, 2009. Her cervix was 6 cm and dilated. An ultrasound done to try to establish gestational age revealed fetal ascites and possibly other congenital anomalies. She was given dexamethasone and started on intravenous ampicillin. Indomethacin and nifedipine were given for tocolysis and arrangements were made for transfer to Hospital B.

The mother arrived at Hospital B at 0116 hours and was fully dilated. Ultrasound was attempted, but

was technically difficult as the patient was pushing. Fetal ascites was confirmed. She went on to spontaneous delivery of the head, but delivery of the rest of the baby was delayed due to abdominal dystocia. This was relieved by ultrasound guided paracentesis and significant pulling on the head. The 2427 g male infant was delivered at 0208 hours. Apgars were 1, 1 and 1 at one, five and ten minutes. Arterial cord blood pH was 7.18. Resuscitation was unsuccessful and the baby was pronounced at 0331 hours.

Post mortem

The prostatic urethra was malformed with multiple tiny blind ending urethral channels rather than a central lumen. This was accompanied by hypoplasia and malformation of the prostate gland. Secondly, there was dilation of the urinary bladder, tortuous ureters and hydronephrosis with extensive fibrosis of the kidneys. There was marked pulmonary hypoplasia secondary to massive ascites. There were 11 pairs of ribs which showed developmental changes which may have also been secondary to the ascites.

Neuropathological examination showed subdural and subarachnoid hemorrhage in the spinal canal and posterior fossa. Skeletal x-rays showed malalignment of the cervical spine at C4-5 with distraction and 7 mm of separation and widening at the occiput-C1 level. These findings were attributed to trauma at delivery.

Chromosome studies showed a normal 46 XY karyotype.

Maternal drug screen was positive for cannabis.

The cause of death was stated as structural malformation of the prostatic urethra with ascites and pulmonary hypoplasia.

Discussion

This infant died shortly after birth due to pulmonary hypoplasia resulting from oligohydramnios and ascites secondary to malformation of the prostatic urethra which caused urinary tract obstruction. With

routine prenatal care, an ultrasound at 18-20 weeks gestation may have detected evidence of urinary tract obstruction. This condition is treatable in utero, thereby preventing the oligohydramnios which leads to the lung hypoplasia and inability to establish ventilation at birth. The mother did not seek prenatal care and the Children's Aid Society (CAS) was notified.

The birth trauma to the cervical spine was a result of the pulling required to affect delivery even after the paracentesis was performed reducing the size of the abdominal cavity and relieving the dystocia. Under the circumstances, the trauma may well have been unavoidable.

Recommendations

No recommendations.

Case: 2011-N-9

OCC File: 2010-12873

History

The mother of the deceased was a 34-year-old G₄P₂ with an EDD of October 27, 2010. She was receiving prenatal care and planned on delivering at a hospital near where she resided.

On the morning of October 12, 2010 at 34 weeks gestation, while visiting a location some distance from her home, she noted decreased fetal movement. At approximately 1300 hours, she experienced the onset of abdominal pain and tightness. The pain was described as fairly constant, crampy and quite severe. At 1530 hours, she presented to Hospital A located near where she was visiting.

Her antenatal course had been complicated by a subchorionic bleed in the first trimester, but there had been no subsequent bleeding. She had mild blood pressure elevation of 150/88, but blood work was normal according to her husband.

Her past obstetrical history included two spontaneous abortions and uncomplicated

pregnancies delivered vaginally in 1997 and 2002. Her past medical history was uneventful.

Her weight at the beginning of the pregnancy was 230 pounds and height 5'4" for a BMI of 40.

Routine prenatal laboratory investigations, IPS and glucose challenge test were normal. She was Group B streptococcus (GBS) negative.

Course in Labour and Delivery

At the time of presentation to the obstetrical unit in Hospital A, the uterus was found to be rigid and not relaxing. There was difficulty auscultating the fetal heart. Her blood pressure was 142/76 and pulse 91. O₂ saturation was 100%. A heart rate of 95-99 was detected on the external fetal heart monitor. The obstetrician on call was notified at 1536 hours and responded at 1538 hours. After being informed of the situation, the obstetrician said she would be on her way in. At 1540 hours, the patient had a generalized seizure lasting four minutes. An IV was started and she was given O₂ by face mask. The fetal heart could not be obtained. The obstetrician on call was informed at 1545 hours of the seizure and the need to attend the hospital immediately. The obstetrician left orders to prepare the patient for Caesarean section while she was on her way in.

She was assessed by the anaesthetist in the operating room at 1554 hours. The decision was made to proceed with a spinal anesthetic as opposed to a general anaesthetic due to a difficult airway. A fetal heart rate of 110 bpm was recorded prior to placing the patient sitting upright for the spinal. The spinal was placed at 1600 hours, but the anaesthetist waited for the obstetrician before injecting the local anaesthetic.

The obstetrician arrived at 1603 hours and a local anaesthetic was injected. The obstetrician could not detect a fetal heart by bedside ultrasound. The surgery started at 1605 hours before the spinal was effective. The obstetrician asked the patient if she could continue for the sake of the baby even though she was in pain. The patient said to continue and the baby was delivered at 1608 hours. Blood clots were present in the amniotic cavity and approximately

80% of the placenta had separated. She had a second seizure during the procedure. She was given MgSO₄ IV and transferred to the intensive care unit for initial postpartum care.

Laboratory investigations of the mother showed platelets 130,000 and INR, PTT and LFT's were normal. Fibrinogen was low at 1.51g/L. She had episodes of hypertension in the ICU requiring IV labetalol and hydralazine. She was ultimately discharged home on oral labetalol.

There was no fetal heart rate at delivery and a "Code Pink" was called. There was a true knot in the cord and the cord gas samples clotted. Chest compressions were commenced and the baby was intubated at five minutes of age. Apgars were 0, 0 and 6 at one, five and ten minutes of age. Capillary pH at 1720 hours was 6.98. The baby developed seizures at two hours of age. The baby was given phenobarbital and passive cooling was commenced. The baby was subsequently transferred to Hospital B (a children's hospital). The baby's course at Hospital B showed subjective markers of severe hypoxic insult with prolonged metabolic acidosis and poor neurologic status. The medical team agreed to the parent's request to withdraw ventilator support. The baby was electively extubated at midnight and was transferred back to Hospital A where he died at 0125 hours on October 12, 2010.

Post mortem

Autopsy findings showed severe hypoxic-ischemic encephalopathy.

The placenta showed evidence of significant abruption. There was a true knot in the umbilical cord.

The cause of death was severe hypoxic-ischemic encephalopathy due to acute placental abruption.

Discussion

This infant died from complications of acute and severe perinatal asphyxia as a result of a large, concealed placental abruption. Hypertension is the major risk factor for placental abruption and

although not significantly elevated prenatally, the maternal blood pressure was not normal. The mother experienced two generalized seizures in keeping with a diagnosis of eclampsia. This condition can develop very acutely although it appears that there had been enough of a change in her blood pressure to warrant blood work investigations at a prenatal visit. It cannot be determined from the records reviewed (as the Antenatal II was not provided) whether there was any advice given or further follow up arranged by her prenatal obstetrician.

Delivery occurred expeditiously at Hospital A, but given the size of the abruption, the hypoxic insult to the baby was significant by the time the mother presented to the hospital. It does not appear she was given MgSO₄ at the time of her first seizure but this is for maternal protection and would not impact the outcome of the baby. The choice of a spinal anaesthetic may be questionable given the need to secure the airway in an eclamptic patient and the risk of a coagulopathy with a concealed abruption. The airway was assessed and graded as Mallampati IV and a judgment was made that intubation would be too difficult. Again, this is for maternal protection and would not impact on the outcome of the baby.

Recommendations

No recommendations.

Case: 2011-N-10
OCC File: 2010-9903

History

The mother of the deceased newborn was a 27-year-old G₃T₁A₁L₁ with an EDD of August 1, 2010. She had a previous post term uncomplicated vaginal delivery in March 2009. Her health history was otherwise unremarkable. She had been followed throughout the pregnancy under midwifery care. There was detailed documentation on the prenatal records with the last notation on August 4, 2010 - three days prior to going into labour. All appropriate antenatal testing had been done which included a normal second trimester ultrasound scanning,

routine prenatal tests, a normal glucose challenge test, and negative GBS testing.

The mother went into labour at approximately 1900 hours on August 6, 2010 and contacted the midwife at 0600 hours on August 7, 2010. This had been a planned hospital birth and the mother was to deliver at Hospital A. However, when the mother called the hospital, she was told they were not able to deliver her baby as all of the birthing suites were full.

While en route to the residence, the midwife was speaking to the mother on the telephone and gave her the option of a home delivery, or delivery at Hospital B which was located a bit farther away. At that time, the mother was not certain what she wanted to do. The midwife arrived at the residence at approximately 0700 hours and after initial assessment, the mother confirmed that she wanted to deliver the baby at home. This decision was made after the initial assessment at 0715 hours where she was 5 cm dilated with bulging membranes with vertex at spines -2 with a normal fetal heart.

A back-up student midwife arrived at 0830 hours. At approximately 0900 hours, the mother was having episodes of vomiting and was on the floor on all fours. From 0715 hours until 0915 hours, the fetal heart rate was normal. At 0928 hours, the fetal heart was difficult to determine, but was recorded as 60 bpm.

Scalp stimulation was attempted by the midwives. An artificial rupture of membranes was done and meconium stained fluid was obtained; the cervix was 7 to 8 cm dilated. 911 was called at 0930 hours. Pushing with contractions was initiated and spontaneous vaginal delivery took place at 0935 hours. Midwifery records indicate that, "the cord was tight around the baby's neck but she was able to loop the cord over the head and free the cord" prior to delivery.

As there were no spontaneous respirations and heart rate was 60 bpm, positive pressure ventilation and chest compressions were commenced shortly after delivery. EMS arrived at 0937 hours and commenced resuscitation efforts.

At 0954 hours, the baby was intubated and epinephrine was given. There was apparently pulseless electrical activity with about ten beats per minute prior to transfer to Hospital B. Advanced resuscitation was continued; however the infant was pronounced dead at 1024 hours.

Post mortem

Post mortem examination revealed a 3580 g normally developed female infant. The placenta was normal in size and had meconium exposure effects. There was moderate umbilical vasculitis and funisitis. There was patchy mild acute subchorionitis. The fetal inflammatory response was felt likely secondary to meconium exposure rather than infection. There was evidence of recent /acute and older marginal abruption. There was vascular congestion and patchy edema in the brain.

The pathological findings showed morphologic stigmata of perinatal asphyxia; however an underlying fetal cause for this was not identified. The placenta showed findings attributable to intra-uterine meconium exposure but a definitive placental etiology for perinatal asphyxia was not apparent.

Discussion

This infant died of perinatal asphyxia of undetermined cause after an unremarkable pregnancy and a labour which appeared normal up until the last half hour. The baby was delivered compromised and could not be resuscitated.

The investigating coroner obtained copies of the hospital policy for bed gridlock and redirect. Apparently, the activity on the obstetrical unit of Hospital A on the day of delivery was very high. Records indicate that the staff on the unit did not recall having a discussion with the midwife.

It is unclear if the outcome would have changed if the mother had gone directly to the hospital after the initial assessment by the midwife.

It would appear to have been a reasonable recommendation by the midwife to have the

delivery at home in this otherwise uncomplicated pregnancy to that point.

Recommendations

No recommendations.

Case: 2011-N-11
OCC File: 2010-6410

History

The mother of the deceased infant was a 22-year-old G₁P₀ with an EDD of May 17, 2010. The pregnancy was complicated by first trimester nausea, group B streptococcus and bacteriuria in late first/early second trimester and false preterm labour treated with betamethasone at 27 weeks 3 days gestation.

On May 18, 2010 at 0700 hours, the mother presented to Hospital A at 40 weeks 1 day gestation with spontaneous rupture of membranes. The fluid was clear. Her cervix was fingertip dilated, long and thick. Assessment of early labour with spontaneous rupture of membranes was made. Pen G 5 million units intravenously was given and the patient was encouraged to ambulate. Pen G 2.5 million units was given intravenously every four hours after the initial loading dose. The admission database was initiated at 1340 hours. At this time, contractions were mild, every 2-4 minutes, lasting 40–60 seconds and the fetal heart baseline was 120-135 bpm with average variability and accelerations present.

Between 2045 and 2115 hours, there were “variable” decelerations from baseline. Contractions increased in frequency and intensity. Her cervix was 2 cm, 25% effaced and presenting part was at spines -2. A review of the EFM strips showed gradual decelerations from 140-110 bpm starting after peak of contraction and persisting beyond the end of contraction. The patient was turned to her left side and nalbuphine 10 mg intramuscularly (IM) was given. The decelerations resolved and amniotic fluid was still clear.

The fetal heart rate tracing was normal until approximately 2300 hours when there were two early decelerations to 90 bpm lasting 60 and 20

seconds respectively. Average variability of 10-15 bpm was noted and the decelerations resolved. An epidural was placed. A Foley catheter was inserted at 0025 hours. Oxytocin augmentation was started at 0055 hours as there was no change in cervical dilatation.

At 0115 hours on May 19, 2010, pain was noted in the right abdomen and the patient was turned. Fetal heart rate was 90-120 bpm for approximately five minutes. Until 0218 hours, there were gradual decelerations from a baseline of 125 to 90-100 bpm that started after the peak of the contraction. Not all decelerations persisted beyond the end of the contraction. Variability between contractions was 5-10 bpm. These were reported to the obstetrician as “variable decelerations.” At 0225 hours, the oxytocin infusion was stopped and the obstetrician was notified.

At 0300 hours, there was a deceleration to 90 -100 bpm for ten minutes with some increases to 120-130 bpm for 20-30 seconds. The obstetrician was notified again and assessed the patient. The cervix was 5-6 cm dilated, head was “low”, and fetal heart rate (FHR) “recovered by change of position to LLDP.” There was no change in management and oxytocin remained off. For the next 45 minutes, the EFM showed a baseline of 140 bpm with some variable decelerations, but not with every contraction. Variability was average.

Baseline FHR increased to 160, and then at 0418 hours, there was a deceleration with moments of loss of contact. The FHR was 90-140 bpm for 3-4 minutes. By 0430 hours, the baseline had increased to 180-190 bpm with a deep, variable deceleration approximately every six minutes to 80 bpm lasting 20 seconds. Maternal temperature was 39.5°C, cervix 7-8 cm, 100% effaced and a note was made that the doctor was aware.

At 0530 hours, a note by the registered nurse in the labour/birth flowsheet indicates that the doctor was in to discuss the plan of care with the patient. No documentation was found about the contents of the discussion. Gentamicin 80mg intravenously, ampicillin 2g intravenously and Tylenol were started

and Pen G was continued. Over the next 90 minutes, the baseline FHR was between 160 – 180 bpm.

At 0700 hours, there was a change in the doctor on call. The heart rate showed a deceleration to 100 bpm lasting one minute, then 120-140 bpm, lasting two minutes. The doctor assessed the patient at 0715 hours and found her cervix to be fully dilated and presenting part was at spines -1. The epidural was turned down to “facilitate pushing.” FHR baseline was in the 160s with beat to beat variability of 10-15 bpm. Some decelerations to 140 were noted, but not consistently related to the contractions.

At 0918 hours, there was a deceleration to 70 bpm lasting 90 seconds. At 0925 hours, the obstetrician assessed the patient and had her start pushing. The FHR was in the 120s between contractions and 140s during contractions.

At 0938 hours, the FHR decreased to 80 bpm. An assisted delivery with Simpson forceps was attempted but the forceps were not successfully applied. The vacuum was applied for one pull, and then it popped off. The presenting part descended to “almost +2.” The FHR was noted to be 70 bpm. At 0945 hours, Wrigley forceps were applied and “slightly further” descent with traction was noted. At 0950 hours, the forceps were removed and the patient was allowed to push on her own.

The EFM showed a baseline FHR of 150-160 bpm with a deceleration to 140 bpm through the peak of the contraction, then a “shoulder” and/or overshoot to 160 bpm with a slow recovery to baseline by the end of the contraction. Variability was minimal. At 1040 hours, the oxytocin was restarted. The FHR pattern continued until about 1100 hours when the tracing showed repeated variable decelerations from 160 bpm down to 80-100 bpm every 20-40 seconds lasting 10-20 seconds. In the last ten minutes prior to delivery, the FHR was in the 140s with pushing and 120s between contractions/pushing efforts. The notes indicated that the obstetrician was present as the mother pushed.

Delivery occurred at 1130 hours and a “Code Pink” was called.

The Apgars were 1 and 3 at one and five minutes respectively. There were no spontaneous respirations, HR was 40/min and the infant’s colour was grey. Birth weight was 4079 g and the head circumference was 33 cm. A subgaleal and cephalohaematoma was noted. The resuscitation team initiated bag and mask ventilation with 100% oxygen. There was no significant response. The infant was intubated at six minutes of age and a meconium aspirator was used to suction meconium-stained mucus. The infant was promptly re-intubated with a # 3.5 endotracheal tube (ETT) and the HR increased to 100 bpm with manual bagging and colour also improved. However, the pupils were 3 mm and fixed and the infant was hypotonic with no respiratory effort.

A peripheral IV was started, a 40 ml bolus of saline was given and the infant was placed on a ventilator. A partial septic work up was done and the infant was started on ampicillin and gentamycin.

The arterial cord gases were: pH=6.8, PCO₂=88, HCO₃=14 and base excess = - 23. Two further boluses of 40 ml of saline were administered.

At 1144 hours, passive cooling was started and the infant’s temperature dropped to 33.7°C. By 1211 hours, a repeat assessment showed the HR was 145 bpm, blood pressure (BP) 89/34, Mean Arterial Pressure (MAP) of 44 and oxygen saturation was 99% in Room Air. However, the infant had no tone and no spontaneous respirations.

At 1238 hours, at approximately 48 minutes of age, some gasping was noted with a rate of 5-8 breaths per minute; pupils became reactive. By 1250 hours, the HR was 112 and O₂ saturation was 99%. At 1302 hours, umbilical venous (UV) and umbilical arterial (UA) lines were placed. A chest x-ray and abdominal x-ray were done for placement of the ETT and catheters.

The transfer team from the children’s hospital arrived at 1330 hours. At 1335 hours, seizure-like activity was noted and the infant was treated with phenobarbital. After a thorough examination and complete assessment of blood work, it was felt that

the infant was significantly compromised and the neurological outcome was very poor. This was discussed with the family and withdrawal of active treatment was agreed upon. The infant was extubated and passive cooling was discontinued at 1717 hours.

The infant was pronounced dead at 0732 hours on May 20, 2010.

Post mortem

Significant findings at autopsy included:

- subgaleal hemorrhage, left cephalo-haematoma and facial abrasion;
- adrenals showed localized hemorrhagic cortical necrosis;
- lungs showed meconium aspiration with acute inflammatory reaction/pneumonia;
- brain showed severe hypoxic - ischemic encephalopathy.

Examination of the placenta indicated acute chorioamnionitis and funisitis-related umbilical artery myonecrosis.

The major findings at post mortem relate to the sequelae of severe perinatal asphyxia, which is attributable to acute chorioamnionitis.

Discussion

This infant died as a result of severe hypoxic ischemic encephalopathy associated with chorioamnionitis. The mother had prolonged rupture of membranes and developed a fever and fetal tachycardia. She was given oxytocin to expedite her delivery and since she became afebrile, the tachycardia resolved. Once fully dilated and after a period of rest to allow descent of the presenting part, an assisted vaginal delivery was attempted unsuccessfully. Although the EFM tracing was at times atypical and abnormal, the mother was allowed to push for an additional 1 hour and 45 minutes and have a vaginal birth.

The literature is not clear with regards to active management to deliver or pursue expectant

management with premature rupture of membranes (PROM). In this case, neonatal resuscitation was effective in establishing respiratory and cardiac functions, but the baby had suffered significant neurological damage.

The SOGC Clinical Practice Guideline on Fetal Health Surveillance: Antepartum and Intrapartum and Consensus Guideline (No. 197, September 2007, pp 7-8), outlines the method of systematic interpretation of continuous external fetal monitoring. In this case, the fetal heart rate pattern from 2045 to 2115 hours was reported as “variable decelerations” when the pattern was more consistent with late decelerations. The appropriate step of turning the patient to her side and giving nalbuphine in a presumed attempt to reduce maternal anxiety resulted in improvement of the EFM pattern. However, decelerations recurred with a change of baseline. Cervical dilatation was changing at a rate of approximately 1 cm per hour.

Health care providers are reminded that it is important to document:

- fetal heart rate data
- uterine activity characteristics obtained by palpation
- interpretation of the EFM as either “reassuring” or “non-reassuring” (and specific actions taken)
- maternal observations and assessments
- fetal and maternal responses to interventions and;
- subsequent return to normal findings

(source: SOGC Guideline No. 197, September 2007, p.11).

When there are variable decelerations with atypical features, this is considered a “non-reassuring” fetal heart rate pattern. Clinical actions for intrauterine resuscitation, confirmation of fetal well-being and consideration of expediting delivery should be discussed, documented and undertaken. (SOGC Guideline No. 197, September 2007, p. 9, table 7)

The SOGC Clinical Practice Guideline for Operative Vaginal Birth (No. 148, August 2004, p. 750), states that:

Fetal injuries have been attributed to delay between a failed operative vaginal delivery and a Caesarean section. Most operative deliveries, including the application of a vacuum, should be considered a trial. Unless the practitioner is certain that an operative vaginal delivery is going to be successful, the possibility of failure needs to be anticipated. In these circumstances, an alternative plan that will result in a safe and expeditious birth must be in place and implemented promptly if the planned operative birth is unsuccessful.

In this case, the Simpson forceps, then the vacuum, then the Wrigley forceps were applied after a prolonged deceleration. When these failed, and with a persistently abnormal fetal heart rate tracing pattern, the time to delivery was still ninety minutes. Furthermore, in this interval, oxytocin augmentation was restarted. Oxytocin increases the contractility of the uterus and thus increases the impact of the impaired uteroplacental insufficiency on the fetus. Oxytocin should not be started with an abnormal EFM tracing.

Recommendations:

1. The obstetrical care providers from Hospital A should review the *SOGC Guidelines for Operative Vaginal Birth (Recommendation 5)* which states, "Failure of the chosen method, vacuum and/or forceps, to achieve delivery of the fetus in a reasonable time should be considered an indication for abandonment of the method" and proceed to immediate delivery."
2. The obstetrical care providers from Hospital A should review the *SOGC Clinical Practice Guidelines for Fetal Health Surveillance: Antepartum & Intrapartum Consensus Guideline (No. 197, September 2007)*.
3. The obstetrical care providers from Hospital A should review the assessment, treatment and implications of chorioamnionitis.

4. The Regional Supervising Coroner should conduct a Regional Coroners Review of the facts surrounding this case.

Case: 2011-N-12
OCC File: 2011-6118

History

The mother of the deceased was a 28-year-old G₄T₂A₁ Mennonite woman with an EDD of May 9, 2011. She had two previous term deliveries of infants weighing 6 pounds 12 ounces and 7 pounds 4 ounces. The most recent birth was in 2009 and the infant was delivered at home after four hours of labour. For that birth, the obstetric history indicated that there had been "fetal distress 2nd stage" and "shoulder dystocia during the delivery with one maneuver." For the current pregnancy, it was noted that the woman was "ok for homebirth, ensure 2nd midwife, possible hospital."

All routine prenatal testing had been done including a normal glucose challenge test. The Ontario prenatal records were completed. The last documented exam on May 10, 2011 at 40 weeks and 1 day gestation suggested a large infant with a symphysis fundal height (SFH) of 41 cm. The mother weighed 166 pounds at term. She was under midwifery care for her pregnancy, until being transferred to a physician's care in the second stage of labour.

On May 11, 2011 at 40 weeks and 2 days gestation, she started labour early in the day. The midwives documented that labour started at 1400 hours. She had an artificial rupture of membranes at 1713 hours for clear amniotic fluid and was fully dilated and pushing starting at 1815 hours with the presenting part at spines -1. Fetal bradycardia was detected with intermittent auscultation for a period of 40 minutes at the beginning of second stage and was resolved after frequent maternal position changes. At 2040 hours, after consultation, an ambulance was called for transfer to hospital as delivery was not imminent after 2.5 hours of pushing. The fetal heart was felt to be reassuring following the initial period of 40 minutes of bradycardia.

The woman was first assessed by the physician at 2145 hours at which time she had been fully dilated for approximately 3.5 hours. The fetal heart was normal. The fetus was felt to be at station 0 between contractions with lots of molding and caput and likely a direct occiput posterior position.

With pushing during contractions, she was able to move the vertex down to +2 to +3. The options of a trial vacuum assisted delivery versus proceeding to Caesarean section were discussed with the patient. The patient “decided to give the trial of vacuum an attempt as the operating room was in use at the present time with another case.”

Another physician was called to tend to the baby after delivery. With the use of a kiwi vacuum and with maternal pushing, it was brought down to the perineum during three contractions. The baby was delivered to the jaw and the vacuum released, at which point shoulder dystocia was encountered. The total vacuum time was six minutes.

The dystocia was felt to be quite severe and physicians noted that they went through multiple maneuvers starting with McRoberts, then supra pubic pressure, then Rubin maneuver, then Wood’s corkscrew, then placing the mother on all-fours and finally returning her to her back and delivering posterior arm and shoulder. The total time to delivery was approximately seven minutes with the baby finally being delivered at 2215 hours. The baby was born flat with no heart rate. The Apgars were 0, 0 and 2 at one, five and ten minutes.

During the shoulder dystocia, a “Code Pink” was called. The baby was intubated and an endotracheal tube placed, chest compressions initiated and epinephrine, oxygen and fluid bolus were administered. The first heart rate was noted at 40 bpm on the monitor at 10 minutes of age. At 26 minutes of age, they were first able to feel palpable pulses with return of spontaneous respiration and heart rate of 118 bpm. The neonatal service at the tertiary hospital was consulted. Over the next hour, bradycardia returned and there was no spontaneous respiration with ongoing poor color and no spontaneous movements. At this point, the possibility of starting a pressor agent was

considered, but after discussion with the mother, they proceeded with the request to withdraw further treatment.

Assisted ventilation was discontinued. The baby continued to deteriorate and was pronounced dead at 1 hour and 54 minutes of age on May 12, 2011.

Post mortem

No post mortem was conducted as the body had already been removed to the funeral home and embalmed.

Examination of the placenta revealed no abnormality.

The newborn weight was 3960 grams (8 pounds 12 ounces). The delivery record indicated that cord gases were taken, however, no results could be found in the documentation provided. Venous gases were done at one hour of age revealing a PH of 6.69.

Discussion

This baby died subsequent to a delayed delivery after a severe shoulder dystocia. The mother had been appropriately transferred to the hospital in labour as she was not making adequate progress with imminent delivery after 2.5 hours of pushing. It is not known if there was discussion between the midwife and physician regarding previous shoulder dystocia, and that she was measuring large for gestational age (LGA) with a SFH of 41 cm on her last visit. This information, along with a very prolonged second stage of labour (i.e. 3.5 hours), may have influenced the caregiver’s consideration of potential shoulder dystocia and subsequent Caesarean section. All appropriate maneuvers were attempted for delivery as well as preparations for assistance with resuscitation of the newborn.

The planned place of delivery may have been reconsidered in late pregnancy with the shared knowledge of large SFH and a documented history of shoulder dystocia. There was no documented informed choice discussion or decision by the parents regarding planning a birth outside the

hospital with their documented obstetric history and the current clinical findings.

As a timely transfer to hospital did ultimately occur, it is unlikely that a change of plan would have affected the outcome, though it may have facilitated communication at the time of transfer of care.

The fetal bradycardia for a period of 40 minutes may have been better interrupted if electronic fetal monitoring had been commenced.

Recommendations

1. Obstetrical care providers are reminded that specific preparation for delivery, including the most appropriate place of birth, should be considered when the risk of shoulder dystocia is considered to be high.
2. Obstetrical care providers are reminded that communication between care providers at the time of transfer of care is essential and that detailed transfer forms may help facilitate this sharing of information.
3. Obstetrical care providers are reminded of the *SOGC Advances in Labour and Risk Management of Labour (2010-2011) Guidelines* with respect to prolonged second stage in labour.
4. Obstetrical care providers responsible for fetal monitoring should follow the guidelines as set out in the *SOGC Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline, JOCG Vol29 No9, Sept, 2007*.
5. The Regional Supervising Coroner (RSC) should conduct a review of the circumstances surrounding the death of this infant.

Case: 2011-N-13
OCC File: 2010-12816

History

The mother of the deceased infant was a 29-year-old G₃A₂ with an EDD of November 1, 2010. Previously, she had two first trimester miscarriages that were otherwise uncomplicated. She was followed under

full midwifery care up until 32.5 weeks gestation. She was referred to physician's care when she presented to the midwifery clinic with an elevated BP at 148/98. She was started on labetalol and her BP settled for a time. On October 8, 2010, her BP was again elevated at 154/94, so the labetalol was increased to 200 mg by mouth, three times a day. She was admitted for induction of labour.

Blood testing done on the day of admission revealed a normal complete blood count (CBC), including platelets, normal liver function tests and creatinine. There was no documentation of any ultrasounds being performed. A physician note on October 8, 2010 indicated that a vaginal exam revealed a closed cervix with the head at spines -2 and a notation that cervadyl was to be used. There were no further physician notes until October 10, 2010 at 0815 hours when the patient was fully dilated.

The nurse's notes and partogram indicated that the "induction" was started at 0840 hours on October 9, 2010. The membranes had spontaneously ruptured at 2022 hours on October 9. She had an epidural inserted shortly before midnight on October 9 and around that time, was felt to be in active labour. She became fully dilated by 0805 hours on October 10, 2010. At that time, an obstetrician was present for the first time since approximately 48 hours before, though nursing staff notations throughout the labour indicate reports to the attending physician. The attending physician was again called by nursing staff at 0915 hours after an hour of pushing with no progress of descent. The physician was asked to come and assess as the nurse felt there were "decelerations to the 90's with good recovery; some late and early decels noted."

The physician came at 0930 hours and again at 0943 hours on October 10. Due to repeat decelerations, oxytocin was stopped, O₂ was given and a scalp clip was applied at 0950 hours. A decision was made to proceed to Caesarean section.

At 0953 hours, the patient was transferred to the operating room (OR) by stretcher. A second team was required for a stat emergency section as another surgery was already in progress. While waiting in the OR, the monitor was re-attached and

there were no further significant decelerations and the patient was not pushing.

With adequate anesthesia from the epidural, a low segment Caesarean section was performed with incision at 1026 hours. The baby was delivered at 1031 hours without incident. The infant was handed off to pediatric staff who were in attendance for delivery as they had been called prior for the emergency delivery. A two layer closure was done and the rest of the procedure was unremarkable.

Apgars were 1, 1 and 1 at one, five and ten minutes. Arterial cord PH was 6.99 with a base deficit of 24 and a venous PH of 7.06 with base deficit of 18. The 1 for Apgar was only for the heart rate that was below 100 per minute. The baby had no tone, respiratory effort, coughing or sneezing and the color remained cyanotic. Positive pressure ventilation (PPV) was started immediately followed by chest compressions and then intubation followed by endotracheal epinephrine. An umbilical venous catheter (UVC) line was started through which further doses of epinephrine, IV fluids and antibiotics were administered. Dopamine was subsequently started and gradually increased. A heart rate above 100 was noted around 37 minutes of age at which time chest compressions were stopped though ventilation through the ET tube continued with the ventilator.

The team from a tertiary hospital was called and asked to assist with the stabilization and eventual transfer. The baby's condition continued to deteriorate however, and after a total of seven doses of epinephrine, the heart rate continued to drop and it was felt that further resuscitation attempts were futile. The baby's resuscitation was discontinued at 1225 hours, almost two hours after birth. The baby was pronounced at 1230 hours.

Port mortem

The autopsy revealed a normally developed 2595 g, thin, male infant with no congenital abnormalities. The placenta had no gross abnormalities. Microscopic examination of the placenta revealed hypertensive changes in the vessels of the placenta with mild muscular hypertrophy supportive of poor

placentation. There was fibrinoid necrosis of some vessels.

The brain examination showed histological features consistent with acute anoxic ischemic encephalopathy affecting predominately the cerebral cortex of the occipital and temporal lobes. All virology tests were negative. Toxicological findings of the presence of lidocaine and bupivacaine are in keeping with the administration of the anesthetic agent to the mother.

The cause of death was attributed to placental insufficiency leading to acute anoxic ischemic encephalopathy.

Discussion

The mother had pregnancy-induced hypertension and was appropriately admitted for induction of labour. There is no evidence that ultrasound examination, biophysical profile, assessment of amniotic fluid volume or cord doppler studies had been done.

There were very scanty physician notes during the whole time that the patient was in hospital prior to delivery. It would appear that a physician did not see the patient while she was in labour until she was fully dilated. A nurse in attendance documented contacting the physician on a number of occasions to report progress.

Throughout the night prior to the delivery, there had been continuous fetal monitoring and variable decelerations with most contractions but with good recovery. From the time of initiating pushing shortly after 0800 hours, the fetal heart pattern changed with more prolonged recovery to baseline. By 0915 hours on October 10, 2010, when the attending physician was asked to come and see the patient, the fetal heart monitor revealed a distinctly abnormal fetal heart. When the physician first attended it would have been prudent to apply a fetal scalp clip. It was however, a further half hour before a decision was made to stop syntocinon, initiate a scalp clip and plan for a Caesarean. This delay, and the subsequent need to wait for a second team to

arrive at the hospital for assisting for delivery, likely contributed to the outcome.

This fetus was the product of a pregnancy with pregnancy-induced hypertension with placental findings compatible with hypertensive affect, which likely predisposed to stress of labour. Information from a biophysical profile with doppler studies prior to the initiation of the induction may have given the attending physician a higher index of suspicion, though the outcome may not have changed.

By the time the baby was delivered, he was extremely obtunded. Despite aggressive and prolonged attempts at resuscitation, the infant could not be revived.

Recommendations:

1. The Regional Supervising Coroner should conduct a Regional Supervising Coroner's Review of the circumstances surrounding the death of this infant.
2. Obstetrical care providers are reminded about the importance of ultrasound assessment of fetal growth and well-being in the management of hypertensive obstetrical patients.

Case: 2011-N-14
OCC File: 2010-15786

History

The mother of the deceased infant was a 37-year-old G₄P₃ with an EDD of December 15, 2010. She had two previous vaginal births in 1992 and 2006. In 2008, she had a Caesarean section at 40 weeks gestation for failure to progress. She had been induced for an increased glucose of +2. Medical history indicated depression x 8 years and a rare occurrence of genital herpes. During the current pregnancy, she was given ranitidine at 28 5/7 weeks gestation to treat nausea associated with heartburn. A notation on the file indicates that an oral glucose challenge test was needed, however no results were

recorded on the antenatal records. She decided to deliver by Caesarean section and was referred to a physician in a larger centre at 36 weeks gestation. A repeat Caesarean section was booked for December 8, 2010. GBS status was unknown.

Course in Labour and Delivery

On December 7, 2010 at approximately midnight, the mother developed irregular contractions. Her membranes were intact and she had no bloody show. Vital signs were normal on admission to hospital. A vaginal examination revealed a posterior, thick multipos although the doctor indicated that he was unable to determine the cervical dilatation. Attempts to reach the mother's physician were unsuccessful and she was seen by another doctor who provided surgical service. Since an elective Caesarean section had already been planned for, the decision was made to proceed with the procedure immediately.

The male infant was delivered at 0527 hours on December 7, 2010 under epidural anesthesia. He cried immediately and Apgars were 9 and 10 at one and five minutes. Birth weight was 3400 g and no abnormalities were noted. Cord gases were normal.

Neonatal History

The infant was located in the room with the mother and appeared stable until approximately 2000 hours. His heart rate fluctuated between 140-168 bpm and respirations were 40-60. Mild grunting was noted at 1100 hours, however no oxymetry was available. His colour was described as "brick dust" and the darkness of his forehead was attributed to bruising or natural colour (based on the father's ethnic background).

The infant was drowsy and feeding poorly. His suck was disorganized and finger feeding resulted in a 6 ml feed. At 1646 hours, he was still in a drowsy state, still feeding poorly and was choking easily. He managed another 6 ml finger feed. The next entry was at 2308 hours when notes indicated that his temperature was low at 35.5C, respirations were 60 and he was still drowsy. A nurse double-wrapped

the infant in warm blankets and spent an hour feeding him another 15 ml.

At 0230 hours, the infant's temperature remained low at 35.8 C. He was still drowsy and his hands and feet were cyanotic. His cry was weak, but lusty at times. At 0313 hours, the mother pumped 10 ml of breast milk and fed it to the infant. The nurse then took the infant to the nurses' station to allow the mother to sleep.

Nursing notes indicated that the infant's respiratory rate was 50-60 during the night. No central cyanosis was noted. The infant's temperature was difficult to maintain and he was wrapped in warm blankets twice during the night. The plan was to continue monitoring the baby.

At 0355 hours, the infant was found in the cot unresponsive, apneic, flaccid and pulseless. CPR was commenced with bag and mask ventilation with 100% O₂. At 0400 hours there was no response and after three attempts, the infant was intubated with a #3.5 endotracheal tube (ETT). Air entry appeared to be symmetrical. The infant received two doses of 1:10,000 epinephrine (3 ml) via ETT. An attempt at peripheral IV and intraosseous (IO) were initially unsuccessful. Eventually an IO was inserted in the left femur. The infant received a total of three doses of epinephrine and two 30 ml saline boluses.

The infant's blood glucose was low at 1.3, so he was given 15 ml of 10% dextrose. His pupils were dilated and fixed. Resuscitation efforts were unsuccessful. The infant died at 0455 hours on December 8, 2010 at 23 hours and 28 minutes of age.

Post mortem

Toxicology: Normal

Cytogenetics: Karyotype XY

Metabolic: Negative except for elevated TSH of 54

Microbiology: All cultures negative. Streptococcus mitis was isolated from the lungs – non pathogenic likely a contaminant

Autopsy: Negative

Neuropathology: Unremarkable

Discussion

Although it appears that there was a sudden change in the infant's condition, there were signs of concern that presented earlier. In particular, the infant's body temperature was low at 35.2 C, his colour was questionable, there was mild tachypnea and his feeding was poor with a disorganized suck and at times a weak cry. Although not impressive in isolation, these constellations are signs of concern and should have been reported to the most responsible physician (MRP).

Further blood work such as CBC, blood gas, lactate, blood sugar may have been helpful in further assessing the infant's condition.

Close monitoring of vital signs, including oxygenation, is invaluable in these circumstances.

During resuscitation, a UV line placement is swift and allows central venous access. This allows efficient drug administration and ease of obtaining blood work. Cardiac compressions were not documented.

The elevated TSH did not likely contribute to the death of this infant as a transient surge in TSH after birth is not unusual.

It is also recognized that the area had been hit with severe winter weather which closed highways and roads making transportation and transfer difficult, if not virtually impossible.

Recommendations

1. Obstetrical and neonatal care providers are reminded that:
 - a) Critical attention is required to monitor, recognize and react to changes in newborns;
 - b) Early investigations and close monitoring are crucial to the management and care of newborns;
 - c) IV access is critical and UV line placement is expeditious and useful;

- d) Neonatal Resuscitation Program (NRP) certification is highly recommended for all individuals involved in the care of newborns;
 - e) Complete and accurate documentation is essential.
2. The Regional Supervising Coroner (RSC) should conduct a RSC review of this case, particularly as it relates to infant monitoring, use of investigations, documentation and communication.

Case Summaries: Stillbirths

Case: 2011-S-1
OCC File: 2010-452

History

The mother of the deceased was a 25-year-old primip with an EDD of January 4, 2010. She had a history of asthma, which did not require regular treatment and her past medical history was otherwise unremarkable. Prenatal testing was normal, including a glucose challenge test at 28 weeks. An 18 week second trimester ultrasound scan was normal. She was positive for GBS with plans for penicillin in labour.

She was seen regularly in the office until January 6, 2010 at 40 weeks and 2 days gestation with a plan for induction on January 12. Her visits were well documented and there was no evidence of concern throughout the pregnancy.

She was admitted in spontaneous labour on the afternoon of January 11, 2010 at 41 weeks gestation. She was given penicillin prophylaxis in labour. She progressed from 4 cm at admission at 1400 hours, to become fully dilated at 2230 hours. Pushing was initiated at that time. A short time later, a vaginal exam found the infant to be in an occiput transverse position.

Oxytocin was initiated and she was advised to stop pushing for the next hour. She resumed pushing at 0100 hours. There were variable decelerations of the fetal heart, but with good recovery when pushing. The baseline had risen to 165-170 bpm, having been in the 138-140 bpm range for most of the day. The obstetrician on call recommended that they proceed to delivery with an attempt of forceps. While in the room, an attempt to manually rotate the fetus from Right Occipit Transverse (ROT) was not successful.

Fetal monitoring continued during the forceps attempt. The patient's epidural was topped up, her

bladder was emptied and she was re-examined and felt to be 2 to 3 cm below spines in ROT with adequate pelvis. Kielland forceps were used and the operative note indicates that a proper application was confirmed. Between contractions, "the infant was easily rotated from ROT to an OA (Occipit Anterior) position."

There was an attempt at traction during two contractions and there was no significant descent. During this time, the fetal heart was between 140 and 160 bpm. The forceps attempt was started at 0203 hours and forceps were removed at 0213 hours. The physician attempted to push the baby's head up. A Caesarean section with a transverse lower segment incision was then performed. It was noted that, "the head was well wedged in the pelvis and there was difficulty in delivering the head up to the incision." Nitroglycerine and trendelenburg were requested. With these maneuvers, the head was finally brought out of the pelvis and with uterine relaxation, the baby turned from vertex to transverse, with the back down. The baby was subsequently delivered as a breech.

Cord blood gases were done indicating arterial PH of 7.09 and normal bicarbonate and cord venous gas of 7.06.

The infant was born with vital signs absent at 0232 hours. Despite resuscitation, there continued to be no pulse and the resuscitation stopped at 0257 hours - 25 minutes after delivery. The anesthetist initially used bag and mask with 100% oxygen and then intubated. The pediatrician had additional help from nursery staff who were paged. The infant was given epinephrine via endotracheal tube and subsequently epinephrine and bicarbonate via umbilical vein catheter. Throughout this time, the baby was bagged with 100% oxygen, but did not gain vital signs.

The coroner attended the hospital at 0500 hours and reviewed the chart and interviewed the nurses,

pediatrician, obstetrician and parents. The infant was examined and there was no evidence of gross abnormality or signs of trauma. After further discussion, the family and the coroner did not feel that an autopsy was required. Two days later, the coroner was advised that a medical autopsy had been performed at the children's hospital. The initial pathological findings included a small, localized, undisplaced skull fracture and some subdural blood. The significance of these findings was unclear. A medico-legal autopsy and further neuropathology testing was requested by the investigating coroner.

Post Mortem

Post mortem findings included:

1. Parieto-occipital scalp hemorrhage bilateral, marked;
2. Parasagittal fracture localized, left parietal bone;
3. Subdural and subarachnoid hemorrhage mild, over cerebral convexities with extension along the basal surface of brain and spinal cord;
4. Diffuse axonal injury;
5. Radiological findings suggestive of subluxation at atlanto-occipital junction with no evidence of soft tissue hemorrhage identified on posterior neck dissection;
6. Placental examination unremarkable.

There was no evidence of skull fracture on x-rays (although this was noted on the initial pathological examination).

The pathologist summarized the findings as:

“parietal and occipital scalp hematoma; isolated fracture of the left parietal bone adjacent to sagittal suture (“attributed blunt force compression of the skull at the time of forceps application”), and evidence of diffuse axonal injury (which in this setting is “most likely related to a traumatic torsional etiology”).”

In conjunction with the radiological findings, they felt that the possibility of cardiorespiratory arrest secondary to spinal cord trauma was a consideration.

Discussion

The course of this pregnancy was unremarkable until shortly before the attempted forceps delivery. At that time, the fetus was in the ROT position with the presenting part below spines. The obstetrician indicated in the notes that it was an appropriate forceps application and easy rotation to the anterior. It would appear from the operative report that significant injury was not likely caused at this point of the procedure.

The fetal head was quite tight in the pelvis. The obstetrician described difficulty on extracting the fetal head. The left parietal bone would have been anterior with the fetus in the ROT position. It is quite possible that the isolated fracture on the left parietal bone was associated with the operator's hand attempting to get the head out of the pelvis. As well, it is possible that there could be torsion of the head during a difficult delivery at Caesarean section. If the central nervous system (CNS) injury is the cause of the failure of the infant to respond at birth, it may have occurred at the time of attempted extraction of the fetal head from the pelvis and not during the forceps application. This will remain uncertain. This type of injury and subsequent stillbirth delivery, under these circumstances and conditions, is quite uncommon.

With the cord PH>7, and despite contribution from CNS injury, it is not clear why this newborn could not be stabilized. The cause of death is not clear. The manner of death was undetermined.

Recommendations

No recommendations.

Case: 2011-S-2
OCC File: 2009-4937

Antenatal History

The mother was a 40-year-old, G₁₅T₅P₂A₇L₆, with no documented prenatal care in this pregnancy. She had an EDD of May 29, 2009. She reported a history of right hip fracture, left eye blindness and multiple dilation and curettage (D and C) procedures. She

believed she was Hepatitis C positive. She had a previous Caesarean section, was blood type O positive and was allergic to penicillin. This woman erroneously believed that she had undergone a tubal ligation procedure at the same time an abortion was conducted in 2007.

The mother had problematic substance use of alcohol, nicotine, marijuana, opiates, benzodiazepines and stimulants. She reported daily use of drugs, alcohol and smoking. Her long-standing substance abuse problems were well documented.

This woman had 14 pregnancies prior to this stillbirth. She had six living children. Three of her children (ages 21, 18 and 8) were still residing at home. During this pregnancy, the mother was being investigated by the Children's Aid Society (CAS) for the care provided to her 8-year-old child. She had previously lost three children to CAS protection.

Course in Labour and Delivery and Postpartum

On April 17, 2009 at 1730 hours, at approximately 34 weeks gestation, the woman began experiencing contractions while at home. At 1930 hours, she began to bleed vaginally and by 2030 hours, she reported blood and clots with each contraction.

Emergency Medical Services (EMS) were called and arrived at 2145 hours. The responding EMS crew found the woman on the living room floor having contractions that were causing clots to evacuate from the vagina. No vertex was visible.

EMS notified the CAS as they were aware the woman had an 8-year-old child (who was not home at the time). When EMS assessed the woman, she stated that she had been smoking marijuana and drinking beer with friends when the marijuana pipe started to taste "different" after it went around the room. She admitted to possibly, but not knowingly, smoking crack cocaine that day.

The woman was transported to hospital at 2207 hours and admitted to the Labour and Delivery Department at 2240 hours. Upon initial assessment, no fetal heart was detected and strong contractions

were palpated. On admission, her platelets were 202 and her haemoglobin (hgb) was 108.

April 18 at 0028 hours, an artificial rupture of membranes (ARM) was performed. Platelets and hgb had dropped and her fibrinogen was low at 1.3. The physician prepared for the possibility of the patient developing disseminated intravascular coagulation (DIC).

By 0110 hours, patient controlled analgesia (PCA) with fentanyl commenced and a Foley catheter was inserted. The patient progressed from being 7 cm dilated at 0200 hours, to being fully dilated at 0209 hours. A spontaneous vaginal birth of a stillborn male took place at 0212 hours. Shortly thereafter, the placenta was delivered and there was copious clotting.

The woman developed DIC following extensive blood loss. A total of 30 units of cryoprecipitate, 6 units red cells and 5 units plasma were transfused and the DIC was corrected with aggressive maternal resuscitation.

Post partum, the woman developed alcohol and nicotine withdrawal. Social workers from the hospital assessed the patient, but the assessment was ended abruptly when the woman began vomiting excessively. She was discharged at three days postpartum with a prescription for Percocet, a business card for a physician clinic and advised to follow up with the clinic for additional post partum care. The woman was anxious to return home, apparently to be with her 8-year-old child.

Post Mortem

Post mortem revealed a chromosomally normal 34 week gestation stillborn male.

Pathology of the placenta was not consistent with abruptio, however it did not exclude that possibility. The stillborn was small for gestational age.

Toxicology on the stillborn (including hair and meconium testing) was positive for alcohol, marijuana and benzoyllecgonine (cocaine

metabolite), and cocaine. The meconium testing identified cocaine, benzoylecgonine and cannabinoids at a medium level in second and third trimester and the hair testing indicated the same drugs, with low to medium exposure, in the third trimester. The baby was negative for Herpes simplex, cytomegalovirus and enterovirus.

Discussion

Drug dealers are known to lace marijuana with cocaine in order to increase the addictive quality of the substance and subsequently bolster the market for illicit drugs. The mother of the stillborn infant had a well-documented and long-standing history of substance abuse, including the use of illicit and prescription drugs.

The mother did not receive (and likely did not seek out) any prenatal care. During the prenatal period, the mother had CAS involvement pertaining to the care provided to her 8-year-old child. The CAS and other social service agencies who knew of her addictions and pregnancy, may have had an opportunity to influence referral of the mother into prenatal obstetrical care for the unborn child.

The mother of this stillborn had a documented and long-standing substance abuse problem. It does not appear that this history was taken into consideration when she was discharged from hospital with a prescription for Percocet and an informal referral (i.e. business card) for follow-up post partum care from a physician.

Recommendations

1. Obstetrical care providers should develop public information and referral materials, both electronic and hardcopy, which can be distributed or accessed by women who are experiencing problematic substance use/abuse issues during pregnancy and post partum.
2. Child protection workers should be encouraged and/or required to refer expectant mothers with a history of substance abuse who are the subject of ongoing child protection investigations, for appropriate and timely

obstetrical care. Consideration could be given to having abstinence testing as a condition of ongoing custody of children.

Case: 2011-S-3
OCC File:2009-705

History

The mother of the stillborn was a 36-year -old G₁₁P₆ Mennonite woman with an EDD of February 11, 2009. She received midwifery care during her pregnancy from 17 weeks to delivery and a Category 1 physician consult was obtained because of post partum complications in previous pregnancies.

She experienced an episode of vaginal bleeding early in the pregnancy. An ultrasound on August 20 showed a viable pregnancy at 14 weeks 4 days with a suspected amniotic chorionic membrane separation anteriorly. Her antenatal course and routine prenatal visits were otherwise uneventful.

Routine prenatal laboratory investigations were normal, except for a mild degree of anemia. She was taking prenatal vitamins and iron. She was Rh negative and antibody screen negative. Records do not indicate if Rh immune globulin was given in association with the episode of early vaginal bleeding. Prenatal genetic screening testing and an 18-20 week ultrasound were declined. She received Rh immune globulin on November 19 and the glucose challenge test (GCT) was normal.

Her past obstetrical history included four spontaneous abortions and six term pregnancies (two home births complicated by retained placenta and four hospital births, one of which was a vaginal breech birth). She was encouraged to deliver at the hospital because of this history, but declined. Her last pregnancy was complicated by post partum depression. Her past medical history was otherwise unremarkable.

The Antenatal Record 1 indicates a family history of cystic fibrosis, but no further information was provided.

The attending midwife was scheduled to be out of the country starting on January 19, so arrangements were made for the expectant mother to be followed by a physician.

Course in Labour and Delivery

The mother began having contractions on January 18 at 1400 hours at 36 weeks 2 days gestation. Vaginal bleeding occurred at 1700 hours. It is noted in the file that the woman wanted to go to the hospital by ambulance, but since access to her residence was closed due to weather, a decision was made to send her husband by horse and buggy to get help. A support person who was involved at the prenatal clinic was nearby and was brought back to the house at the request of the midwife, who was not available to personally attend as she was about to leave the country on a pre-scheduled trip. The support person, who was not a registered health care professional, could not confirm the presence of a fetal heart with a Doppler. Arrangements were made with a neighbour for transportation to the hospital at 1735 hours.

The woman was admitted to hospital at 1832 hours. The fetal heart rate was 80-90 (maternal pulse 110). The cervix was 3 cm dilated and findings were consistent with placental abruption. The fetal heart could not be detected and a bedside ultrasound was done at 1855 hours. A written note indicated there was no fetal heart detected, but a dictated note indicated the fetal heart was "visualized and was overtly hypodynamic." An emergency Caesarean section was requested at 1857 hours. Notes dictated by the surgeon indicated that there was a few minutes delay locating appropriate nursing staff as on call staff was not available.

Under general anaesthesia, the woman was delivered by Caesarean section of a stillborn female infant at 2004 hours. Resuscitation was attempted, but was unsuccessful.

Post Mortem

An autopsy was not performed. Pathology of the placenta showed a small amount of marginal and retroplacental hematoma consistent with abruption.

Discussion

This infant was stillborn as a result of placental abruption at 36 weeks gestation.

Delays in getting to the hospital appear to be primarily a function of the rural setting where the patient lived and possibly the weather at the time. It cannot be determined from this review whether earlier transportation to the hospital would have resulted in a more positive outcome. However, upon being notified of the vaginal bleeding, and realizing the environmental challenges of the family transporting the woman to hospital, the midwife should have immediately contacted the local Emergency Medical Services (EMS).

Midwives in Ontario often utilize the services of attendants and/or support persons who have been trained and pre-approved for participation in this alternate practice arrangement model. In this case, the role of the support helper/attendant was to provide assistance to the registered midwife for the birth. Ontario College of Midwives policy does not allow support people to attend a woman in labour without a registered midwife present. It was not the appropriate course of action for the midwife to contact/deploy a support person who was not a registered health care provider for a patient that was G₁₁P₆ at less than 36 weeks gestation, with frank bleeding and a history of prenatal bleeding and prior retained placentae, who was in labour.

While the delay in locating operating room staff at the receiving hospital likely did not play a role in the outcome of this case, it is possible that if the EMS had been contacted, the woman would have been transported to a hospital with more appropriate and available emergency obstetrical services.

Recommendations

1. Obstetrical care providers are reminded that when a woman or baby experiences an obstetrical crisis in a home setting, Emergency Medical Services (EMS) should be immediately contacted for transportation to an appropriate

health care facility providing emergency obstetrical services.

Case: 2011-S-4
OCC File: 2008-13842

History

The mother was a G₂P₁ with no documented prenatal care. The father stated that there had been routine prenatal screening and he was not aware of any difficulties. There was no history of alcohol or drug abuse. The mother was a recent Chinese immigrant and spoke little English.

At an apparent 8 months gestation, she had several days of abdominal pain. There was no vaginal bleeding or fluid loss. The father called EMS when he saw the baby's head coming out. When EMS arrived, the baby had delivered and was still attached to the umbilical cord. The placenta had not delivered. The baby was cyanotic and obviously dead. No resuscitation took place. The baby was taken to the children's hospital and pronounced dead. The mother was taken to another hospital for delivery of the placenta and postpartum care.

Post mortem

The baby was a female infant weighing 1230 g. There was evidence of maceration.

There were congenital anomalies consistent with Trisomy 18. Subsequent cytogenetic studies confirmed 47, XX +18 karyotype.

Discussion

This infant was stillborn at home. Post mortem studies confirmed Trisomy 18 which is associated with a high risk of perinatal mortality. The presence of maceration was consistent with death having occurred in utero at least a couple of days before delivery.

The investigating coroner attempted to ascertain whether the mother had received any prenatal care. No record of prenatal care could be found.

Recommendation

1. Immigration Canada and Health Canada should review their policies regarding resources, timely access to, and funding for, prenatal care for women who immigrate to Canada.

Case: 2011-S-5
OCC File: 2008-442

History

The mother of the deceased was a 37-year-old G₂P₁ with an EDD of June 22, 2008. Her past history included the spontaneous vaginal delivery of a 7 pound male infant at term after an uncomplicated pregnancy in 1996. Her health history was otherwise unremarkable. Her prenatal blood tests were normal and her blood type was B negative without antibodies. A second trimester ultrasound scan revealed an "echogenic focus" in the heart. Subsequent fetal echo was done, however there was no report included with the documentation provided for review.

The prenatal 1 and 2 were completed. The last recorded documentation in the prenatal record that was available on the chart showed appropriate following and a normally developing pregnancy up until mid December 2007 at approximately 34 weeks gestation. The patient was admitted on the afternoon of January 21, 2008 at 39 6/7 weeks gestation in active labour and was 9 cm dilated with full effacement. Nursing notes indicate that they "requested strip review for prolonged variable decels." The patient was admitted at 1315 hours and already had an urge to push. At the time of admission, there were repeated deep and prolonged decelerations for a period of at least 20 minutes. This improved for short periods of time over the next 2.5 hours. However, by 1530 hours, the fetal heart persisted with a very concerning non reassuring pattern. A scalp electrode was attempted at 1615 hours, but was not effective. An ultrasound was used at 1643 hours to confirm bradycardia and a decision was made for an operative delivery.

A low segment Caesarean section was performed

under epidural anesthesia. A male infant was delivered with the cord tied around the neck and loosely around the trunk at 1707 hours. The remainder of the procedure was unremarkable.

Arterial cord gases taken at delivery revealed a PH of 6.92 and a base deficit of 20.5. Venous blood was not reported. The Apgars were 0 at one, five and ten minutes.

The pediatric staff was available at delivery. At birth, the baby was “flat - no spontaneous respirations - no heart rate.” They proceeded with intubation, cardiac compression, and epinephrine by endotracheal tube (ETT) with no response. As the pupils were fixed and dilated, the resuscitation was stopped at 1725 hours.

Post mortem

This term baby weighed 3212 g. The summary of findings included: intrauterine asphyxia with congestion and edema of brain; petechial hemorrhages of the visceral pleura and thymus; minimal pleural and peritoneal effusions; and congestion of internal organs, especially kidneys, liver and spleen. A term mature placenta revealed a placental infarction and the description of the placental parenchyma indicates, “marginal pale firm area, 3 X 3.5 X 2 cm, which is small compared to the overall size of 17 X 14 X 3.5 cm. All cultures of the fetus and placenta were negative.”

Discussion

This pregnancy was normal until the admission of the mother to hospital at term. Continuous external monitoring from the time of admission revealed a non-reassuring fetal heart rate. This reverted to a normal pattern for periods of 20, 30 and 25 minutes over the first 2.5 hours. However, from approximately 1530 hours until 1643 hours, at which time a decision was made to proceed with Caesarean section, the fetal heart tracing was abnormal and became increasingly concerning. From 1610 hours, there was no continuous tracing as the care team attempted to find the fetal heart.

There appears to have been a delay in making a

decision for an operative delivery. The abnormal tracing at the time of admission should have given the care team a high index of suspicion of potential problems. These initial and repeated episodes of abnormal heart beat should have led to an earlier delivery.

The arterial cord gases, the state of the baby at delivery and pathological findings are all compatible with intrauterine asphyxia occurring during the process of labour.

The Regional Supervising Coroner has been advised by the hospital involved that the following actions have been taken to address issues related to the care of this baby and his mother:

- An external review of the obstetrical care provided by the delivering obstetrician has taken place. The review was conducted by an obstetrician who regularly undertakes such reviews for the College of Physicians and Surgeons. The recommendations arising from this review were utilized to enhance the overall performance of the obstetrical care provider;
- The hospital has implemented a second-on-call obstetrical coverage model to provide backup in situations of unanticipated high volume, when there are multiple high-risk situations occurring simultaneously, or when the first-on call obstetrician requires specialist assistance;
- The hospital has implemented a double-on-call anesthesiology coverage model that enhances coverage to Labour and Delivery at all times, but is particularly important during evenings and on weekends. The model is designed to improve urgent access to an anesthesiologist for obstetrical care even if a case is already underway in the Operating Room.

Recommendations

1. All obstetrical care providers are reminded of [Fetal Health Surveillance in Labour Guidelines as per SOGC Guidelines September 2007](#).

History

The mother of the stillborn was a 32-year-old G₂P₁ with an EDD of February 8, 2011. She had a pre-pregnant BMI of 27.5. She gained 29 pounds during the course of the pregnancy. Her past medical history included a cyst removal (2006) and wisdom teeth extraction (1994), both under general anesthetic with no complications. Her family history indicated that her father had Type II diabetes. In 2009, she delivered a live male at 39 weeks weighing 8lb 9oz after 6 hours of active labour. She experienced spontaneous rupture of membranes (SRM) followed by spontaneous labour and her delivery was vacuum assisted. She had an episiotomy that healed well. She reported breastfeeding for 13 months.

She was a repeat client to the midwives and was planning a hospital birth. She declined all genetic screening prenatally and was blood type A negative with no antibodies at intake. She received Rh immune globulin at 27 weeks 6 days. Her haemoglobin and mean cell volume (MCV) were 127g/L and 88.1fL, respectively, at intake and haemoglobin was 124 g/L at 27 plus weeks. Her platelets were normal. An oral glucose challenge test was normal at 6.0 mmol/L. There was normal urinalysis and negative urine culture at intake and 28 weeks. She was HIV, Hep B and Syphilis negative and rubella immune.

Ultrasound at 19 weeks 6 days showed normal fetal morphology, posterior placenta clear of 3.9 cm long cervix. Her antenatal visit schedule was appropriate and she was seen at 13, 18, 21 weeks 6 days, 29 weeks 6 days and 32 weeks 2 days. Her fundal heights were as expected for her number of weeks and her blood pressure was normal throughout. At the 32 week 2 day visit, she presented with a report of vomiting throughout the night and having had reduced fetal movement over the last day. On auscultation, the midwife was unable to detect a fetal heart rate and the mother was sent immediately to the Level I hospital triage for

assessment by the on call Registered Midwife and an ultra sound.

She arrived at the hospital and was assessed at approximately 1100 hours on December 16, 2010. When no fetal heart was detected, an ultrasound was performed. The report was consistent with a fetal demise in the 3rd trimester. Gestational age was assessed at 31 weeks with evidence of abdominal swelling and mild overlap of cranial bones and no comment on the placenta or fluids. A physician was consulted and care was transferred. Routine labs for stillbirth, including complete blood count, group & screen, haemoglobin, ALC and Kleihauer-Betke tests, were ordered and collected. Misoprostol 200 mcg per vagina every three hours was ordered and at 1130 hours, the first dose was administered. The GBS status was unknown.

Although the patient settled into her room at 1530 hours, she found it difficult to rest, so she requested permission to return home with misoprostol to be self administered. At 1830 hours, laboratory results were received that indicated a massive maternal fetal bleed and orders for Rh immune globulin for IV administration were received. Nubain 20 mg intramuscularly at 1900 hours was followed by an epidural for pain relief.

At 2030 hours, she was being administered the 1000 mcg IV of Rh immune globulin following an informed choice discussion and consent regarding the risks and benefits of the isoimmunisation status and treatment. At this point, the physician performed an artificial rupture of membranes for meconium stained fluids. Her cervix was 3 cm dilated, 50% effaced and the fetus was at -1 spines. Due to the meconium, maternal fever and her history of gastrointestinal upset, the physician screened for listeriosis and treated the patient with ampicillin 2 g intravenously. At 2145 hours, the patient commenced pushing and at 2152 hours an extremely pale stillborn male infant was delivered weighing 2065 g. The male infant was 42 cm long with head circumference of 31 cm. There was swelling around the joints and neck areas and peeling on his extremities with meconium stained lips.

The placenta was delivered with controlled cord traction and was noted to be grossly edematous. She was given Gentamycin 120 mg intravenously, followed by 80 mg intravenously every eight hours until her temperature was normal. She experienced a first degree tear that was repaired and a postpartum hemorrhage of greater than 800 ml that was treated with oxytocin intravenous infusion, ergonovine 0.25 mg intramuscularly, fundal massage and a Foley catheter was placed. Following this bleed, her Gentamycin was changed to 300 mg every 24 hours intravenously. She continued to be treated in accordance with the laboratory protocol with Rh immune globulin for the isoimmunisation due to fetal maternal bleed, 1000 mcg intravenously every 8 hours, to a total of 6000 mcg and to discontinue only if stillborn was RH negative (which it was not).

Midwives provided care after delivery in hospital and after discharge in the woman's home at day 1, 2 and 4 and made phone contact at two weeks when a home visit and discharge were planned and completed.

Post mortem

No post-mortem examination was done.

Placenta was 371 g, 14 cm in diameter, pale grey fetal surface and maternal surface pale tan. 40 cm cord thick and edematous with a central non membranous insertion and 3 vessels. Negative cultures reported from cord and placenta.

The report indicated, "There is no evidence of any etiological causes in this placenta."

Recommendations

No recommendations

Case: 2011-S-7
OCC File: 2010-12039

Antenatal Course

The mother was a 22-year-old primiparous woman who had an uncomplicated pregnancy. She received appropriate antenatal care. She had a normal

Maternal Serum Screen, dating and morphology ultrasound. She had no risk factors from her medical or family history. Her GBS screen was positive.

Labour onset was at approximately 0100 hours on September 25, 2010. She was admitted to the labour birthing room at 0431 hours. Her temperature, blood pressure and admitting blood work were normal. She received intravenous penicillin G 5 million units. The cervix was 8 cm dilated. At 0500 hours, she had the urge to push and was 9 cm dilated. A low fetal heart rate (FHR) baseline was noted. Her membranes were ruptured at 0520 hours by the obstetrician. The amniotic fluid was clear. She continued to progress quickly and had a spontaneous vaginal delivery at 0624 hours.

The baby was unexpectedly flat at delivery. Apgars of 1, 0, and 0 were recorded at one, five and ten minutes. There was evidence of a loose nuchal cord. There was no meconium staining of the amniotic fluid. A Code Pink was called, but they were unable to resuscitate the baby and could not obtain arterial cord gases. Venous cord gases showed a pH of 6.8, with a pCO₂ of 65mmHg and a pO₂ of 8mmHg. At the time of delivery, there was one or two skin blisters present on the body. The placenta delivered spontaneously with no obvious abnormalities. The stillborn infant weighed 3245 grams.

Review of FHR Tracing

There was a continuous fetal heart rate recording from approximately 0430 hours until delivery. The baseline ranged from 110 to 120 bpm. There was average variability and accelerations were present. The maternal heart rate was recorded on the partogram at 0530 hours as 98 bpm and the EFM was 130 bpm. At 0545 hours, the maternal heart rate was 84 bpm and the EFM was 115 bpm. There was no simultaneous recording of maternal and fetal heart rate on the EFM tracing.

Post Mortem

Post mortem examination revealed a male stillborn that was appropriately grown for 38 weeks gestation. There was evidence of skin maceration and internal organ autolysis. Lung bacteriology was

positive for yeast and multiple bacterial organisms. Placenta showed acute chorioamnionitis and acute funisitis with evidence of Candida infection. The pathologist concluded that the fetal death occurred 1-2 days prior to delivery.

The cause of death was attributed to Candida amniotic infection.

Discussion

This case was referred to the Maternal and Perinatal Death Review Committee because of the discrepancies in the clinical assessment of fetal wellbeing in labour and the pathologic report of an antenatal demise. There were no maternal concerns about fetal wellbeing on presentation to the labour and delivery room. There were no antenatal or intrapartum risks factors. Fetal wellbeing was assessed with a continuous external FHR monitor. The nurses and physician were aware of the low baseline. They believed they were recording the fetal heart rate and in review of the tracing, it has features of a normal intrapartum recording. The partogram records the maternal heart rate on two occasions significantly lower than the recorded electronic heart rate recording.

Despite all these reassuring signs of fetal wellbeing, the pathology concludes the demise occurred days prior to the delivery. This discrepancy cannot be resolved with certainty by this review, however it suggests the presumed electronic recording of the fetal heart rate was maternal.

The cause of death reported in the autopsy was reviewed. Vaginal candidiasis is very common in pregnancy and yet Candida amniotic infection is extremely rare. There are only a few case reports in the world literature, and most of these cases had risk factors such as a coincident IUD or premature rupture of the membranes.

Recommendation:

1. [Obstetrical care providers are reminded of the importance of differentiating fetal and maternal heart rates, particularly in cases with a low fetal heart rate baseline or bradycardia.](#)

[In cases where this is difficult, the simultaneous recording of the maternal heart rate with pulse oxymetry or the placement of a fetal scalp clip will help confirm monitoring of the fetal heart rate.](#)

Case: 2011-S-8
OCC File: 2010-6518

Summary

The mother of this stillborn was a 29-year-old T₁P₀A₁L₂ with Type 1 diabetes mellitus. Her obstetrical history included a previous Caesarean section for twin pregnancy.

Medical History

The mother developed Type 1 diabetes following the birth of her twins seven years prior. She had no other medical problems. She was followed by an endocrinologist, however she was not always compliant with treatment recommendations. She was admitted to hospital in 2004 and 2009 with diabetic ketoacidosis (DKA). She was only testing her blood sugar once or twice a day. She had been given a prescription for folic acid pre-conceptually but did not start this. She was noted to smoke and to use alcohol socially.

Antenatal Course

The pregnancy was identified early in the first trimester. She was seen in the diabetes education centre at six weeks gestation. Her hemoglobin A1C was 11.4%. She was well counseled in the diabetes education centre about the importance of glucose control and frequent testing. She had a normal morphology ultrasound. She declined an integrated prenatal screen (IPS). It is uncertain if she had a fetal echocardiogram.

She was followed by a dietician, her endocrinologist and by an obstetrician in the high risk clinic at Hospital A from nine weeks until the last recorded visit at 28 weeks 3 days. On that visit, her blood sugars were "great." Her good blood sugar control on May 14 was confirmed by the endocrinologist.

Course in hospital

The mother was seen in obstetrical triage on May 15, 2010 at 1700 hours. The electronic triage record has minimal documentation, but indicated the reason for the visit was abdominal pain. She was discharged approximately one hour later with instructions to follow up in the high risk clinic as scheduled or to return if she had further concerns. She returned to the emergency department at Hospital A just after midnight and was assessed in the early hours of May 16, 2010. The admission history indicated that she was having emesis over the day and was unable to keep anything down. She did not respond to dimenhydrinate and ondansetron in the emergency department. She was hydrated with two litres of normal saline. Her blood sugar at 0330 hours was 6.4 mMol/L. Her admission urinalysis did show ketones. Her electrolytes were normal, including her total CO₂ (bicarbonate) of 23 mMol/L. As she was unresponsive to antiemetic therapy, she was admitted to hospital.

She was initially allowed to manage her own blood sugars, but endocrinology was subsequently consulted and followed her during her admission. When she was seen by endocrinology on May 17, she was thought to be in mild DKA. On May 18, she was continuing to have emesis and developed some shoulder tip pain. She had an ultrasound which did not show any evidence of biliary tract disease. The appendix was not well seen. On May 18, her acidosis worsened and she was started on intravenous D5W and an insulin infusion with a dosage sliding scale. The fetus was to be monitored twice daily with a non stress test while her DKA was being treated.

Following her admission, she developed progressive diabetic ketoacidosis. Her glucose readings were labile, ranging from 7 – 20 mMol/L. On May 16 at 1135 hours, her bicarbonate was 18 Mmol/L, and serum ketones were positive. On May 17 at 2300 hours, her bicarbonate was 18 Mmol/L. On May 18 at 1235 hours, her bicarbonate was 15 Mmol/L and her serum ketones were positive. A verbal alert of critical results was sent to the ward at 1305 hours. At 2230 hours, her bicarbonate was 11 Mmol/L. The alert of critical results was sent to the ward at 2301 hours. On May 19 at 0700 hours, her bicarbonate

was less than 5 and an alert of critical results was sent at 0859 hours.

Just prior to 1200 hours, she was requesting oxygen. The obstetrician in hospital was paged and assessed her at 1250 hours. She was hyperventilating and looking quite unwell. She was contracting every two minutes. The fetal heart rate strip placed by the nurses was shown to the obstetrician and the heart rate was in the 120s. She was taken to labour and delivery and the “RACE” (Rapid Assessment of Critical Event) team was paged. In the delivery room, they were unable to detect a fetal heart. A bedside ultrasound showed a heart rate of 50 beats per minute. She was taken to the operating room for an emergency Caesarean section.

At the time of birth at 1332 hours, the infant was unresponsive with no respiratory effort. Apgars were 0 at one, five, ten and fifteen minutes. The resuscitative efforts were terminated at 16 minutes.

The mother was transferred to the intensive care unit (ICU) post operatively with severe ketoacidosis. Her initial blood gases showed an arterial pH of 6.8. In the ICU, her severe metabolic acidosis was slowly corrected and she made an uneventful recovery from her emergency Caesarean section.

Post Mortem Report

The post mortem revealed a 29 week gestation, premature infant, with a hypoplastic aortic arch. The weight was 1420 grams which is above the 50th percentile. The placental weight was 199 grams which is less than the 10th percentile. The cause of death was thought to be perinatal asphyxia with complications of maternal diabetic ketoacidosis.

Expert Consultation

The MPDRC sought input from an external specialist in endocrinology for further assessment. After a thorough review, the specialist’s opinion was that the mother’s medical and obstetrical care was appropriate overall. The specialist did, however, provide the following comments and suggestions for consideration by the hospital involved:

- a) *“Accurate fluid balance ins/outs may have helped the physicians note when IV fluids needed alteration – her DKA worsened or even developed while in hospital and DKA can do so if dehydration is not corrected. Therefore, the Attends were not ideal for her; rather a catheter may have been a better choice/negotiation.*
- b) *Intravenous insulin could have been started on admission and may have helped avoid the subsequent deterioration in her metabolic balance. Further, for someone who is nauseated and vomiting, it would not be ideal for her to have to judge her own insulin dose, and she was left to do this for herself for at least one day.*
- c) *A chart flow sheet for DKA treatment may have helped all concerned to more clearly note the progression/improvement of her condition. This should include BG, lytes, bicarb, anion gap as well as IV fluid rates, K administration, insulin drip rates, BP, etc.*
- d) *The bedside charts of BG results indicate that the BGs were lab values, but there is no corroboration in the lab test section, so I suspect these were capillary, bedside values. As a rule, only lab values should be used to monitor DKA, due to inherent errors in reading with dehydration as well as the errors that can occur at upper and lower end of readings.*
- e) *Routine gases, lytes, bicarb, BGs would also have helped her physicians better document her progress, and could have been more helpful than ordering these on a prn basis.*
- f) *The fluid balance sheets seem to show a 10 hour window on May 18 when the patient was receiving negligible fluid by IV. This situation may have worsened her hydration status and furthered the DKA progression.”*

Summary and Discussion

The mother was admitted to hospital at 28 weeks gestation with severe nausea, vomiting and mild DKA. Despite medical management, she developed worsening acidosis. She had an emergency operative delivery and the baby did not respond to resuscitative efforts. She was admitted to the ICU with severe DKA. Other than the emesis, no precipitating cause for the DKA was identified.

Recommendations

1. Obstetrical care providers are reminded that diabetic ketoacidosis is associated with fetal and maternal morbidity and mortality and should be treated aggressively.
2. Hospital “A” should review or develop the protocol for management of diabetic ketoacidosis.
3. Hospital “A” should review the response to, and documentation of, critical laboratory results.

Case: 2011-S-9

OCC File: 2010-10638

Antenatal History

The mother of the deceased was a 30-year-old primigravida patient with an EDD of August 20, 2010 after regular predictable menses. The review of the prenatal records revealed that she had an unremarkable past medical history. She was allergic to penicillin and would break out in a rash when exposed. She was followed in prenatal care from early in the first trimester. Records indicated normal prenatal course. Fetal activity was documented as normal throughout the third trimester and there were no expressed concerns.

Investigations included ultrasounds at 8, 12 and 20 weeks. There was normal nuchal translucency at 12 weeks and a normal anatomic scan at 20 weeks. Integrated prenatal screening testing was not done. All the other routine prenatal blood testing was normal.

She presented to triage at Hospital A on the early morning of August 19, 2010 at 39 weeks and 6 days gestation. She had been having contractions throughout most of the previous day, becoming more regular over the last three hours. She reported that fetal movement had decreased in the last three days.

On admission, her vital signs were normal. Pelvic examination revealed a cervix that was posterior, thick and fingertip dilated with vertex above spines.

A continuous external monitor was placed. There was moderate variability, but infrequent fetal heart acceleration. The attending physician was contacted at 0330 hours and again at 0500 hours and was asked to attend. The patient was re-examined and still was not dilated, but was partially effaced. A bedside ultrasound was done as the external monitor was "barely reactive, but good variability." There was decreased amniotic fluid and one pocket measured approximately 2 X 2 cm. The patient was admitted at 0600 hours and given morphine 10 mg and dimenhydrinate 50 mg as she was feeling uncomfortable.

At 0750 hours, intravenous cefazolin was started for Streptococcus prophylaxis. Continuous electronic monitoring was continued. The fetal heart rate remained in the normal range, with variability indicated to be from minimum to moderate. There were intermittent short variable decelerations.

At 0910 hours, examination revealed no change and oxytocin was started to augment labour. At 1000 hours, a second dose of morphine 10 mg along with dimenhydrinate 50 mg, was given for analgesia. Epidural was placed shortly after 1200 hours with analgesic effect.

At 1320 hours, examination by the attending physician revealed the cervix to be 3 cm dilated with the presenting part still above spines. An artificial rupture of membranes revealed thick meconium; a fetal scalp clip was applied. At that time, the fetal heart tracing was described as having a "baseline of 110 to 120's with occasional small variables and normal variability." The plan was to continue with the course of treatment and have pediatrics available at delivery.

At approximately 1330 hours, the low dose oxytocin was stopped due to increased resting tone and prolonged contractions. It was restarted at 2 milliunits per minute at 1405 hours. Continuous fetal tracing continued until approximately 1453 hours when there was a prolonged deceleration for approximately eight minutes with a fetal heart rate of 40 to 60 bpm. Appropriate resuscitation measures were used including a change of position, stopping oxytocin and the provision of IV fluids and

oxygen. The attending physician was called. As there was no significant change on the cervical examination, Caesarean section was recommended at 1456 hours and the patient was transferred from the labour room to the operating room at 1501 hours.

The patient and nurse were documented as being in the operating room at 1505 hours and the anesthetist was present at that time. The epidural was topped up for analgesia and the procedure started at 1526 hours with delivery at 1535 hours. The operative note indicates that the patient was hooked up to the monitor on arrival to the operating room and at that point, the fetal heart was 130 bpm with average variability. From the tracing available for this review, it is unclear whether the reading represented the fetal or maternal heart rate. Initial vital signs by the anesthetist revealed a maternal pulse of 130 bpm. The staff indicated to the father (who was present for the procedure), that both mother and baby were doing well.

A stillborn male weighing with Apgars of 0 and 0 was delivered. There were no difficulties with the delivery. The cord was found wrapped around the neck five times. Cord gases were taken and revealed an arterial pH of 7.03 with base deficit of 18 and a venous pH of 7.09 with base deficit of 14.

The baby was "extremely limp and covered in meconium with no heart rate, spontaneous respirations or tone." Intubation was initiated immediately with a meconium aspirator and the tube became a third full with thick meconium. The tube was removed, bag and mask was used, and then reintubation was performed. Compressions were started shortly after the delivery and several doses of epinephrine were given. After approximately 20 minutes, there was still no heart rate detectable, no response to bagging or epinephrine and the pediatrician and a colleague who had come to assist recommended resuscitation be stopped.

Post Mortem

At post mortem examination, this stillborn male weighed 3500 grams. There were normal growth

parameters and organ weights. No intra-uterine growth restriction (IUGR), anomalies, infection or disease were found. Lung sections showed small amounts of pigmented material in the large airways suggestive of meconium. However, there was no evidence of reactive pneumonitis.

The placenta showed a green discoloration of the membranes, but otherwise was unremarkable. There were no meconium-stained macrophages within the fetal membranes to suggest long standing meconium exposure. There was only 10 cm of cord available for examination. An intrapartum cord accident such as a true knot could not be excluded as it was not available for assessment. There was however, no suggestion in the operative notes that this was the case. Brain examination was unremarkable. Cultures for virus and bacteria were negative.

Discussion

The mother of the deceased had a normal course of her pregnancy up until the time when she was admitted to hospital at term. There was reported decreased fetal movement over the previous three days. She was monitored continuously from the time of arrival and subsequently admitted, though she was not in active labour, due to lack of fetal heart accelerations and a finding of decreased amniotic fluid on ultrasound. She was appropriately given Streptococcal prophylaxis. Narcotic (morphine) was given on two occasions for analgesia and this is an appropriate practice particularly in a patient who is not yet in active labour. The last dose was given approximately five hours prior to delivery and should not have had any affect on the outcome. The fetal heart rate during this time, though not showing accelerations, did not indicate any concerning decelerations apart from intermittent variable decelerations.

A sudden prolonged and persistent fetal bradycardia occurred at 1453 hours. The physician attended quickly, appropriate resuscitation measures were attempted and a decision was made to proceed with a Caesarean section. Further heart rate monitoring revealed a heart rate of approximately 130 bpm, though it is unclear whether this was a maternal or

fetal reading. The first maternal heart rate taken by the anesthetist in the operating room was 130 bpm. It is possible that the last recorded true fetal heart rate was during the persistent bradycardia while still in the labour room and that there was no recording of the fetal heart rate until the time of delivery approximately 40 minutes after the initial bradycardia occurred.

As it was presumed that the fetal heart rate had recovered, the Caesarean was not expedited. The epidural was topped up and subsequent delivery was not until 1535 hours.

The baby was born with Apgars of 0 and 0. The cord gases revealed a pH above 7. It is possible that this was not representative of the fetal PH when finally delivered due to the multiple loops of cord which may have been under compression and more representative of the fetal PH closer to the initial deceleration.

The neonatal staff was available at delivery and made appropriate attempts at resuscitation. There was evidence of meconium in the endotracheal tube and this may have contributed to the difficulty in resuscitating the infant. As noted, if the true pH of the fetus was lower than that in the cord sample, that may have contributed to the unsuccessful resuscitation.

The family of the stillborn infant indicated several concerns pertaining to the presence of meconium and the cord around the neck, the fetal heart rate at the time of delivery and the use of morphine for analgesia.

In response to these concerns, the Committee has found:

1. The presence of meconium at artificial rupture of membranes (ARM), though affecting the plan for delivery to have pediatrics available, would not in this case have indicated a change in the course of action that was followed by the staff. It is possible that because the baby was stressed shortly prior to delivery, that some meconium aspiration did occur which may have made resuscitation more difficult.

2. Morphine is commonly used as an obstetrical analgesia when a patient is not in active labour, though needing analgesia prior to the institution of an epidural. The mother of this stillborn was appropriately administered morphine as she was still not in active labour at the time. The epidural was subsequently administered at an appropriate time early in the labour.
3. The family of the stillborn questioned whether the cord around the neck could have been diagnosed prior to delivery through the use of ultrasound or colour Doppler. While colour Doppler may have shown multiple loops of cord around the neck, it is not a standard practice in labour and delivery. Without significant decelerations occurring in the fetal heart tracing, it is unlikely that this information would have affected subsequent care. The loops of cord may have contributed to the fetal stress and sudden onset of fetal bradycardia.
4. It is possible that the fetal heart monitor was recording the maternal pulse immediately before the Caesarean delivery. However, the tracings prior to this were clearly fetal, given the evidence of the cord pH above 7 and the tracing prior to the final significant bradycardia.

Recommendations:

1. [Obstetrical care providers are reminded that concurrent monitoring of maternal and fetal heart rate is essential when there is maternal tachycardia and/or a small difference between the rates.](#)

Case: 2011-S-10
OCC File: 2010-16223

Antenatal History

The mother of this stillborn child was a 35-year-old G₂P₁ with an EDD of December 17, 2010. She was referred for genetic counseling for maternal age at 12 weeks gestation. After counseling, she elected to have Integrated Prenatal Screening (IPS) done. The

IPS was positive for Down syndrome, but she declined anything further be done.

On October 21, 2010 at 31 weeks gestation, she was diagnosed with gestational diabetes and was referred to the Diabetic Education Centre. Her blood sugars were controlled by diet alone.

The antenatal course was otherwise uneventful. Her past medical history was non-contributory and more specifically, there was no history of any uterine instrumentation.

Her past obstetrical history included one previous term pregnancy delivered vaginally for a 7 pound, 5 ounce female infant after an eight hour labour in 2005.

She was 5 feet tall and her pre-pregnancy weight was 175 pounds (BMI=34).

Course in Labour and Delivery

Labour began at 0600 hours on December 15 at 39 weeks 5 days gestation. She was assessed in triage at 0955 hours and her cervix was 6 cm dilated with bulging membranes and station spines -2. She was admitted to the hospital delivery suite under the care of her family doctor at 1125 hours. At 1140 hours, she was assessed and her cervix was 7cm dilated. Artificial rupture of membranes was carried out for clear fluid. On reassessment at 1203 hours, the cervix was 8 cm dilated with the vertex at spines -2. Subsequent assessment by a different observer at 1225 hours found the cervix to be 5-6 cm dilated.

An epidural was placed at 1422 hours.

Because of the slow progress, an obstetrical consultation was obtained. The obstetrician's assessment at 1853 hours showed the cervix to be 6 cm dilated with the vertex at spine -2, not well-applied. At the time, with contractions every 2-3 minutes lasting 30-50 seconds, oxytocin augmentation was ordered. At 2000 hours, the cervix was 7cm dilated and the vertex at spines +1.

At 2100 hours, variable decelerations occurred and contractions were every 1-3 minutes. The oxytocin

infusion was discontinued and was not restarted. The obstetrician was paged at 2138 hours because of the variable decelerations. At 2200 hours, the patient complained of pressure pain. The cervix was fully dilated with the vertex at spines -2. The obstetrician and family doctor were called. At 2225 hours a fetal bradycardia occurred. The obstetrician was in attendance. Full dilation was confirmed, but the presenting part was too high for a forceps delivery. A stat Caesarean section was called. The operating room was busy, so a second team was called in. A Caesarean section was performed under epidural for a female infant weighing 7 pounds, 9 ounces. The fetus was found in the peritoneal cavity and the uterus had ruptured transversely along the lower uterine segment. The baby was handed off to the paediatrician who was in attendance. Apgars were 0 and 0 at one and five minutes. A "Code Pink" was called. Resuscitation was discontinued after 35 minutes with no response.

Post Mortem

Autopsy revealed an appropriately grown female stillborn with no congenital anomalies. There was generalized organ congestion.

The placenta was not available for pathological examination.

The cause of death was intrapartum uterine rupture.

Discussion

Rupture of an unscarred uterus is a rare event and is estimated to occur in 1/5,700 to 1/20,000 pregnancies. The only identifiable risk factor in this case is protracted labour and the use of oxytocin. Manifestations of uterine rupture are variable. The most common fetal manifestation is bradycardia which may be preceded by variable or late decelerations, but no pattern is pathognomonic. Maternal manifestations include abdominal pain and signs of intra-abdominal bleeding although signs and symptoms of the latter may be subtle.

In this case, progress in labour was slow. This was identified several hours after admission with no change in cervical dilation and an obstetrical

consultation was obtained. The obstetrical consultation note does not identify any findings to explain the slow progress. Specifically, there is no mention of the position or attitude of the presenting part or an estimate of the fetal weight. Contractions at the time were every 2-3 minutes lasting 30-50 seconds and described as moderate in intensity with normal resting tone. It is not clear from the record whether this assessment was from palpation or the external tocodynamometer. Given the description of the patient's body habitus, it is likely that accurate assessment would be difficult using either approach. It appears that the dystocia was attributed to inadequate contractions and based on this assessment, oxytocin augmentation was ordered. An intrauterine pressure catheter should have been considered to accurately assess uterine contractions before augmenting the labour.

The oxytocin augmentation was appropriately discontinued with the onset of an atypical heart rate pattern. It does not appear that the contraction pattern changed significantly despite this. When the fetal bradycardia occurred, the operating room staff was busy with another case. The second team responded in a timely fashion, but inevitably there was a delay in delivery. With uterine rupture and expulsion of the fetus, the outcome may not have been any different if the first team had been immediately available.

Recommendations

1. [Obstetrical care providers are reminded to assess and consider the possible underlying causes for dystocia.](#)
2. [Obstetrical care providers are reminded of the utility of an intrauterine pressure catheter in accurately assessing the quality of uterine contractions, particularly in patients with an increased BMI.](#)

Case: 2011-S-11
OCC File: 2010-14960

Case History

The mother of this stillborn child was a 23-year-old primagravida with an EDD of December 1, 2010 as confirmed by first trimester ultrasound scan at 9 weeks gestation. Her past medical history was unremarkable. She was overweight at 215 pounds at 12 weeks gestation.

Routine prenatal testing was normal. She had integrated prenatal screening which showed low risk of Down syndrome. A normal second trimester ultrasound scan was done at approximately 19 weeks gestation. She was sickle cell trait positive.

Gestational glucose screening was done at 27 weeks gestation and a one hour screen was positive. She was referred to the Diabetes and Pregnancy Program at Hospital A and she attended an informational session on September 21, 2010. She started, and was provided with instruction for, self insulin administration on that date. She was followed closely by the clinic staff for the duration of the pregnancy. Her fasting sugars normalized and readings ranged from 5 to 13 mMol/L, the highest prenatal post prandial dinner. On her last clinic visit at 37 weeks gestation, she was on a total of 64 units of rapid acting insulin and 20 units of NPH long acting insulin daily.

Her last biophysical profile was performed on November 8, 2010 at 36 weeks and 5 days gestation. The biophysical score was 8/8 with an estimated fetal weight of 3939 grams at the 88th percentile.

She was admitted for induction of labour at 38 weeks gestation on November 18, 2010 due to suboptimal control of gestational diabetes.

The patient's cervix was unfavorable as it was posterior, thick and closed. She was given 2 mg of vaginal prostaglandin gel. A second 1 mg prostaglandin gel was given approximately eight hours later as there was insignificant change of her cervix. By 0630 hours on November 19, 2010 - 14 hours after the first gel - an epidural was placed due

to painful contractions. By 0640 hours, the attending staff and residents were asked to assist as the fetal heart was difficult to trace. At the time, the cervix was 3 cm dilated and fully effaced with presenting part at spines -2. After an artificial rupture of membranes (ARM), fluid was meconium stained. A Fetal Scalp Electrode (FSE) was applied. By 1235 hours, on assessment of the cervix, there was insignificant change since the ARM. A Caesarean section was recommended.

The nursing notes pertaining to monitoring were completed throughout labour. A non-stress test (NST) had been done prior to the initial gel and had no accelerations. A repeat NST two hours after the first gel showed no accelerations and was reported as non-reassuring. On a number of occasions during the labour, variable decelerations were documented, but no concern was expressed.

On reviewing the fetal heart strips from the time just prior to the time that the first gel had been administered, there was poor variability. A heart rate at that time was 150 to 160 bpm. From about four hours after the gel, there became more frequent subtle depressions of the baseline which always occurred after the contractions. At around 0330 hours, there was some concern expressed about difficulty in the monitoring and picking up of the maternal heart, so the monitor was adjusted. At that time, the baseline was in the 120 to 130 bpm range. By 0630 hours, when the FSE was placed after ARM, the heart rate being monitored was in the 110 to 120 bpm range. The pattern remained in the 120 to 130 bpm range over the next several hours until delivery.

A low segment Caesarean section was done under epidural anesthesia due to arrest of labour. A stillborn male with Apgars of 0 was delivered at 1330 hours. The cord blood was clotted.

The placenta was intact and was sent to pathology for examination. The stillborn was documented as being macerated with the skin peeling in large sheets and the abdomen was distended. Notes indicated that the fetal heart (by the fetal scalp electrode) was 110 bpm prior to the Caesarean section and the maternal heart rate was 108 bpm at the same time.

The pediatric staff in attendance at delivery assessed the baby as being flaccid, cyanotic, no heart rate or respiratory effort and with abdominal distention. The infant was intubated and positive pressure ventilation (PPV) was started as the infant was felt to have “stiff lungs” and needing high pressure to bag. Epinephrine was given by endotracheal tube (ETT).

At five minutes of age, chest compressions were administered. The chest skin started peeling off and more maceration was seen on the baby’s back and upper arms. The code was called at 1350 hours. There was a full attempt at resuscitation with four doses of epinephrine given by ETT as well as fluid and bicarbonate.

In the attending note documenting discussion with the parents after the procedure, there was disclosure that there was significant evidence that the demise of the infant had occurred long before the delivery as shown by the macerated fetus, no heart rate in the operating room, clotted umbilical blood and the suggestion that the fetal heart rate and maternal rate had likely been the same throughout labour. There was a note of last fetal movement on Wednesday (the day prior to admission), presumably reported by the mother.

Post Mortem

The stillborn male weighed 4030 g. There was extensive skin maceration indicating that death likely occurred at least six to twelve hours prior to delivery. There was significant meconium contaminated amniotic material within the air spaces of the lungs. There were “stigmata intrauterine fetal stress including evidence of meconium release with placental meconium histiocytosis, moderate thymic cortical lymphocyte depletion and cardiac myofiber necrosis with early calcification, all of which indicating a relatively prolonged period of stress prior to demise.”

Apart from the meconium affects, the placenta showed “marginal placental infarct” and changes suggestive of an acute abruption with “retroplacental hematoma with dissection of decidua and focal villus edema.” The brain

examination revealed diffuse vascular congestion and acute hypoxic-ischemic injury.

The pathologist’s opinion on the cause of intrauterine death was intrauterine asphyxia due to placental abruption with potential contributing factors of maternal sickle cell trait and maternal gestational diabetes. There was no suggestion of significant abruption at the time of delivery.

Discussion

This was the mother’s first pregnancy and it was complicated by gestational diabetes diagnosed late in the second trimester and treated with insulin. This was followed closely in the outpatient diabetic clinic. She had improved sugars, though not optimal control. The biophysical profile one week prior to admission showed large for gestation age, but otherwise was unremarkable. She was appropriately admitted at 38 weeks gestation for induction of labour. At that time, she had an unfavorable cervix so prostaglandin gel was used for preparation of the cervix.

From the time prior to administration of the first gel and throughout labour, there was a non-reassuring tracing; there were never accelerations. On many occasions, there were subtle decelerations which came after contractions, though not consistently. There was no charting of discussion about the mother’s sense of fetal movement. The only time fetal movement was documented was after the delivery when there was a suggestion that last movement had been felt on the day prior to admission. Following labour, the recorded heart rate was in the 150-160 bpm range. At the time of ARM and fetal scalp electrode, the recorded heart rate was less than 120 bpm. Monitors do not record maternal vital signs concurrent with fetal heart rate.

This stillborn fetus likely died several hours prior to delivery. It is not clear when exactly the death occurred however; this was not considered by the nursing, resident or attending staff until after the delivery. As fetal demise had already occurred, the Caesarean section was not necessary. The mother did not have any complications from the procedure.

There was a 20 minute full resuscitation effort on this infant immediately following delivery. This resuscitation occurred even though it was documented that there was significant sloughing of the skin and “stiff lungs”.

This case was reviewed by the hospital and recommendations were made.

Recommendations

1. The Regional Supervising Coroner should assess the findings from the internal review and subsequent recommendations made by the hospital from their investigation into the circumstances of this stillbirth.
2. Obstetrical care providers are reminded that concurrent monitoring of maternal and fetal heart rate is essential when there is maternal tachycardia and/or a small difference between the rates.
3. Ultrasound assessment for fetal wellbeing should be considered in situations of persistent non-reassuring fetal heart rate monitoring to get a fuller determination of fetal health.

Case: 2011-S-12

OCC File: 2010-8585

History

The mother of this stillborn infant was a 33-year-old G₁P₀ with an EDD of July 20, 2010. The mother’s past medical history was non-contributory. There was no history of hypertension.

Routine prenatal laboratory investigations were normal. First trimester ultrasound for dating and routine second trimester ultrasound were normal. Genetic screening testing was declined. A second trimester glucose challenge test (GCT) was 9.5 and a two hour oral glucose tolerance test (OGTT) was normal. She was GBS positive.

On her first prenatal visit with her obstetrician at 29 weeks gestation, her weight was 228 lbs – an increase of 6 lbs from the beginning of pregnancy –

and her height was 5’7”, giving her a BMI of 35. Her BP at that time was 120/70. At the prenatal visit on June 15, 2010 at 35 weeks gestation, her BP was 120/80 and urine was negative for protein.

On July 2, 2010, she was sent to triage for BP elevation detected at her 37 week prenatal visit. At the time of presentation, her BP was 140/102 – 135/93. She was asymptomatic with 2+ pedal edema and normal reflexes. Laboratory investigations showed normal platelet count, liver function tests and no proteinuria. She was admitted to hospital with a diagnosis of gestational hypertension.

The following day, on July 3, 2010, the induction process was started with cervical ripening. At 0830 hours, the cervix was 1-2 cm dilated, 50% effaced, posterior with the vertex at spines -2. BP was 135/96 – 134/90. A non-stress test was normal. Dinoprostone was inserted. She had some mild cramping and required nalbuphine at 1410 hours. BP was 141/97.

She was reassessed by the obstetrician at 1700 hours. The cervix was 2 cm dilated, 75% effaced with the vertex at spines -2. The dinoprostone was removed and another application was inserted. She continued to cramp during the night.

Course in Labour and Delivery

At 0430 hours on July 4, 2010, the patient’s cervix was 2-3 cm dilated, 100% effaced with the vertex at spines -1. Artificial rupture of membranes was performed for large clear fluid and she was given penicillin G intravenously.

At 0855 hours, an epidural was placed and oxytocin began shortly thereafter. At 1030 hours, the obstetrician was informed of late decelerations and the oxytocin was stopped. She was assessed by the obstetrician and the cervix was found to be 5 cm dilated and a fetal scalp clip (FSC) was applied. The fetal heart rate tracing was normal and the oxytocin was re-started.

She became uncomfortable at 1630 hours and the epidural was re-enforced with good effect. The first stage of labour was otherwise uneventful and she

reached full dilation at 1930 hours. She did not have an urge to push at that time and continued to labour. Her temperature was elevated at 38.5°C at 2000 hours. Pushing commenced at 2040 hours. During the second stage of labour, the fetal heart rate rose to 170-180 bpm. The obstetrician was notified of the fetal tachycardia. The patient was assessed at 2135 hours and the vertex was in the left occiput posterior (LOP) position at 2-3 cm below spines. Further descent was observed with pushing. The decision was made to expedite delivery with vacuum extraction because of the fetal tachycardia and episodes of variable decelerations.

The vacuum extraction cup was placed at 2140 hours with a pressure of 100 mmHg and increased to 500 mmHg with maternal pushing efforts. There was no significant descent after three pulls. The procedure was terminated at 2148 hours and a stat Caesarean section was called. The external fetal monitor was re-applied at 2204 hours. The fetal heart rate was recorded at 110 bpm. Just prior to transfer to the operating room at 2215 hours, the fetal heart was auscultated at 115 bpm. The patient arrived in the operating room at 2220 hours and the procedure was started at 2233 hours under general anaesthesia. She was delivered of a 3325 g male infant at 2240 hours. Apgars were 0 and 0 at one and five minutes. Arterial cord blood gas pH was 7.14 and bicarb was 19. Full resuscitation was carried out by the anaesthetist and an emergency room physician, but was unsuccessful. The baby was pronounced at 2258 hours.

Post Mortem

Autopsy findings were that of a male infant with above average growth parameters. No congenital anomalies or dysmorphism was identified. There was evidence of extensive subgaleal hemorrhage measuring 7X6 cm. There was vascular congestion of the brain and spinal cord which was attributed to intra-partum asphyxia.

Blood and lung cultures were negative.

There was no placental pathology to account for the intra-partum demise.

The cause of death could not be ascertained. The traumatic subgaleal hemorrhage was identified as a potential contributing factor.

Discussion

This infant was delivered stillborn by Caesarean section following a failed vacuum extraction. The most significant finding at autopsy was a large subgaleal hemorrhage and although it was described as a potential contributing factor, it was large enough to cause hemorrhagic shock and death. Subgaleal hemorrhage is a recognized complication of vacuum extraction. The incidence in spontaneous vaginal deliveries is estimated to be 4 in 10,000 versus 59 in 10,000 vacuum extractions. The reported mortality of subgaleal hemorrhage is about 12-14 per cent. Inappropriate placement of the vacuum extractor, excessive suction and rotational forces are mechanisms associated with subgaleal hemorrhage.

In this case, the operative report was detailed and described appropriate use of the vacuum extractor with respect to "good application", appropriate pressures and abandonment of the procedure after three pulls with no descent. The record did not indicate whether the baby was still in the occiput posterior position at the time of vacuum extractor application. Failure to account for this can result in an application which does not correct deflexion thereby increasing the chances of a failed procedure. It cannot be determined from this review if this was a factor in this case. In this regard, the autopsy report indicated that the boggy scalp was located on the "top of the head" without reference to the posterior fontanelle.

When the vacuum procedure failed, a "stat" Caesarean section was called. The time interval from the calling for the Caesarean section until the start of the procedure was approximately 45 minutes. Bleeding from the subgaleal hemorrhage would be ongoing during this time. Continuous electronic fetal monitoring (EFM) was not continued after the patient left the obstetrical ward to go to the operating room. This may have revealed the deteriorating status of the fetus. It does not appear from the record that consideration was given to the

possibility of failed vacuum extraction and the need to notify the operating room staff of the potential for a Caesarean section. Otherwise, it cannot be determined from this review whether it was possible to mount a Caesarean section quicker with the resources available in this centre and if so, whether this would have changed the outcome.

Recommendations

1. Obstetrical care providers are reminded of the increased risk of subgaleal hemorrhage associated with vacuum extraction.
2. Obstetrical care providers are reminded to consider the resources available at their centre for mounting a timely Caesarean section and the need to give advance notice to hospital staff of the potential need whenever possible.
3. It is recommended that electronic fetal monitoring be continued while preparations are made for emergent Caesarean section.

Case: 2011-S-13

OCC File: 2010-16173

History

The mother of the stillborn infant was a 38-year-old G₁P₀ who conceived by in vitro fertilization (IVF). Her integrated prenatal screening was negative and the pregnancy was otherwise uncomplicated. A GBS swab done at 36 weeks gestation was positive. At a routine prenatal visit at 39 weeks and 3 days gestation, she reported decreased fetal movements but when she lay down, she counted movements.

At 40 weeks gestation, she presented to obstetrical triage with decreased fetal movement for two hours, mild contractions every 7-8 minutes and spotting. Continuous external fetal monitoring (EFM) showed irregular uterine activity and the fetal heart rate had a baseline of 130 bpm with normal variability of >15 bpm. There were no accelerations (increase from baseline of 15 bpm x 15 sec) and no decelerations. The cervix was out of reach. Five hours later, the

cervix had not changed, the contractions had stopped and fetal movement was felt by the patient, so she was discharged home.

The following morning (24 hours after the first visit), she returned to obstetrical triage with increasing contractions and discomfort. The fetal heart rate (FHR) was in the 130s, contractions were irregular and mild every 2-5 minutes, and her cervix was 1 cm and posterior. FHR had a baseline of 140 bpm and there was normal variability of >15 bpm. One acceleration was noted, there were no decelerations and the tocometer showed uterine activity (as described above). The patient was encouraged to ambulate. Six hours later, she was given morphine 10 mg with dimenhydrinate 50 mg. Two hours later, the cervix was still unchanged so the patient was given the option to stay in hospital or go home. The patient chose to go home. Fetal heart rate at this time was recorded as 120 bpm.

Six hours after discharge, the patient presented to obstetrical triage with contractions every five minutes. She had not felt fetal movements for the previous two hours. No fetal heart beat could be auscultated. Intrauterine fetal demise was confirmed by ultrasound.

Labour was induced by artificial rupture of membranes and oxytocin. The stillborn infant was delivered with mid-forceps and a third degree tear was repaired. The family requested an autopsy and the coroner was contacted.

Post Mortem

The post mortem examination found Group B Streptococcus at multiple sites on the baby with early and mild chorioamnionitis in the placenta. The cause of death was determined to be Group B Streptococcus sepsis.

Discussion

There was some concern raised by the family regarding the administration of morphine at the second visit to obstetrical triage. A literature search revealed no association, other than neonatal respiratory depression, when morphine is administered shortly before birth.

Recommendations:

1. Obstetrical care providers are reminded of the SOGC Clinical Practice Guidelines for Antenatal Fetal Assessment when dealing with women with decreased fetal movement.

Chapter Four: Lessons Learned from MPDRC Reviews

One of the mandates of the MPDRC is to help identify the presence or absence of systemic issues, problems, gaps, or shortcomings in order to facilitate appropriate recommendations for prevention of future similar deaths. This is achieved by identifying trends, risk factors and patterns from the cases reviewed and making recommendations for effective intervention and prevention strategies.

Electronic Fetal Monitoring

One such area of concern that has emerged over the last few years is the administration and interpretation of electronic fetal monitoring (EFM). From 2004-2011, the MPDRC has made a total of 62 recommendations specifically pertaining to electronic fetal monitoring. Of these recommendations, 39 involved neonatal deaths and 23 were stillbirths. None of the recommendations involved maternal deaths.

Some of the recommendations pertaining to EFM include:

Obstetrical departments in hospitals are reminded that electronic fetal monitor strips should be run at a standard speed of 3 centimetres per minute to facilitate interpretation of the strip. (Neonatal 2005-24-7)

Hospitals providing obstetrical care are reminded of the importance of having health-care providers able to recognize and respond appropriately to non-reassuring fetal heart rate tracings. (Stillbirth 2006-9-1)

Obstetrical care providers are advised to become familiar with the current classification of intrapartum electronic fetal monitoring and the recommended actions in the setting of atypical and abnormal tracings. (Neonatal 2009-1 -1)

Obstetrical care providers are reminded of the importance of ensuring that the correct date and time is printing on the electronic fetal monitor (EFM) paper each time that a monitor is applied. The print out should be verified and corrected as necessary. (Neonatal 2010-2-4)

Obstetrical care providers in this case should ensure that they are able to interpret fetal heart rate monitoring and know appropriate and timely responses to an atypical/abnormal tracing. (Stillbirth 2008-7-1)

Obstetrical care providers are reminded that the documentation and interpretation of electronic fetal heart rate monitor strips should be done in a uniform way following the Guidelines of the SOGC. (Neonatal 2005-24-1)

Obstetrical care providers are reminded that concurrent monitoring of maternal and fetal heart rate is essential when there is maternal tachycardia and/or a small difference between the rates. (Stillbirth 2011-11-2)

Local, provincial and national organizations; hospitals; and obstetrical care providers should consider strategies to incorporate the recommendations and the lessons learned pertaining to EFM that may prevent deaths in the future.

Appendix A

Summary of 2011 Recommendations – Maternal

Case	Theme/Issue	Recommendations
M-1	n/a	No recommendations.
M-2	n/a	No recommendations.
M-3	Medical Policy and Procedures	<ol style="list-style-type: none">1. Obstetrical care providers are reminded that blood loss from postpartum hemorrhage is difficult to estimate and therefore clinical markers should be used as outlined in the SOGC Clinical Practice Guideline <i>“Active Management of the Third Stage of Labour: Prevention and Treatment of Postpartum Hemorrhage”</i> No. 235 October 2009.2. Hospital “A” should review its Post Anaesthetic Care Unit (PACU) policies regarding post delivery care.

Summary of 2011 Recommendations – Neonatal

Case	Theme/Issue	Recommendations
N-1	n/a	No recommendations.
N-2	Policy and Procedures	1. All obstetrical care providers at Hospital “A” should review the Fetal Health Surveillance in Labour Guidelines as per SOGC Guidelines September 2007.
	Policy and Procedures	2. Hospital “A” should review its policies regarding its intrapartum documentation.
	Quality	3. The Regional Supervising Coroner should consider a Regional Coroner’s Review of this case specifically addressing documentation by all obstetrical care providers involved.
N-3	Education/Training	1. All nurses working in remote nursing stations and remote health centres where unplanned births may occur, should be certified in the Neonatal Resuscitation Program (NRP) on an annual basis.
	Education/Training	2. Nurses working in remote nursing stations and health centres where unplanned births may occur should be required to spend a period of time on the labour and delivery unit of the hospital they transfer to and consult with.
	Education/Training	3. Air transport crews, Emergency Medical Services (EMS) attendants and nurses working in remote locations should complete an emergency obstetrical skills course annually in collaboration with their transport/receiving consulting centre.
	Resources	4. The provincial and federal governments should consider providing remote nursing stations with the capacity for electronic fetal monitoring and distant telemetry.
	Resources	5. Health Canada should conduct research to explore barriers to obstetrical care in remote First Nations communities with the goal of achieving a consistent standard of obstetrical care for all women in Ontario.
N-4	Policy and Procedures	1. Obstetrical care providers are reminded of the SOGC guidelines on antepartum and intrapartum fetal surveillance.
	Policy and Procedures	2. Obstetrical care providers are reminded of the indications for, and the SOGC guidelines on, Induction of Labour at Term. (Aug. 2001)
N-5	Diagnosis and testing	1. Obstetrical care providers are reminded that abnormalities noted on ultrasound examinations require further assessment.
N-6	n/a	No recommendations.

Case	Theme/Issue	Recommendations
N-7	Medical Resources	<ol style="list-style-type: none"> 1. The causative association between skin-to-skin care and sudden unexpected collapse of newborns remains unproven. Obstetrical care providers charged with the management of new mother-baby diads in the immediate post-partum period should, when initiating and supporting skin-to-skin care and breastfeeding, ensure that the infant's position is safe, the nose and mouth are not occluded and that parents are properly instructed. Intermittent, frequent observation should occur when skin-to-skin care and breastfeeding are being practiced, especially with primiparous mothers. 2. Further study into the association between skin-to-skin care and sudden unexpected collapse of newborns and possible preventive strategies should be undertaken.
N-8	n/a	No recommendations.
N-9	n/a	No recommendations.
N-10	n/a	No recommendations.
N-11	Policy and procedures Policy and procedures Quality Quality	<ol style="list-style-type: none"> 1. The obstetrical care providers from Hospital A should review the SOGC <i>Guidelines for Operative Vaginal Birth (Recommendation 5)</i> which states, "Failure of the chosen method, vacuum and/or forceps, to achieve delivery of the fetus in a reasonable time should be considered an indication for abandonment of the method" and proceed to immediate delivery." 2. The obstetrical care providers from Hospital A should review the SOGC <i>Clinical Practice Guidelines for Fetal Health Surveillance: Antepartum & Intrapartum Consensus Guideline (No. 197, September 2007)</i>. 3. The obstetrical care providers from Hospital A should review the assessment, treatment and implications of chorioamnionitis. 4. The Regional Supervising Coroner should conduct a Regional Coroners Review of the facts surrounding this case.
N-12	Medical Communication/ documentation	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded that specific preparation for delivery, including the most appropriate place of birth, should be considered when the risk of shoulder dystocia is considered to be high. 2. Obstetrical care providers are reminded that communication between care providers at the time of transfer of care is essential and that detailed transfer forms may help facilitate this sharing of

Case	Theme/Issue	Recommendations
	<p>Policy and procedures</p> <p>Policy and procedures</p> <p>Quality</p>	<p>information.</p> <p>3. Obstetrical care providers are reminded of the <i>SOGC Advances in Labour and Risk Management of Labour (2010-2011) Guidelines</i> with respect to prolonged second stage in labour.</p> <p>4. Obstetrical care providers responsible for fetal monitoring should follow the guidelines as set out in the <i>SOGC Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline, JOCG Vol29 No9, Sept, 2007</i>.</p> <p>5. The Regional Supervising Coroner (RSC) should conduct a review of the circumstances surrounding the death of this infant.</p>
N-13	<p>Quality</p> <p>Diagnosis and testing</p>	<p>1. The Regional Supervising Coroner should conduct a Regional Supervising Coroner's Review of the circumstances surrounding the death of this infant.</p> <p>2. Obstetrical care providers are reminded about the importance of ultrasound assessment of fetal growth and well-being in the management of hypertensive obstetrical patients.</p>
N-14	<p>Medical</p> <p>Communication/ documentation</p> <p>Quality</p>	<p>1. Obstetrical and neonatal care providers are reminded that:</p> <ul style="list-style-type: none"> a) Critical attention is required to monitor, recognize and react to changes in newborns; b) Early investigations and close monitoring are crucial to the management and care of newborns; c) IV access is critical and UV line placement is expeditious and useful; d) Neonatal Resuscitation Program (NRP) certification is highly recommended for all individuals involved in the care of newborns; e) Complete and accurate documentation is essential. <p>2. The Regional Supervising Coroner (RSC) should conduct a RSC review of this case, particularly as it relates to infant monitoring, use of investigations, documentation and communication.</p>

Summary of 2011 Recommendations – Stillbirths

Case	Theme/Issue	Recommendations
S-1	n/a	No recommendations.
S-2	Communication and documentation Policy and procedures	<ol style="list-style-type: none"> 1. Obstetrical care providers should develop public information and referral materials, both electronic and hardcopy, which can be distributed or accessed by women who are experiencing problematic substance use/abuse issues during pregnancy and post partum. 2. Child protection workers should be encouraged and/or required to refer expectant mothers with a history of substance abuse who are the subject of ongoing child protection investigations, for appropriate and timely obstetrical care. Consideration could be given to having abstinence testing as a condition of ongoing custody of children.
S-3	Policy and procedures	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded that when a woman or baby experiences an obstetrical crisis in a home setting, Emergency Medical Services (EMS) should be immediately contacted for transportation to an appropriate health care facility providing emergency obstetrical services.
S-4	Policy and procedures	<ol style="list-style-type: none"> 1. Immigration Canada and Health Canada should review their policies regarding resources, timely access, and funding for prenatal care for women who immigrate to Canada.
S-5	Policy and procedures	<ol style="list-style-type: none"> 1. All obstetrical care providers are reminded of Fetal Health Surveillance in Labour Guidelines as per SOGC Guidelines September 2007.
S-6	n/a	No recommendations.
S-7	Diagnosis and testing	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded of the importance of differentiating fetal and maternal heart rates, particularly in cases with a low fetal heart rate baseline or bradycardia. In cases where this is difficult, the simultaneous recording of the maternal heart rate with pulse oxymetry or the placement of a fetal scalp clip will help confirm monitoring of the fetal heart rate.
S-8	Medical	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded that diabetic ketoacidosis is associated with fetal and maternal morbidity and mortality and should be treated aggressively.

Case	Theme/Issue	Recommendations
	<p>Policy and procedures</p> <p>Communication and documentation</p>	<ol style="list-style-type: none"> 2. Hospital "A" should review or develop the protocol for management of diabetic ketoacidosis. 3. Hospital "A" should review the response to, and documentation of, critical laboratory results.
S-9	Diagnosis and testing	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded that concurrent monitoring of maternal and fetal heart rate is essential when there is maternal tachycardia and/or a small difference between the rates.
S-10	<p>Medical</p> <p>Medical</p>	<ol style="list-style-type: none"> 1. Obstetrical care provides are reminded to assess and consider the possible underlying causes for dystocia. 2. Obstetrical care providers are reminded of the utility of an intrauterine pressure catheter in accurately assessing the quality of uterine contractions, particularly in patients with an increased BMI.
S-11	<p>Quality</p> <p>Diagnosis and testing</p> <p>Diagnosis and testing</p>	<ol style="list-style-type: none"> 1. The Regional Supervising Coroner should assess the findings from the internal review and subsequent recommendations made by the hospital from their investigation into the circumstances of this stillbirth. 2. Obstetrical care providers are reminded that concurrent monitoring of maternal and fetal heart rate is essential when there is maternal tachycardia and/or a small difference between the rates. 3. Ultrasound assessment for fetal wellbeing should be considered in situations of persistent non-reassuring fetal heart rate monitoring to get a fuller determination of fetal health.
S-12	<p>Medical</p> <p>Resources</p> <p>Diagnosis and testing</p>	<ol style="list-style-type: none"> 1. Obstetrical care providers are reminded of the increased risk of subgaleal hemorrhage associated with vacuum extraction. 2. Obstetrical care providers are reminded to consider the resources available at their centre for mounting a timely Caesarean section and the need to give advance notice to hospital staff of the potential need whenever possible. 3. It is recommended that electronic fetal monitoring be continued while preparations are made for emergent Caesarean section.

Case	Theme/Issue	Recommendations
S-13	Policy and procedures	1. Obstetrical care providers are reminded of the SOGC Clinical Practice Guidelines for Antenatal Fetal Assessment when dealing with women with decreased fetal movement.

Appendix B - Glossary of Terms

AIH	autoimmune hepatitis
ARDS	acute respiratory distress syndrome
ARM	artificial rupture of membranes
BMI	body mass index
BPP	biophysical profile
CBC	complete blood count
CMV	cytomegalovirus
CNS	central nervous system
CPAP	continuous positive airway pressure
CPSO	College of Physicians and Surgeons of Ontario
CSF	cerebrospinal fluid
D and C	dilation and curettage
DIC	disseminated intravascular coagulation
DVT	deep vein thrombosis
EBM	expressed breast milk
EDD	estimated date of delivery
EFM	electronic fetal monitoring
EMS	emergency medical services
FFP	fresh frozen plasma
GBS	Group B streptococcus
GCT	glucose challenge test
GDM	gestational diabetes management
HIE	hypoxic-ischemic encephalopathy
ICP	intrahepatic cholestasis of pregnancy
ICU	intensive care unit
IPS	integrated prenatal screening
IVF	in vitro fertilization
LMA	laryngeal mask airway
LMWH	low molecular weight heparin
LP	lumbar puncture
MFM	maternal fetal medicine
MPDRC	Maternal and Perinatal Death Review Committee
MSS	maternal serum screening
NICU	neonatal intensive care unit
NST	non-stress test
OGTT	oral glucose tolerance test
PCOS	polycystic ovary syndrome
PICC	peripheral central catheter
PIH	pregnancy induced hypertension
PPV	positive pressure ventilation
PTT	partial thromboplastin time

QCIPA	Quality of Care Information Protection Act
RCT	randomized control trials
ROA	right occipital anterior
SFH	symphysis fundal height
SGH	subgaleal hemorrhage
SIADH	Syndrome of Inappropriate Antidiuretic Hormone
SIDS	sudden infant death syndrome
SOGC	Society of Obstetricians and Gynaecologists of Canada
SRM	spontaneous rupture of membranes
TPN	total parenteral nutrition
UAC	umbilical arterial catheter
URTI	urinary tract infection
UVC	umbilical vein catheter
VBAC	vaginal birth after Caesarean
VTE	venous thrombembolism
WBC	white blood count

Questions and comments regarding this report may be directed to:

Ms. Kathy Kerr
Executive Lead – Committee Management
Office of the Chief Coroner
26 Grenville Street
Toronto, Ontario
M7A 2G9
Kathy.M.Kerr@Ontario.ca