

Maternal and Perinatal Death Review Committee

2012 Annual Report

Office of the Chief Coroner for Ontario

April 2014



Maternal and Perinatal Death Review Committee 2012 Annual Report

This report is available in an alternative accessible format on the Office of the Chief Coroner Publications webpage.

Table of Contents

Message from the Chair	1
Committee Membership	2
Executive Summary	3
Chapter One: Introduction	3
Chapter Two: Statistical Overview (2004-2012)	6
Chapter Three: Summary of Cases Reviewed in 2012	12
Case Summaries:	
Maternal Deaths	13
Neonatal Deaths	19
Stillbirths	72
Chapter Four: Lessons learned from MPDRC reviews	92
Appendix A: Summary of Recommendations - with Identified Themes 2012 MPDRC Cases	93

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.

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 received by the mother and/or child 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 291 cases and generated 542 recommendations towards the prevention of stillbirths and deaths involving mothers and neonates. In 2012, 32 cases were reviewed and 76 recommendations were made.

The top areas of concern identified in recommendations made from 2004-2012 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.

It is my privilege to present to you the 2012 Annual Report of the MPDRC.

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

Committee Membership

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 Kryzanas
Midwife (Rural)

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.
- 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 291 cases and generated 542 recommendations aimed towards the prevention of future similar deaths.
- Each year, an average of 32 cases are reviewed and 60 recommendations are made.
- The top areas of concern identified in recommendations made from 2004-2012 relate to: medical and nursing issues; policy and procedures; communications/documentation; and diagnosis and testing involving electronic fetal monitoring.
- In 2012, 32 cases were reviewed and 76 recommendations were made.
- Of the 32 cases reviewed in 2012, three were maternal, 20 were neonatal and nine 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.

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 Deaths, Stillbirths, Perinatal and Neonatal Deaths

The MPDRC 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 – during pregnancy at >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. (Vital Statistics Act of Ontario)

Perinatal deaths are defined as deaths during, at the time of, or shortly after birth, including home births.

Neonatal deaths are defined as deaths within the first seven days after birth.

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 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

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

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

Investigating 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 to agencies and organizations which 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 MPDRC 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 rely 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 MPDRC 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 (2004-2012)

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 2004-2012, the MPDRC has reviewed a total of 291 cases. Of these cases, 87 (30%) were maternal, 129 (44%) were neonatal and 75 (26%) 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, investigating coroner or Regional Supervising Coroner have concerns about the care that the mother or child received.

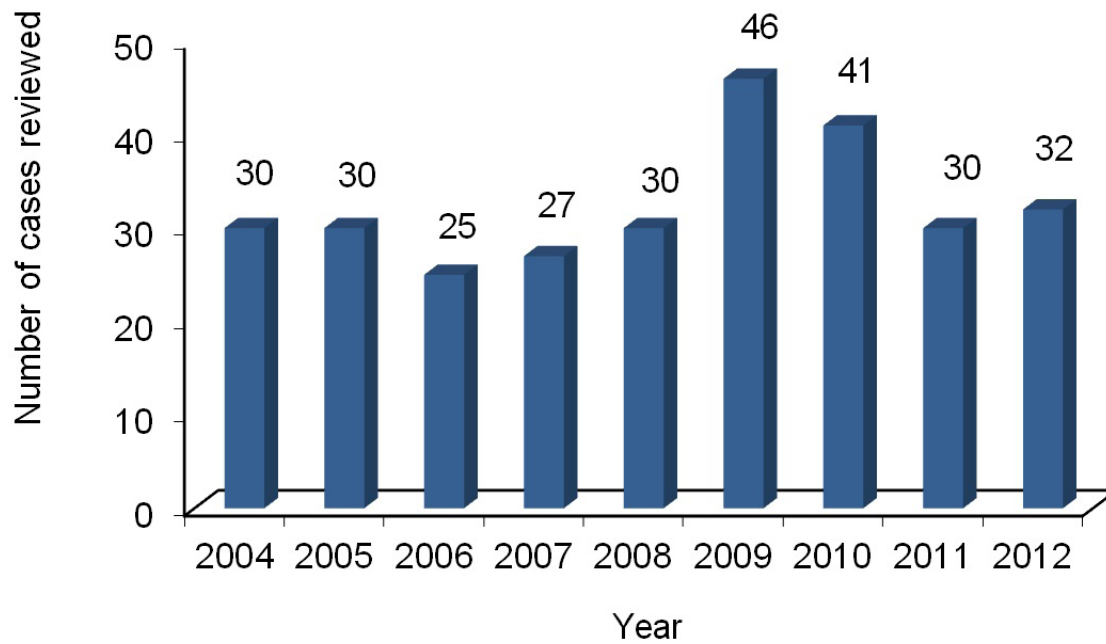
The number of cases noted in Chart One is based on the year the case was reviewed, which, in many cases, is not the same year in which the death actually occurred.

Chart One: MPDRC - # of Cases Reviewed (2004-2012)

	2004	2005	2006	2007	2008	2009	2010	2011	2012	Total
Total # of cases reviewed	30	30	25	27	30	46	41	30	32	291
Maternal	10	12	4	15	8	21	11	3	3	87
Neonatal	12	11	13	12	12	16	19	14	20	129
Stillbirth	8	7	8	0	10	9	11	13	9	75

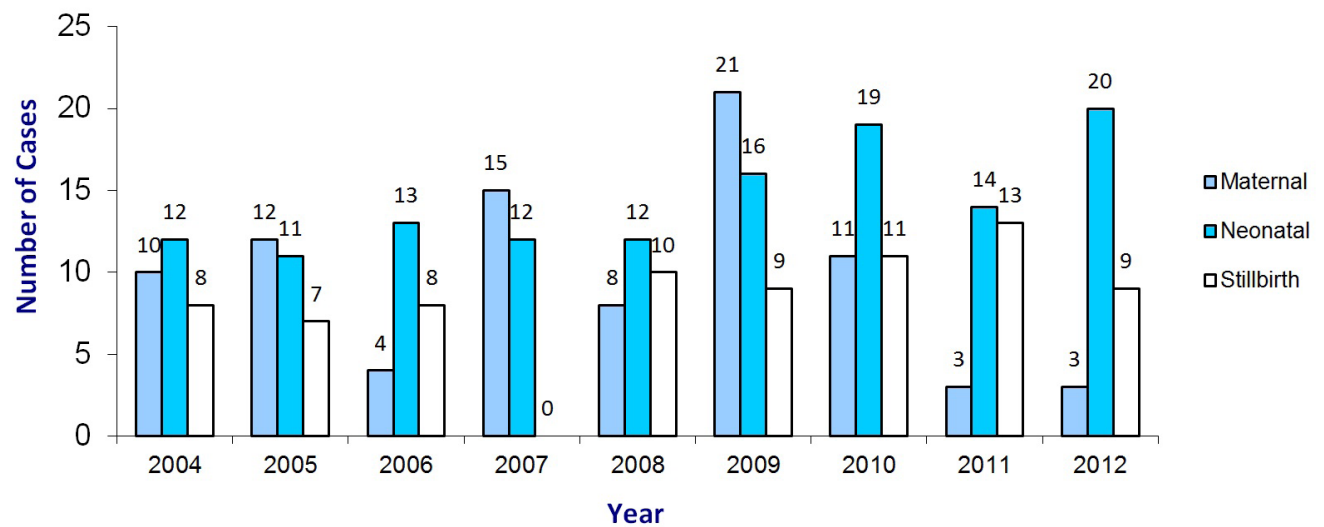
Chart One indicates that the number of total cases reviewed from 2004-2012 has varied from a low of 25 cases in 2006, to a high of 46 cases in 2009. This variance is likely reflective of committee administrative practices (e.g. time required for processing of review materials and compilation of final reports).

Graph One: Total number of cases reviewed by the MPDRC based on year (2004-2012)



Graph One demonstrates how the number of cases reviewed from 2004-2012 has remained relatively consistent. On average, the MPDRC reviews 32 cases per year.

Graph Two: Number of cases reviewed based on type of case (2004-2012)



Graph Two demonstrates that from 2004-2012, the majority of cases reviewed each year are neonatal deaths, followed by stillbirths.

Chart Two: MPDRC - # of Recommendations (2004-2012)

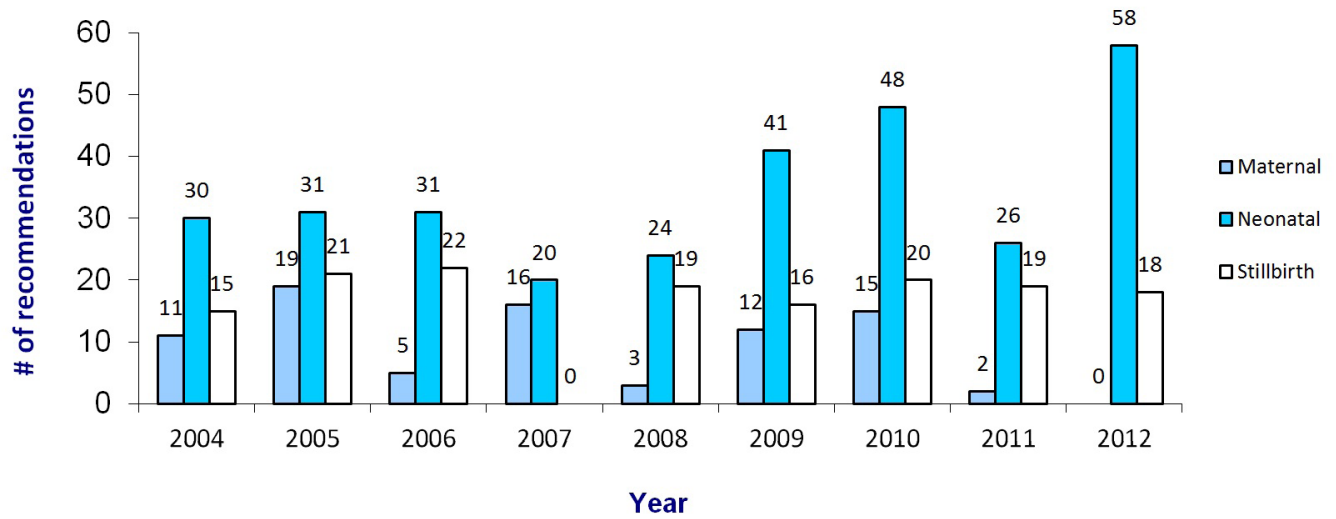
	2004	2005	2006	2007	2008	2009	2010	2011	2012	Total
Total # of Recommendations made	56	71	58	36	46	69	83	47	76	542
Maternal	11	19	5	16	3	12	15	2	0	83
Neonatal	30	31	31	20	24	41	48	26	58	309
Stillbirth	15	21	22	0	19	16	20	19	18	150

Chart Two indicates that the MPDRC has generated a total of 542 recommendations from 2004-2012. From this total, 83 (15%) were related to maternal cases, 309 (57%) from neonatal cases and 150 (28%) from stillbirth cases. Consistently over the years, the majority of cases and recommendations relate to reviews of neonatal deaths. On average, 60 recommendations are made per year.

Upon reviewing the recommendations that have been made, certain areas of concern 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 (EFM) (e.g. interpretation of 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)

Graph Three: Number of recommendations based on type of case 2004-2012



Graph Three demonstrates that from 2004-2012, the majority of recommendations generated each year pertain to neonatal cases.

Maternal and Perinatal Death Review Committee 2012 Annual Report

Chart Three: MPDRC – Number and percentage of recommendations based on area of concern/theme and type of case (2004-2012)

	Maternal	Neonatal	Stillborn	Total	% of Total
Medical	36 7%	61 11%	35 6%	132	24%
Policy and procedure	20 4%	59 11%	28 5%	107	20%
Communications/documentation	7 1%	50 9%	27 5%	84	15%
Quality	11 2%	31 6%	10 2%	52	10%
Diagnosis and testing	1 0%	38 7%	18 3%	57	10%
Diagnosis and testing - EFM	0 0%	40 7%	24 4%	64	12%
Education/Training	1 0%	16 3%	4 1%	21	4%
Resources	2 0%	11 2%	3 1%	16	3%
Transfer	4 1%	6 1%	2 0%	12	2%
Other	0 0%	2 0%	0 0%	2	0%

Chart Three demonstrates that 24% of all recommendations made by the MPDRC from 2004-2012 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 15% to communication and/or documentation and in particular, the timely and accurate sharing of information between healthcare providers and with the patient.

Chart three also demonstrates the following key areas (based on type of case and theme):

- 11% of all recommendations were from neonatal cases and had a medical/nursing theme
- 11% of all recommendations were from neonatal cases and had a policy and procedure theme
- 9% of all recommendations were from neonatal cases and had a communication/documentation theme

One area of specific concern that has been identified over the past few years relates to the use of EFM technology, how EFM results are interpreted by obstetrical care providers and what follow-up actions are taken in response to the findings. From 2004-2012, there have been 64 recommendations made specifically pertaining to EFM.

Chapter Three: Summary of Cases Reviewed in 2012

This annual report includes summaries of reviews conducted by the MPDRC in 2012. Cases reviewed may involve deaths that occurred in previous years.

Total number of cases reviewed: 32

Total number of recommendations: 78

Number of maternal cases reviewed: 3

Number of maternal cases noted for statistical purposes only*: 5

Total number of maternal deaths: 8

Number of recommendations from the maternal deaths reviewed: 0

Number of neonatal cases reviewed: 20

Number of recommendations from the neonatal deaths: 58

Number of stillborn cases reviewed: 9

Number of recommendations from the stillborn cases: 18

* The MPDRC 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 contribute 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 2012, 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.

A summary of recommendations made in 2012, together with the identification of themes, is included as Appendix A.

Case Summaries: Maternal Deaths

Reporting of Maternal Deaths in Ontario

The MPDRC 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: 2007-2994

The deceased was a 29-year-old woman who was 14 weeks pregnant. The cause of death was external neck compression and the manner of death was homicide. The homicide appears to be unrelated to the woman’s pregnancy and not domestic in nature.

2. OCC file: 2011-6998

The deceased was a 26-year-old woman who was four to five months pregnant. The cause of death was blunt force trauma of the head and external neck compression. Manner of death was homicide. This death will also be reviewed by the Domestic Violence Death Review Committee.

3. OCC file: 2012-903

The deceased was a 22-year-old woman who was 28 weeks pregnant. The cause of death was pulmonary complications of right sided infective endocarditis due to intravenous drug use. The manner of death was natural.

4. OCC file: 2009-2115

The deceased was a 39-year-old woman who had delivered a premature infant eight days before her death. The child was doing well, but was still in hospital as it was born

at 34 weeks gestation. The deceased had a history of pregnancy induced hypertension and had delivered a child 13 months prior to her death. During this pregnancy, she was maintained on antihypertensive medication and was on the antibiotics for premature rupture of membranes. The cause of death was shown to be a ruptured Berry aneurysm leading to a subarachnoid hemorrhage. Manner of death was natural.

5. OCC file: 2011-7319

The deceased was a 34-year-old woman in her first trimester of pregnancy. The cause of death was blunt impact to the chest and abdominal injuries due to a motor vehicle collision. The manner of death was accident.

The following cases received full reviews by the MPDRC:

Case: 2012-M-1

OCC file: 2011-3694

History

The deceased was a 36-year-old G1P0 with an estimated date of delivery (EDD) of March 25, 2011. She had a history of endometriosis and infertility and had conceived on clomiphene. An early ultrasound showed a twin pregnancy with demise of one of the twins. The antenatal records do not indicate if prenatal genetic screening testing was offered.

Routine second trimester ultrasound showed an 18 week five day singleton pregnancy. A succenturiate lobe placenta was identified. Routine prenatal laboratory investigations were normal. She was Rh negative and received Rh immune globulin as indicated. An ultrasound at 34 weeks gestation showed normal fetal growth and amniotic fluid index. The succenturiate lobe was again identified. She was group B streptococcus (GBS) negative. Her pregnancy was complicated by nausea and vomiting and she experienced syncopal episodes. Cardiology and neurological referrals were made and investigations were normal.

Her past medical history included a laparoscopy in 2003 for endometriosis. She had asthma and psoriasis. Her family history was non-contributory.

Course in Labour and Delivery

The patient was admitted to the labour and delivery unit on March 25, 2011 at 0115 hours after spontaneous rupture of membranes at 1800 hours on March 24, 2011. Labour was induced with oxytocin. At 0745 hours, the cervix was 2 cm dilated. An epidural was placed at 0858 hours. At 0904 hours, there was a fetal heart rate (FHR) deceleration which resolved with oxygen and re-positioning. A second FHR deceleration occurred at 0930 hours which recovered spontaneously. The cervix was 5 cm dilated.

At 1055 hours, there was a moderate amount of vaginal bleeding. At 1103 hours, she vomited, her eyes rolled back and she became unresponsive. She was attended by the anaesthetist. Her blood pressure dropped and she was given ephedrine 15mg. The FHR pattern was abnormal. Preparations were made for emergency

Caesarean section. While en route to the operating room, she regained consciousness.

Under general anaesthesia, she was delivered of a 6 pound 13 ounce male infant at 1130 hours. There was no evidence of abruption. Apgars were 2, 4 and 8 at one, five and ten minutes. Cord blood pH was 6.7 arterial and venous. The baby was subsequently transferred to the neonatal intensive care unit (NICU) at a larger hospital with a specialized unit.

Following delivery, there was heavy bleeding and uterine atony. Interventions including carboprost tromethamine (Hemabate®), ergometrine, misoprostol and a uterine balloon were ineffectual. A vascular surgeon was summoned and the aorta was cross-clamped and the internal iliac arteries were ligated. The left common internal iliac was injured and repaired. A hysterectomy was performed at 1250 hours. At 1300 hours she arrested and was successfully resuscitated. She had a second arrest shortly thereafter and was again successfully resuscitated. On completion of the surgery, the abdomen was packed open and she was transferred to the intensive care unit (ICU) at 1550 hours. She had a third arrest just prior to transfer to the ICU. She received 6L of crystalloid, 1.5L of colloid, 29U of packed red cells, 24U of fresh frozen plasma, 25U of platelets, 30U of cryoprecipitate and 3 doses of Factor VII.

In the ICU, she continued to receive blood products and inotropes. Her blood pressure and urine output stabilized but she remained acidotic. She suffered a fourth arrest at 1945 hours and the cardiac output could not be restored. The abdomen was opened in the ICU to rule out an abdominal compartment syndrome and a pericardiocentesis was performed without any improvement. She was pronounced dead at 2040 hours on March 25, 2011.

Post mortem

The pertinent findings at autopsy were the finding of widespread collections of intravascular anucleate squamous and keratinaceous debris.

The injury and repair to the right common iliac artery was noted. It was felt not to have contributed to the death. A literature review did not reveal an association with fetus papyraceous or succenturiate placenta.

The cause of death was amniotic fluid embolus and its sequelae.

Discussion

The patient died in labour due to an amniotic fluid embolus and the resultant shock and disseminated intravascular coagulation (DIC).

Amniotic fluid embolus is estimated to occur in one in every 8,000 to 80,000 births. Factors such as precipitous or tumultuous labour, medical induction, placenta previa and abruption have been associated with amniotic fluid embolus; however it is best considered an unpredictable and unpreventable event.

The main clinical findings are the sudden and severe onset of cardiogenic shock, respiratory failure causing severe hypoxemia and DIC. Non-specific symptoms such as vomiting and agitation may precede the onset of shock. Tonic-clonic seizure activity may also occur. The profound maternal cardiorespiratory

collapse commonly results in fetal compromise and an abnormal FHR tracing necessitating rapid delivery. Caesarean section performed in the setting of DIC further contributes to maternal morbidity and mortality, but the fetal condition often does not allow time for the coagulopathy to be treated. As a result, both maternal and fetal outcomes are poor. The maternal mortality is estimated between 20-60%.

The presentation in this case was very typical for amniotic fluid embolus. The decision to move quickly to Caesarean section was predicated by the abnormal FHR tracing. Cardiogenic shock and DIC became manifest during the operation and were treated appropriately. Despite heroic efforts, the patient suffered a fourth cardiac arrest from which she could not be resuscitated.

Recommendations

None

Case: 2012-M-2

OCC file: 2011-14913

Antenatal History

The deceased was a 33-year-old G1P0 with an EDD of April 20, 2012 by ultrasound on October 7, 2011 done for integrated prenatal screening (IPS). She had been previously diagnosed with sickle cell trait and beta thalassemia trait. There was a family history of hypertension in her mother.

She attended routine prenatal visits at 10, 15 and 18 weeks gestation. Her blood pressure readings were 140/82, 130/78 and 130/80 respectively at these visits. At the last visit on November 22, 2011, the uterine size was appropriate for dates and a positive fetal heart was auscultated. A notation was made that the alpha fetoprotein (AFP) was elevated.

On December 4, 2011, at 20 weeks gestation, she presented to the emergency department of Hospital A. The triage record

indicated that her presenting complaint was increasing shortness of breath since December 3, 2011. She had a cough for four days and had shortness of breath on exertion the day before. Air entry was reported to be decreased in the left lower lobe. Vital signs were P 93, RR 20, T35.5 C and BP 154/91. O2 saturation was 100%.

The emergency department physician noted a one week history of upper respiratory tract infection symptoms. She had no nausea or vomiting and no vaginal bleeding or discharge. On examination, she was noted to be tender in the left lower quadrant, possibly due to round ligament pain. A urinalysis and ultrasound were ordered.

The ultrasound showed a 20 week size fetus with no fetal heartbeat; this was consistent with fetal demise. Urine dipstick showed 25 leukocytes, nitrite positive, protein 5 g, urobilinogen 17, bilirubin 50 and erythrocytes 250. Microscopic urinalysis showed 0-5 erythrocytes, 5-10 leukocytes, 1+ bacteria and 1+ fine granular casts.

The woman was informed of the ultrasound results and was

instructed to follow up with her obstetrician at her routinely scheduled prenatal visit the next day. She was discharged home at 1910 hours with a prescription for cephalexin (Keflex®) for a presumed urinary tract infection.

Her pain and shortness of breath continued and later that night/early morning, she awoke with severe shortness of breath, abdominal pain and vaginal spotting. Her husband called 9-1-1 and she was transported to Hospital B.

She arrived at emergency department of Hospital B at 0333 hours on December 5, 2011. Her vital signs were pulse 76, respiratory rate 30, temperature 36.0 C, blood pressure 189/114 and O2 saturation 95%.

At 0405 hours, the emergency department physician was called to see her. She was found pale and in severe respiratory distress. While being moved stat to the resuscitation room, she became apneic and seized. A code was called. Full resuscitation was commenced, but was called after 50 minutes. The time of death was 0504 hours on December 5, 2011.

Blood work drawn just prior to the arrest subsequently reported Hb 54, platelets 45, creatinine 175, anion gap 36.

Post mortem

Autopsy of the fetus revealed evidence of intrauterine growth restriction (IUGR) and changes consistent with recent (24-48 hours) intrauterine demise.

Examination of the placenta revealed extensive syncytial necrosis. There was sickling of maternal red blood cells within the maternal space. There was extensive and severe decidual vasculopathy with thrombosis of some spiral arteries.

The significant finding on maternal autopsy was extensive and widespread trophoblast emboli in the pulmonary arterioles and capillaries. The cause of the trophoblast emboli was attributed to the abnormal placentation. As the cells became necrotic in the placenta, they gained access to the maternal circulation and subsequently embolized to the lungs.

The underlying cause of the placental abnormalities is uncertain.

The autopsy report suggested a pregnancy disorder either pre-existing or pregnancy induced, including pre-eclampsia or HELLP (hypertension/elevated liver enzymes/low platelets) syndrome.

The cause of death was trophoblastic pulmonary embolism due to decidual vasculopathy and trophoblastic necrosis of the placenta due to hypertensive disorder occurring during pregnancy.

Discussion:

This woman died from trophoblastic embolus at 20 weeks gestation. Abnormal placentation resulted in hypo-perfusion of the placenta causing syncytial necrosis and a breakdown between the fetal and maternal circulation allowing trophoblastic tissue access to the maternal circulation and subsequent pulmonary embolization. The underlying cause of the abnormal placentation is not certain.

Hypertensive disorders are a common cause of placental dysfunction although such severe manifestations are very unusual at this gestational age. The severe hypertension evident in the emergency department may have been due to the underlying disorder or secondary to the consequences of the underlying disorder such as the severe pain produced by the local ischemia in the uterus. An underlying thrombophilia may produce thrombosis and hypo-perfusion of the placenta causing early pregnancy loss. There was no family history to suggest an inherited thrombophilia and no events in her past medical history that would have led to a thrombophilia work up.

The woman presented to Hospital A's emergency department on December 4, 2011 with a chief complaint in triage of shortness of breath. The triage nurse identified decreased air entry in the left lower lobe. Her vital signs were stable and oxygen saturation was normal. The emergency department physician did not appear to have done any further evaluation of her cardiopulmonary function and focused rather on her abdominal pain and the fetal demise. No comment was made on the abnormal urinalysis results other than suspecting a urinary tract infection. The possibility of a hemolytic process and resulting anemia was not investigated.

Trophoblastic release into the maternal circulation can trigger DIC and hemolysis. This would explain the severe anemia and thrombocytopenia when she presented early the next day to the emergency department at Hospital B.

It cannot be determined from this review whether such a process could have been identified by doing a complete blood count

(CBC) at the time of her presentation to Hospital A's emergency department and whether this would have ultimately changed the outcome.

Recommendations

No new recommendations.

Case: 2012-M-3

OCC file: 2012-3065

Clinical History:

The deceased was a 26-year-old formerly healthy gravida 1 patient who had an EDD of April 8, 2012. Apart from a prior history of migraines, she had no significant previous health problems. She had all routine testing, including antenatal blood testing, integrated prenatal screening, glucose challenge test and second trimester ultrasound scan; all tests were normal.

She was followed by an obstetrician from 14 weeks gestation and her last recorded visit was on February 27, 2012 at 34 weeks and two days. Up until that time, her blood pressure was normal. Her symphysis-fundal height was 31 which may have indicated early IUGR. Streptococcus screening on that date was negative. An ultrasound done on February 29, 2012 showed a fetus weighing 1859 g (in the 14th percentile). There was evidence of asymmetric IUGR with the abdominal circumference lagging by the equivalent of three weeks. There was otherwise normal amniotic fluid and cord dopplers.

On March 4, 2012 at 35 weeks gestation, she complained of a severe headache and vomiting that was not relieved by aspirin.

On March 5, 2012 at 0056 hours, the woman's husband took her to the obstetrical unit of Hospital A. When assessed, she had scotoma of her right eye and her pressure was 168/103 with brisk reflexes. She initially complained of weakness in her left arm, however on re-checking, the power and grip in both hands were normal. She had significant proteinuria. Pregnancy induced

hypertension blood work was normal and she was started on oral labetalol as well as intravenous magnesium sulfate.

As a result of the diagnosis of severe preeclampsia and her concerning symptoms and associated early IUGR, it was recommended that she undergo an emergency Caesarean section. In preparing for the Caesarean, the anaesthetist did a spinal for a bloody tap. A second site was used and again a grossly bloody tap was observed. The procedure progressed under effective spinal anaesthesia. A low segment Caesarean section was performed and the 35 week female infant weighing 1835 grams was delivered at 0341 hours on March 5, 2012. Apgars were 9 and 9 and cord gases were within the normal range.

The baby was taken to the NICU for care. The remainder of the procedure was unremarkable. Placental pathology revealed the weight just below the 10th percentile which was not unexpected with the IUGR and severe preeclampsia. There were no other significant findings.

At approximately 0530 hours, the mother was assessed and was becoming increasingly confused and slow to respond. She demonstrated numbness to the left arm and hand and decreased grip strength in the left hand. These findings, together with the prior blood tinged cerebrospinal fluid and the persisting headache, suggested a presumptive diagnosis of subarachnoid bleed. Referral was made to Medicine as well arrangements for a CT scan. There was evidence of a right frontoparietal intracranial hemorrhage measuring 5.7 cm, shift and some uncal herniation. With these findings, arrangements were made through Criticall (a 24-hour-a-day emergency referral service for physicians across the province) for transfer to the neurosurgical

service at Hospital B.

On March 5, 2012, she departed Hospital A at 1046 hours and arrived at Hospital B at 1112 hours. CT angiography was done at approximately 1200 hours. Due to a worsening Glasgow coma score, she went to the neurosurgical operating room for a right craniotomy and evacuation of a 5 cm intracerebral hematoma secondary to a presumed superior sagittal sinus thrombosis. She continued to be followed by repeated brain imaging and was stabilized post operatively.

On March 7, 2012, however, she deteriorated with fixed dilated pupils bilaterally. There was marginal improvement with mannitol. An urgent CT scan showed slight expansion of the clot, but significant midline shift. She was taken back to the operating room for a right hemicraniectomy with subtemporal decompression, partial right frontal lobectomy and evacuation of frontal hemotoma.

She again stabilized after the second surgery and obeyed commands with her right side; she was still intubated.

Overnight and into the morning of March 9, 2012, she developed increasing intracranial hypertension, refractory to medical treatment. She was sent back to the operating room for decompressive craniectomy. Post operatively, after she had stabilized, they reintroduced heparin because of the sagittal sinus thrombosis. This was done after close consultation with neurology and hematology. Despite these treatments, she developed progressive increase of intracranial pressure refractory to all medical methods of management. Her clinical status deteriorated and on March 13, 2012, her husband talked to staff about withdrawing care.

The woman died on March 14, 2012, nine days post partum. The cause of death was reported as superior sagittal sinus thrombosis associated intracranial bleed.

Post mortem

A post mortem examination was not conducted.

Discussion:

This patient was a primip who developed sudden onset of severe preeclampsia. An ultrasound a couple of days prior revealed mild IUGR, however her clinical course had been normal, with normal blood pressures prior to that. From the time of admission to hospital, she had early signs of a cerebral bleed with a bloody tap and some transient weakness, complicating severe preeclampsia. Shortly post-operatively, she developed definite neurological signs, had emergency imaging and consultation, and then was urgently transferred to a neurosurgical centre.

Despite close following with imaging, clinical care, and repeated brain surgeries, the woman's clinical course, though stabilizing on a number of occasions, gradually deteriorated until she died of this complication of severe preeclampsia.

A cerebral bleed is a known, though relatively uncommon and unpredictable, complication of severe preeclampsia. This woman had expedient investigation and care.

Recommendations:

No new recommendations.

Case Summaries: Neonatal Deaths

Case: 2012-N-1

OCC File: 2011-6803

History:

The deceased infant was born on March 20, 2011 to a 31-year-old primagravid woman at 25 weeks and six days gestation.

The mother was healthy and had experienced an uneventful pregnancy until March 15, 2011 when she presented to her community hospital with abdominal cramping. She was not felt to be in premature labour and was sent home, but returned a few days later with more cramping. She was admitted on March 19, 2011 with a diagnosis of threatened preterm labour. She was given betamethasone and transfer to a local tertiary perinatal facility was arranged.

Upon arrival at the tertiary centre, she was found to have bulging membranes and was having occasional contractions. She was transferred to the high-risk antepartum floor for ongoing monitoring. A consultation with the neonatal team took place and the parents were offered the opportunity to have their baby participate in a clinical study called "GentleR" if early delivery was to take place. The parents agreed to participate in the study.

Several hours after transfer, membranes were found to be bulging into the vagina. The risks of infection, malpresentation or unattended birth were discussed and a recommendation was made to initiate oxytocin and deliver the baby. Vaginal delivery of a live baby boy followed. Delayed clamping of the umbilical cord for 45 seconds took place and the baby was handed to a member of the neonatal team who was present to receive the baby. Members of the GentleR Study team were in attendance to guide the early respiratory management of the baby.

The baby received support with mask continuous positive airway pressure (CPAP), supplemental oxygen and a sustained inflation. He responded well with good heart rate, respiratory drive and oxygenation. Apgar scores were 8 at one minute and 9 at five minutes. Surfactant was given into the trachea via a small catheter as per the GentleR protocol as the baby was exhibiting signs

of Respiratory Distress Syndrome. He tolerated the procedure well and the supplemental oxygen was weaned. Intravenous caffeine was administered to support respiratory drive. Blood cultures were drawn and he was started on antibiotics. The baby was transferred to the NICU on nasal CPAP with a peripheral intravenous catheter in place.

On examination, the baby appeared active, well perfused and pink in room air. His birth weight was 800 grams. His physical examination was normal for his gestational age of 25 weeks and six days.

The baby was quite stable on nasal CPAP over the first four days. Feeds were commenced when expressed breast milk became available and were advanced to 1cc q4h by day four. A head ultrasound performed on day two revealed bilateral subependymal haemorrhages and a small left Grade 2 intraventricular haemorrhage.

On day five, the baby began to exhibit an increased frequency of apnoeic and bradycardic spells with desaturation. He was given an extra dose of caffeine and CPAP pressures were increased. The spells persisted (with more than 3/hr), so he was switched to nasal high-frequency oscillation ventilation (HFOV) as per the GentleR protocol. The baby's abdomen became distended and "loopy" and bilious aspirates were recovered from the orogastric tube. He was placed on "nil per os" (NPO) [nothing by mouth].

There was a discussion with the mother where she expressed concern that, by participating in the GentleR study, she might be "torturing the baby as he was having increased spells and his respiratory rate was very low." It was explained to her by the study team that, "while the threshold for intubating him is high as he is part of the study, our first priority is that he comes to no harm." A family meeting was arranged for the next day with an interpreter to assist the father. The baby's clinical status was reviewed and the specific elements of the GentleR study explained again.

The baby was described as being "better on HFOV," but continued to have fairly frequent apnoeic spells. However, he recovered from each spell with gentle stimulation. He was restarted on feeds and tolerated 1cc q4h of expressed breast milk. A murmur

was heard for the first time, suggesting the emergence of a patent ductus arteriosus (PDA). Three doses of indomethacin were administered on Days 8-9 resulting in successful closure of the PDA which was confirmed by an echocardiogram.

The baby remained relatively stable for the next few days on nasal HFOV with FiO₂ requirements of 0.21 to 0.30. Apnoeic spells continued, but were reduced in frequency and severity. Feeds were maintained at 1 cc q2h until the late evening of day 11 when there was a small bilious aspirate followed by a small amount of blood aspirated from the orogastric tube. The abdomen was mildly distended, but soft with bowel sounds present. The baby was placed on NPO, an abdominal x-ray was ordered and ranitidine started. The abdominal film revealed only gaseous distension with no pneumatosis intestinalis. A repeat abdominal x-ray taken later in the day revealed the same findings with no radiologic evidence of necrotizing enterocolitis (NEC).

An attempt was made to transition the baby from nasal HFOV to biphasic nasal CPAP on Day 12. This resulted in an increase in apnoeic/bradycardic spells and, after considerable discussion, it was decided to place the baby back on nasal HFOV. The spells continued and the baby appeared lethargic with mildly mottled skin. Sepsis was suspected, so he was started on antibiotics after a septic workup. He continued to have frequent apnoeic spells, but was maintained on nasal HFOV until a decision was reached to intubate him on the morning of day 14 after several spells that required bag and mask ventilation. This resulted in a reduction in the spells, but he remained "fragile to handling" and lethargic.

Over the next few days, the baby's general condition improved and he was restarted on feeds. The urine sample from the septic workup revealed yeast and amphotericin B was added to his antibiotic regimen. He received a seven day course of antibiotics including amphotericin B which was discontinued when a second urine culture was negative and other investigations revealed no evidence of systemic fungal infection. His feeds were gradually advanced until he was on full feeds by Day 18 at which time Human Milk Fortifier (HMF) was added into the expressed breast milk feeds.

On Day 23, the baby was found to be extubated and was given a trial of biphasic CPAP. He did well initially, with FiO₂ requirements

generally from 0.30-0.40 and having only occasional spells. His abdomen was somewhat distended, but soft with bowel sounds present. Stooling pattern was normal.

On Day 26, he had several severe apnoeic spells requiring bagging and was reintubated. His FiO₂ requirements had increased and a chest x-ray revealed loss of lung volume. He was placed on HFOV and appeared to improve at first, but became unstable again the next evening with frequent swings in oxygenation. Ventilator pressures were difficult to maintain due to a leak around the small endotracheal tube (ETT), so the tube was replaced with a larger 3.0 ETT. With this manoeuvre, the baby's oxygenation improved and he became more stable. Feeds were temporarily held and an abdominal x-ray revealed gaseous distension. Feeds were restarted and gradually increased.

Over the next week, the baby did well on ETT HFOV at relatively low settings. The abdomen had been intermittently distended, but was soft with bowel sounds present. His feeds were advanced to full by Day 32, fortifier was reinstituted and the PICC (peripheral intravenous in the central circulation) removed. He was extubated to biphasic CPAP and tolerated it well. He received a red blood cell transfusion on Day 33 for a haemoglobin level of 91 g/L.

On Day 35, the baby was found to be doing well when assessed by the team physician at 1500 hours. The baby had one loose stool, but was tolerating full feeds of fortified expressed breast milk; his abdomen was described as soft and non-distended with good bowel sounds.

Later in the evening on Day 35, after having several additional loose stools, the baby presented with vomiting, increased abdominal distension and an increase in apnoeic spells. An abdominal x-ray was suggestive of NEC, so he was placed NPO and started on antibiotics after a blood culture was drawn and a lumbar puncture attempted. The baby rapidly developed evidence of severe NEC with peritonitis, shock and acidosis. He was intubated, provided with fluid resuscitation and transferred to the children's hospital for consultation with general surgery.

When the baby arrived at the children's hospital, he was considered moribund and too unstable for laparotomy. The

parents were counselled that the prognosis was grave and no further intervention was undertaken. The baby died at 1005 hours on April 24, 2011.

Post mortem

This case was not reported to a coroner at the time of the death. A coroner's warrant autopsy was therefore not ordered, and consent for a hospital autopsy was not obtained.

Cause of death was Necrotizing Enterocolitis (NEC) with Extreme Prematurity as a contributing factor

Discussion:

This baby died at 35 days of age after developing severe NEC. He had been born very preterm at 25 weeks and six days gestation and had experienced several of the common conditions affecting babies born this early, including respiratory distress syndrome, patent ductus arteriosus, apnoea of prematurity, probable nosocomial infection and mild, early bronchopulmonary dysplasia. Despite these complications, the baby had reached a period of relative stability only to subsequently develop a severe form of NEC from which he could not recover.

Accompanying the hospital records detailing the baby's clinical course was correspondence from the mother outlining a number of incidents that caused her to be increasingly anxious about the care her newborn baby was receiving.

The mother was extremely dissatisfied with the care that her baby and the family received. While it is not possible for the MPDRC to substantiate the allegations made, it was clearly evident that an extreme lack of trust developed between the baby's parents and the members of the healthcare team and this created a great deal of friction/tension that had substantial personal impact on all involved.

Although the baby's parents appear to have had a great deal of difficulty in their dealings with the care team and a number of examples of unsatisfactory communication were identified, it is evident from a review of the medical charts that the healthcare team took a number of steps to improve communication and alleviate tension. Despite these efforts, considerable conflict

and disagreement remained.

Issues and concerns identified by family:

The MPDRC sought additional input from an expert in paediatric medicine to determine if there were any issues, errors or omissions in the care provided to this baby that may have contributed to the death and if so, what could be done to prevent future, similar occurrences.

Comments are provided on the following areas:

a) Necrotizing enterocolitis – presentation and causation

Although no autopsy was performed, the cause of this baby's death was almost certainly severe NEC. Those caring for the baby at the tertiary perinatal centre and at the children's hospital agreed that the clinical and radiological findings were typical. NEC occurs in approximately 7-14% of preterm infants born at less than 1500 g birth weight and there is a case fatality rate of between 20-50%.

NEC is a frustrating disease that often attacks without warning and results in devastating consequences. To date, there is no consensus on causation and preventive strategies have been largely unsuccessful or inconsistent in their impact. Many of the early clinical signs of NEC are similar to those exhibited by babies without NEC (e.g. apnoea, abdominal distension, aspirates, unusual stooling, etc.) and there is little evidence that early identification and medical intervention can influence the natural history. The title of a recent review article highlights the problematic nature of this disease: Necrotizing enterocolitis: A multifactorial disease with no cure.¹

There were two themes regarding causation that the baby's mother identified in her correspondence. Firstly, she felt that the baby exhibited abnormal signs from very early on and was suffering from a bowel problem that led to the fatal case of NEC. She suggested that if the clinical team had taken some different action, the outcome would have been different. She was most concerned with the baby's recurrent abdominal distension that was present from early on and attributed to the effect of nasal CPAP by the clinical team.

Abdominal distension is a common finding in preterm babies on CPAP and, although it can be a presenting feature of NEC, it is most commonly mechanical and, unless severe, requires no change in management. In this baby's case, there were several times when his distension became more pronounced, prompting the performance of abdominal x-rays. None of these films revealed evidence of NEC until the one taken on the day before he passed away.

NEC tends to present rapidly and definitively and is not a grumbling, insidious disease that appears over a period of weeks. In this baby's case, the first sign of NEC appears to have been the watery stools on the day before his death. The abdominal signs presenting prior to this were not a manifestation of the disease. No different management strategy was indicated based on a review of the records.

The second issue identified by the mother was the possible role of breast milk fortifier in causing NEC. Feeding with breast milk has been the only accepted, effective measure to reduce the risk of NEC and all NICUs promote its use for this and many other reasons. Use of standard cow's milk-based infant formulas appears to increase the risk of NEC. However, if tiny preterm infants are fed with human milk alone, growth and bone mineralization are impaired. It has become standard to use proprietary, human milk fortifiers as additives to breast milk being fed to preterm babies to help boost protein, caloric and mineral intakes. As the protein source for these products is cow's milk, the possible link between use of fortifiers and NEC has been raised.

Most preterm babies fed human milk who subsequently develop NEC were also receiving these fortifiers, but the association between use of these products and the development of NEC remains unproven. One study has been performed showing that preterm babies fed breast milk augmented with a human milk-based fortifier are less likely to get NEC than those fed formula or human milk with cow's milk based fortifier.² This study however, did not compare only human milk fed infants, but combined the group receiving formula with those receiving human milk and cow's milk-based fortifiers.

In this case, the baby was fed only his own mother's milk and never received formula although he did receive standard, cow's

milk-based human milk fortifier. His growth was generally very slow and it is understandable that the clinical team was keen to increase his protein and caloric intake by adding fortifier. Fortification of human milk is necessary to achieve optimal growth and body composition in preterm infants and its use is standard. An association with the use of cow's milk-based fortifiers and NEC has not been established. Although there is a human milk-based fortifier available commercially, it is extremely expensive and until such time as the benefits are clearly established, no Canadian centres have made the switch to it.

One final point on causation may be germane. An association between red blood cell transfusion and NEC has been described in several publications in the past few years and transfusion-related NEC is becoming accepted by the medical community as a real entity. It appears that up to 25% of the cases of NEC that are seen in modern NICUs follow shortly after red blood cells transfusion³.

In the case reviewed, the baby received a red blood cells transfusion approximately 48 hours before the first signs of an abdominal problem (i.e. the loose stools). Although the interval between transfusion and onset of NEC was a little longer than described in the literature, it is possible that the baby developed a case of Transfusion-Related Acute Gut Injury (TRAGI). In March-April 2011, when the baby was in the NICU, TRAGI had not been fully accepted as a definite clinical entity and, other than avoiding blood transfusion when not indicated, there are no proven ways to prevent it.

Placing a baby "nil per os" [nothing by mouth] during transfusion has been suggested as a possible protective strategy and while many NICUs are doing so at present, this is not a universally accepted standard practice and questions remain about its application and utility.

b) Participation in Research Studies

The family questioned whether involvement in the GentleR study might have disadvantaged the baby and/or contributed to his death, and suggested that there may have been some impropriety in the recruitment process and study methods.

A review of the GentleR protocol revealed a number of differences from usual clinical practice, but there was no direct evidence that any of these practices would have led to the development of NEC. There was an increased tolerance for apnoeic/bradycardic spells that, in usual circumstances, would have prompted intubation and mechanical ventilation and it is possible that recurrent hypoxemia and bradycardia could have led to some degree of gut ischaemia, thus setting the stage for NEC. However, this association is theoretical and it is acceptable, general practice to continue to support preterm babies non-invasively in the face of fairly frequent apnoea as long as spells are not severe or continuous. It would be appropriate for the study steering committee and safety committee to examine this question and perform a general review of the family's complaints in order to ensure that there were no breaches of research ethics.

c) Notification of coroner

It would have been warranted and helpful if the members of the clinical team at the tertiary neonatal centre and the children's hospital had informed the coroner of the baby's death. It was evident that the family were very concerned about the clinical management and this would have been sufficient grounds to inform the coroner, even if the cause of death seemed clear.

An autopsy may have been helpful in reassuring the family that there were no underlying bowel or systemic problems that might have contributed to the baby's death and that the findings were consistent with the clinical diagnosis of severe NEC.

Recommendations:

1. Those overseeing the operations of the NICU at the tertiary neonatal centre should perform a thorough review of this case and the parents' complaints.
2. Health care providers are reminded that the coroner's office should be contacted when family members appear to have concerns about the care provided.
3. Neonatal health care providers are reminded of the possible adverse effects of red blood cell transfusion including Transfusion-Related Acute Gut Injury (TRAGI) and the need

to limit transfusions to situations in which there are clear indications. Consideration should be given to placing a transfused patient "nil per os" (NPO) [nothing by mouth] for several hours before and after the transfusion.

4. The committee recommends that the Regional Supervising Coroner (RSC) follow up with the hospital regarding the issues identified. If, in the opinion of the RSC, systemic issues persist, the RSC should consider conducting a Regional Coroner's Review with appropriate representatives from the hospital.

References

1. Schnabl KL, Van Aerde JE, Thomson ABR, Clandinin MT. Necrotizing enterocolitis: A multifactorial disease with no cure. *World J Gastroenterol* 2008;14(14):2142-2161.
2. Sullivan S, Schanler RJ, Kim JH et al. An exclusively human milk-based diet is associated with a lower rate of necrotizing enterocolitis than a diet of human milk and bovine milk-based products. *J Pediatr* 2010;156:562-7.
3. Blau J, Calo J, Dozor K, Sutton M, Alpan G, La Gamma EF. Transfusion-related acute gut injury: Necrotizing enterocolitis in very low birth weight neonates after red blood cell transfusion. *J Pediatr* 2011;158(3):403-9.

Case: 2012-N-2

OCC File: 2011-7492

History

The mother of the deceased infant was a 31-year-old G1P0 with an EDD of June 8, 2011 by ultrasound at 14 weeks six days gestational age. The mother was obese with a BMI of 31.6. Ultrasounds were normal for anatomy, but growth was in the 97th percentile at 24 weeks and in the 86th percentile at 33 weeks gestational age. Amniotic fluid levels were normal. Weight gain over the course of the pregnancy was 15 pounds. An oral glucose challenge test (OGCT) at 28 weeks was positive; however the two hour fasting 75g glucose tolerance test was negative.

On June 8, 2011 at 0515 hours, the mother presented to obstetrical triage. Two hours later, she was admitted in active labour with spontaneous rupture of membranes. Demerol was given upon admission and IV fentanyl was given twice in labour for sedation. At 1410 hours, the mother was fully dilated and started pushing. For the next thirty minutes there was either loss of contact or significant decelerations. There was no documentation of maternal heart rate and no request for a fetal scalp clip. At 1440 hours, the FHR tracing showed a baseline of 130-140 bpm which was considered to be a normal tracing.

Atypical FHR tracings started at 1525 hours. By 1533 hours, the ante-partum FHR tracings were persistent and as low as 60 bpm. The obstetrician on call was notified at 1545 hours and attended at 1550 hours. On examination, the mother was fully dilated; occiput transverse with caput and molding was at station 0. The FHR tracing was found to be "reactive and variable." The baseline was in the 120's with good variability and increases from baseline during contractions. There is no record of maternal heart rate at this time. The plan was to re-assess the mother in thirty minutes and proceed to a Caesarean section if no progress.

By 1604 hours, there were decelerations to 60 bpm. Oxygen was applied and the obstetrician on call was notified at 1610 hours and reviewed at 1620 hours. Consent for Caesarean section was obtained. The FHR tracing was abnormal. At 1630 hours, a fetal scalp clip was applied. At this time, the FHR was shown

to be in the 120's with no accelerations or decelerations. There was no documentation of maternal heart rate. At 1700 hours, the mother was moved to the operating room. At 1710 hours, while in the operating room, the fetal scalp clip was found to be "off." At 1728 hours, after spinal anaesthesia was established, the FHR was documented at 136-140 bpm by doptone and the anesthetic record shows the maternal heart rate to be 110 bpm.

The infant was delivered by Caesarean section at 1738 hours. The fetal head was noted to be in "deep occiput transverse arrest." The operative note reads, "fetal head was disimpacted with great difficulty in view of the presence of caput and molding, which made the procedure quite difficult."

The 4534 g infant was delivered and resuscitation started prior to the physician arriving despite being notified prior to the start of surgery. The physician was working a shift in the emergency room and arrived when the infant was approximately three minutes of age. Chest compressions and ventilation by bag and mask were already started. Heart rate increased (although bpm was not documented). By five minutes of age, the heart rate was in the 140s and respirations were weak at 40 per minute. Bilateral chest crackles, flat tone and acrocyanosis were noted. The infant was suctioned for "lots of clear fluids." Intubation was attempted, but not performed as the epiglottis was seen but, "could not visualize the tip of ET [endotracheal] tube so no attempt at intubation was done." Spontaneous respirations started at 1800 hours. A "fair amount of overlapping of sutures but no obvious bruising" was noted.

Apgar scores were noted as 1, 4 and 7 at one, five and ten minutes of age, respectively. Cord gases were: arterial pH <6.80, pCO₂ 94.0, pO₂ 17.0 and; venous pH 6.81, pCO₂ 36, pO₂ 93, HCO₃ 5.7, BE 29.1, O₂ sat 85, Total CO₂ 7.

The infant was taken to the nursery where oxygen was weaned to room air, but the tone remained poor. Mean arterial pressure was noted to be 43 so an umbilical vein catheter was placed and an intravenous (IV) of 10% dextrose was started after a glucose reading of 3.1. Blood cultures were drawn, then IV ampicillin and gentamicin were started. At 1930 hours, the infant was "stabilized" so the physician returned to the emergency room.

Capillary gases:

Time	pH	pCO ₂	pO ₂	HCO ₃	TCO ₂	BE	O ₂ sat
1952	6.99	28.0	66	6.7	7.6	-23.8	78
2140	7.05	30.0	52	8.3	9.2	-21.1	67
0039	6.99	44.0	50	10.6	12.0	-20.0	60
0212	7.06	27.0	58	7.6	8.4	-21.4	75

At 0130 hours, the physician was called and returned to reassess the infant since the capillary gas showed a pH of 6.99. The tertiary level NICU was called and a dopamine infusion was started. Arrangements were made for transfer. Two hours from the time of the initial call, the tertiary level transport team arrived to attend to the infant. In that interval, the infant started to have apneic spells and seizures. Intubation was again attempted, but was not successfully performed.

The transport team arrived and the infant was intubated and stabilized prior to transfer. At the tertiary level hospital, despite extensive resuscitation efforts, the acidosis persisted. Disseminated intravascular coagulopathy (DIC) developed with multi-organ system failure. Care was withdrawn after extensive consultation with other specialties, palliative care and the parents.

Post mortem

Perinatal death due to hypoxia at delivery. Significant contributing factors were: Skull fracture with evidence of traumatic brain injury and disseminated intravascular coagulopathy.

Discussion

The infant died of severe hypoxia. Skull fracture at the time of delivery was a significant contributing factor. After an aggressive resuscitation where an airway could not be obtained, the infant continued to require acute care without improvement of the metabolic state. Communication with the tertiary centre was delayed and was not obtained until eight hours after delivery.

Once the mother was fully dilated and pushing, there was a significant period of time when it is uncertain if there was loss

of contact with the FHR or if the tracing was maternal. Then, after a period of decelerations to 60 bpm, there was an abrupt improvement. It is important to ensure that the heart rate being monitored is fetal and not maternal, especially when there are abrupt changes in the FHR tracing.¹

Consent for Caesarean section was obtained. The rationale was the persistent late decelerations to 60 bpm. When a fetal scalp clip was applied ten minutes later, the heartbeat reported was 120 bpm with no accelerations or decelerations. Again, there was no documentation of maternal heart beat to determine which heartbeat - maternal or fetal - was being monitored.

The time from obtaining consent to arrival in the operating room was 40 minutes. It was another 28 minutes to delivery. In this time, it was noted that the fetal scalp clip was not attached and therefore, the FHR was not being monitored. The SOGC Policy Statement, "Statement on Wait Times in Obstetrics and Gynaecology"² states that an "Emergency Caesarean section must be considered and implemented whenever acute fetal compromise is suspected and vaginal delivery is not imminent." The proposed wait time benchmark for emergency Caesarean section is "within approximately 30 minutes."² In this case, the time of 68 minutes to delivery falls well outside that benchmark.

When there is a long second stage (in this case three hours) and a suspected macrosomic fetus, a difficult delivery (even at Caesarean section) should be anticipated. At Caesarean section, when the fetal head is impacted deep into the pelvis, it may be difficult to deliver without extensive trauma to the lower genital tract and fetal head. It is important to anticipate this potential problem and have an approach for delivery. Robert L. Barbieri suggests delivery either by reverse breech extraction or an assist from a vaginal hand.³

Even with time delay to delivery, the physician responsible for the neonatal resuscitation was not present at the time of delivery. Extensive resuscitation was required and intubation could not be performed. There was no documentation that assistance with the intubation was requested from the anesthetist in the operating room. Given the extensive resuscitation required, early communication with an appropriate referral centre to discuss management of the neonate would be appropriate.

Recommendations:

1. Obstetrical care providers are reminded that:
 - a) Application of a fetal scalp clip should be considered when it is difficult to monitor fetal heart in labour; it is important to ensure that the heart rate being monitored is fetal and not maternal. This is especially important when there are abrupt changes in the FHR tracing;¹
 - b) Efforts should be made to proceed to “emergency” Caesarean section in a timely manner;
 - c) When the fetal head is known to be well down in the pelvis and difficult delivery is anticipated, consideration should be given to manoeuvres that will facilitate delivery in order to decrease the risk of trauma to the fetal head at delivery;
 - d) If intubation is deemed to be necessary but cannot be successfully performed, assistance should be sought from another skilled operator in the use of laryngeal mask airway;
 - e) When active resuscitation is required and severe acidosis and encephalopathy are present, immediate consultation with a pediatrician at the referral centre or tertiary centre should be undertaken for consideration of total body cooling and transfer.
1. A Regional Supervising Coroner’s Review should be conducted on the circumstances surrounding the death of this infant.

References:

1. Simpson, K, “Avoiding Confusion of Maternal Heart Rate with FHR during Labour”. American Journal of Maternal Child Nursing: July/August 2011 - Volume 36 - Issue 4 - p 272.
2. SOGC Policy Statement, “Statement on Wait Times in Obstetrics and Gynaecology.” No. 204, March 2008.
3. Barbieri, RL, “Difficult fetal extraction at cesarean delivery: What should you do?” obgmanagement.com, January 2012, Vol. 24, No. 1.

Case: 2012-N-3

OCC File: 2011-1648

History

The mother of the deceased infant was a 26-year-old G3P1 with an EDD of February 11, 2011 by first trimester ultrasound. Routine prenatal laboratory investigations and second trimester ultrasound were normal. Prenatal genetic screening testing was declined. A second trimester ultrasound was normal and GBS testing at 33 weeks gestation was negative.

Her past obstetrical history included one term pregnancy delivered vaginally of a 7 pound, 7 ounce male infant in 2005. Her past medical history was non-contributory.

The antenatal course was uncomplicated.

Course in Labour and Delivery

The mother was admitted to Hospital A on February 9, 2011 at 39 weeks five days gestation for induction of labour because of maternal discomfort. Dinoprostone (Prostin®) gel was administered at 0800 hours with the cervix 2 cm dilated. At 1115 hours, the cervix was 2-3 cm dilated and artificial rupture of membranes was performed for clear fluid. Labour became established at 1400 hours. Oxytocin was started at 1513 hours. The cervix was 5 cm dilated and 100% effaced with the vertex at spines -2. She became uncomfortable with her labour and an epidural was started at 1730 hours. She reached full dilation at 1900 hours. Her temperature was 38.1C. The FHR was in the 150-160's. She continued to have a low grade fever and the FHR increased to 160-200 bpm during the second stage of labour.

At 1940 hours, she was uncomfortable and crying and was not pushing well. At 2024 hours, she stated that she could not push anymore and was "begging" for help. The obstetrician attended and assessed the pushing efforts. The decision was made to proceed with assisted delivery using the Kiwi vacuum extractor.

The vacuum extractor was applied at 2033 hours at station spines +1/+2 and a medio-lateral episiotomy was made. At 2036 hours, the vacuum extractor was abandoned. The respiratory technician and special care nursery (SCN) were called to attend.

Forceps were applied and at 2042 hours she was delivered of a 3080 g female infant in the occiput posterior position. Apgars were 5, 7 and 8 at one, five and ten minutes. Cord gases were not done. The baby was admitted to the special care nursery.

Telephone orders were received from the paediatrician at 2200 hours for "continued observation in the SCN, start IV D10W @ 8cc/hr, CBC and blood cultures, IV ampicillin and gentamycin and O2 to keep sat >90%." The paediatrician subsequently attended. A bolus of normal saline 30cc over 15 minutes was ordered at 0030 hours and phenobarbital was given. The paediatrician's note at 0100 hours indicated that, "at 2 hours of age the baby was noted to be pale, tachycardic and floppy. Oxygen saturation was low but responded to CPAP [continuous positive airway pressure] given by the RT [respiratory technician]." When the paediatrician arrived, a CBC and culture had been done and an IV started. The nurse's notes indicated that this was done at 2330 hours. The baby was noted to be pale and shocky with a weak cry and felt dehydrated with a large "caput/cephalohematoma."

It was believed that the baby was experiencing asphyxia secondary to the difficult delivery. The plan was to continue IV hydration, IV antibiotics, obtain an ultrasound of the head in the morning and to feed when ready.

At 0230 hours, the baby arrested and a "code pink" was called. The baby was initially attended to by the emergency room physician. CPR was initiated. The baby was given 0.3cc 1/10,000 epinephrine for asystole and a pulse was detected. The baby was given a bolus of 80cc normal saline. The paediatrician was notified and arrived at 0300 hours. Repeat hemoglobin drawn at 0318 hours was 62 gm down from 117 gm at 2330 hours. Blood gas drawn at 0328 hours showed a pH of 6.80. A dopamine drip was started. The baby was also given epinephrine and bicarbonate. The baby was transfused with 10cc of blood at 0433 and 0435 hours. The transport team from the children's hospital was contacted. Resuscitative efforts continued.

The transport team arrived at 0531 hours. The baby received another 30cc of blood at 0544 hours. The baby again became asystolic at 0600 hours. At that time, the transport team fellow discussed the situation with the physician at the children's hospital and resuscitative efforts were stopped. The paediatrician declared the baby deceased at 0810 hours on February 10, 2011.

Post mortem

The significant finding at autopsy was a large bilateral subgaleal hemorrhage, more marked on the left side. There were multiple minor abrasions including: left mid-lateral 1.3 cm X 0.2 cm; right-lateral 0.8 cm X 0.3 cm; along posterior midline 0.5 cm X 0.2 cm; and along midline forehead/brow 1.1 cm X 0.2 cm and an abrasion above the right eyebrow 0.4 cm x 0.2 cm.

The cause of death was hemorrhagic shock due to subgaleal hemorrhage due to forceps/vacuum assisted vaginal delivery.

Discussion

This infant died from hemorrhagic shock resulting from a large subgaleal hemorrhage. The incidence of subgaleal hemorrhage is approximately 4/10,000 spontaneous vaginal births versus 59/10,000 vacuum-assisted deliveries. Potentially, 20-40% (50-100cc) of the newborn's blood volume can be lost into this space resulting in pallor, tachycardia and shock. The mortality rate is 12-14%. Early recognition is vital if mortality is to be prevented. Neonates who have experienced a difficult operative delivery, particularly with vacuum extraction, should be monitored closely.

Despite much research, many health care workers have limited knowledge of subgaleal hemorrhage. Subgaleal hemorrhage is most often associated with vacuum extraction and forceps delivery, but many occur spontaneously. Outcome can be improved by early recognition of subgaleal hemorrhage and differential diagnosis of cephalohematoma and caput. Proper therapy by transfusion and correction of coagulopathy are major determinants of successful outcome. Recommendations by many authors include: screening of high risk infants, monitoring for 24 - 48 hours (including vital signs at two, four and six hours); head circumference measurements at birth and four hours; and CBC/Haematocrit at four and six hours.

It appears that in spite of the delivery history, monitoring by the special care nursery was limited. The physical examination was also limited, particularly with regards to cranial swelling. The diagnosis of subgaleal hemorrhage was not considered and therefore monitoring was not exercised. Vital signs did not include blood pressure monitoring and medical documentation

of perfusion and neurological assessment was limited. Birth asphyxia and possible sepsis were considered, although there was no significant evidence. There was also a delay in blood work resulting in a delay in blood transfusion.

It is not clear from this review whether the hospital involved had a protocol in place for monitoring neonates after a difficult or failed vacuum extraction or whether the caregivers involved were aware of the risk. Although the paediatrician was aware of the difficult operative delivery when the baby was first attended, the combination of pallor, tachycardia and a large "caput/cephalohematoma" did not lead to the consideration of subgaleal hemorrhage. In addition, the hemoglobin result of 117 gm done at 2330 hours did not appear to have been reported in a timely fashion, followed up on, or appreciated for its significance.

The indication for operative delivery must be compelling. The indications given in this case included maternal fever and fetal tachycardia in the second stage of labour. Review of the FHR tracing showed a fetal tachycardia between 160 -200 bpm developing at approximately 1948 hours. The highest maternal temperature recorded during this time was 38.1C. In addition, there were complex variable decelerations. Given this pattern, the decision for delivery was appropriate. It is not clear from the record whether the baby's posterior position was appreciated at the time of the procedure. The station was recorded at +1-+2 when the vacuum was applied. Further details of the procedure, such as the pressure used, the number of pulls and whether there were any pop-offs, were not documented.

It cannot be determined from this review whether the vacuum was applied in the appropriate position. Improper positioning increases the risk of trauma. It does not appear that the operator persisted with this approach for an undue length of time before changing to forceps. The forceps procedure does not appear to have been difficult, although again, the documentation is minimal.

Given the indications for operative intervention and the difficulty encountered, it would have been prudent to have had the paediatrician in attendance at the time of delivery. Although a written note by the obstetrician at 0500 hours describing the delivery indicated that cord gases were obtained, the delivery

summary does not indicate that they were done and there are no results documented on the chart.

Recommendations

To Obstetrical and Neonatal Care Providers:

1. Obstetrical and neonatal care providers are reminded of the risk of subgaleal hemorrhage with vacuum extraction, forceps or difficult delivery.
2. Obstetrical care providers are reminded of the need to thoroughly document operative interventions.
3. Obstetrical care providers are reminded to obtain cord blood gases at delivery.
4. Neonatal care providers are reminded to document a complete and full newborn assessment on initial examination in a timely fashion.

To Hospital A:

5. The hospital should review/establish a protocol for monitoring newborns delivered by vacuum extraction, forceps or difficult delivery.
6. The hospital should review/establish its policy regarding indications for the attendance of a paediatrician at delivery.
7. The hospital should review its policy for communicating critical laboratory results.
8. The hospital should perform a quality of care review of this case.

Case: 2012-N-4

OCC File: 2011-7608

History

The mother of the deceased infant was a 39-year-old primiparous woman. The deceased infant was delivered by emergency Caesarean section at 37 weeks gestation. The neonate, with unexpected hydrops and congenital anomalies, died two hours after birth.

Antenatal Course

The first trimester prenatal screen for aneuploidy was normal and the nuchal translucency was normal. The morphology ultrasound at 19 weeks gestation was normal; however the face and lower extremities were not well seen. A limited ultrasound one month later demonstrated a normal face and feet. An additional limited ultrasound was done at 32 weeks gestation which showed normal growth, fluid and wellbeing. The biophysical profile (BPP) was 8/8.

The mother developed Bell Palsy and was treated with prednisone in early second trimester. The glucose challenge test was positive, but the two-hour glucose tolerance test was normal.

The mother experienced two or three episodes of vaginal spotting in the third trimester. She was assessed at a walk in clinic at 35 weeks gestation and was told that she had a cervical polyp. Ten days later, at 36.5 weeks gestation, she was assessed in obstetrical triage and monitored for several hours prior to discharge. The following day, she was seen by her obstetrician. She reported the baby was active, but less so over the previous week. She was found to be 2-3 cm dilated.

There were no risk factors identified in the mother's medical or genetic history.

Intrapartum course

On June 23, 2011 at 37 weeks gestation, the woman was admitted to Hospital A at 1107 hours in active labour, at 7 cm dilation. The FHR tracing showed decreased variability, no accelerations and

some early decelerations. An amniotomy at 1155 hours was performed and thin meconium was observed. She progressed quickly to 10 cm dilation by 1247 hours and began to push by 1300 hours. In the second stage, the tracing showed worsening variable decelerations. At 1400 hours, the presenting vertex was left occipitoposterior (LOP) at sp +2-3 cm.

Because of the decelerations, the vacuum was applied and over two to three contractions there was good descent to the perineum. As the head was crowning, an episiotomy was made. However, at that point, there was "significant concern that the shoulders would not descend and the head was pushed up" and they proceeded to an emergency Caesarean section under general anesthetic.

At 1414 hours, a male baby weighing 4153 g was delivered. The Apgar scores were 1, 3 and 4 at one, five and ten minutes. The cord gases were normal and were recorded as arterial pH 7.17 and BE -6.

Shortly after birth, a "code pink" was called. The baby was extremely edematous and hydrops fetalis was suspected. It was a difficult intubation however it was successful after multiple attempts. The baby was very difficult to ventilate and remained bradycardic with poor perfusion. Central lines were placed, however the baby remained hypotensive and acidotic despite vasopressors, fluid boluses, sodium bicarbonate, a pleural tap and placement of a chest tube. Contact was made with the children's hospital, but the baby was too unstable for transport and died about two hours after birth.

Post mortem

The findings at autopsy included: severe fetal hydrops, dysmorphic facies, complex congenital heart abnormality (including VSD with overriding aorta, aortic ring with pre-ductal coarctation, tricuspid and mitral valve dysplasia, cardiac hypertrophy and ventricular trabeculations with numerous false tendons), tracheal stenosis, pulmonary hypoplasia and normal male karyotype.

Positive newborn screen indicated a biotinidase deficiency, low normal, likely due to "poor sample integrity."

Discussion

The mother of the deceased infant presented at 37 weeks gestation in spontaneous labour and advanced cervical dilation. The FHR was atypical, but she progressed quickly in labour. She underwent an emergency Caesarean section following a failed vacuum attempt and concern about shoulder dystocia. The cord gases were normal. The neonate was hydropic and deteriorated despite efforts to resuscitate. The time frame over which the fetal hydrops developed is unknown. There were no antenatal risk factors or clinical evidence of the hydrops.

The autopsy revealed significant cardiac abnormalities which may have resulted in the hydrops. The fetal ultrasounds did not report any evidence of congenital heart disease. The technologist's work sheet from the morphology scan indicated the four chamber heart view was normal, but there was no assessment of the cardiac outflow tracks. Other investigations for the etiology of the hydrops, including neonatal chromosomes and an infectious screen, were negative. The newborn was not anemic.

In general, the recurrence risk of cardiac abnormalities is 1-5 %.

Recommendations

To prenatal ultrasound providers:

1. Prenatal ultrasound providers are reminded of the SOGC and American Institute of Ultrasound Medicine (AIUM) Guidelines requiring full second trimester morphology assessments and reporting.

To obstetrical care providers:

2. Mothers who have suffered fetal or neonatal loss due to cardiac abnormalities should be offered fetal echocardiography in subsequent pregnancies.

Case: 2012-N-5

OCC File: 2011-794

Antenatal History

The mother of the deceased neonate was a 39-year-old G3P0 with an EDD of January 23, 2011 by first trimester ultrasound. Routine prenatal laboratory investigations were normal. Prenatal genetic screening testing was declined. Routine second trimester ultrasound was normal. Second trimester glucose challenge test was normal and GBS was negative. The antenatal course was uneventful.

Her past medical history included two spontaneous early pregnancy losses. The couple was in the process of adopting a child when she became pregnant for the third time. She was treated with progesterone (Prometrium©) early in the pregnancy. She had a history of asthma for which she was prescribed budesonide inhalation and albuterol sulfate.

Family history was non-contributory.

Course in Labour and Delivery

The woman was admitted to the labour and delivery unit of Hospital A at 0032 hours on January 20, 2011 at 39 weeks and four days gestation. Contractions had started at 2000 hours on January 19, 2011. At the time of presentation, she was 3 cm dilated and 90% effaced. Membranes were intact and the FHR was normal. She was assessed by her physician at 0045 hours.

She was given nitrous oxide at 0130 hours. The cervix was 5 cm dilated at 0155 hours. She was given meperidine hydrochloride 25mg with dimenhydrinate 25 mg intravenously at 0200 hours. At 0325 hours she was 8 cm dilated. She continued using nitrous oxide and meperidine hydrochloride for pain relief. She was re-assessed by her physician at 0604 hours. The cervix was 9 cm dilated and an artificial rupture of membranes was performed for clear amniotic fluid. She was fully dilated at 0745 hours. The fetal heart tracing was normal during the first stage of labour.

Pushing commenced at 0756 hours. She tried different positions, initially squatting on all fours, then re-positioned sitting in bed, standing and sitting on the toilet and finally back in bed. By 1220 hours she was very tired. At 1300 hours the decision was made to proceed to an epidural.

After insertion of the epidural, there was a FHR deceleration at 1345 hours. This resolved with patient re-positioning and oxygen.

The epidural block was to T5. Maternal vital signs were stable, but she could not feel her contractions. She was re-assessed by her physician at 1455 hours. The vertex remained at the same station. She slept, then began to feel the contractions at 1547 hours. At 1600 hours, she was feeling rectal pressure, but was afraid to push. At 1618 hours, she was given oxygen by mask and repositioned because of minimal FHR variability. At 1626 hours, the FHR was 85 bpm and returned to baseline after a vaginal examination. She was assessed by her physician at 1640 hours and after a vaginal examination was performed, she was encouraged to push, but she refused due to exhaustion. After some discussion, it was agreed that she would start pushing at 1645 to 1700 hours.

She continued to receive oxygen by mask. At 1650 hours she was positioned on all fours and started pushing. At 1755 hours meconium was noted. At 1810 hours she was positioned on the toilet to try to facilitate pushing. At 1831 hours the FHR was 77-100 bpm after a contraction. Preparations were made for vacuum delivery.

The vacuum was applied at 1833 hours and a pop-off occurred. After re-application at 1836 hours and three pulls, she was delivered over a medio-lateral episiotomy of a 3420g male infant. The cord was around the neck once. Apgars were 0, 0 and 0 at one, five and ten minutes. A sample of umbilical cord was obtained and sent, but cord blood gases were not done. A "code pink" was called at 1850 hours. Bag and mask ventilation and cardiac compressions were commenced. An ETT was inserted by the physician at 1851 hours and epinephrine was administered. An umbilical venous catheter was inserted at 1856 hours. Resuscitation efforts continued.

The paediatrician arrived at 1923 hours. The ETT was replaced and position confirmed by CO2 monitor. Bicarbonate was given intravenously. Compressions were stopped at 1930 hours when the heart rate was in the 130's.

A capillary gas at 2001 hours showed a pH of 7.00 and bicarbonate 17.5. Subsequent gases showed increasing metabolic acidosis. The transfer team from the children's hospital was called at 2109 hours. Tremors were first noted at 2120 hours. The team from the children's hospital arrived at 2232 hours. Resuscitation continued until 0300 hours at which time the baby was pronounced.

Post mortem

The MPDRC sought input from an external pathologist for a detailed review of post mortem findings.

The findings from the external pathologist indicated that:

The main pathology is in the placenta where there is acute chorioamnionitis and a well-established necrotizing fetal inflammatory response. There do not seem to be established changes in the brain and stress effects are not seen in the adrenals or thymus implying that the terminal insult was acute. The increased mucin observed in the tracheobronchial tree and vermiform appendix is of uncertain significance and does not appear to have played a role in the baby's death.

Conclusion: placenta showing acute chorioamnionitis with a marked fetal inflammatory response.

Discussion

This infant died from hypoxic-ischemic encephalopathy resulting from intrapartum asphyxia during a prolonged second stage of labour.

The second stage of labour lasted in excess of 10 hours. There was no progress in the second stage of labour between 0745 hours and 1210 hours. The options of Caesarean section, vacuum extraction or an epidural with a plan to re-start pushing after a period of rest were discussed. The latter was chosen. There is no documentation of this discussion in the progress notes. There is no documentation in the record indicating whether there had been an assessment of the “powers, passageway or passenger” to provide a rationale as to why the latter option held some prospect for a successful vaginal birth. The implication from the notes is that the issue was primarily due to the “powers” i.e. maternal pushing efforts. Other factors such as the strength of contractions, the position and attitude of the vertex and a clinical assessment of the size of the baby relative to the pelvis, should have been considered and documented, particularly when the second stage of labour is allowed to extend well beyond the guidelines.

Review of the FHR tracing indicates the development of an abnormal tracing shortly before 1650 hours with no documented return to normal at any time leading up to delivery. The mother was assessed by the attending physician at 1640 hours, just prior to the development of the abnormal FHR tracing and not again until 1831 hours at which time delivery was expedited by vacuum

extraction. There is no indication from the progress notes that the physician was notified about the abnormal FHR tracing prior to 1831 hours. However, if the second stage of labour is allowed to be prolonged beyond the guidelines, and given the increased risk of FHR abnormalities in this setting, it would have been prudent for the obstetrical care provider to take a much more “hands on” approach by attending and assessing the situation frequently.

The baby's course and capillary gases were consistent with intrapartum asphyxia, however no cord gas results were obtained.

Recommendations

1. Obstetrical care providers are reminded of guidelines concerning the length of the second stage of labour.
2. Obstetrical care providers are reminded of the need to clearly document the rationale for a plan of management that deviates from the guidelines.
3. Obstetrical care providers are reminded of the guidelines for the identification and management of abnormal FHR patterns.¹
4. Obstetrical units are reminded to obtain cord blood gases at the delivery of compromised neonates.
5. The committee recommends that the Regional Supervising Coroner (RSC) follow up with the hospital regarding the issues identified. If, in the opinion of the RSC, systemic issues persist, the RSC should consider conducting a Regional Coroner's Review with appropriate representatives from the hospital.
6. The parents of this infant should be advised to seek genetic counselling regarding the possibility of Cystic Fibrosis.
7. Hospital A should perform an Internal Review of this case.

References:

1. Liston R, Sawchuck D, Young D; Society of Obstetrics and Gynaecologists of Canada; British Columbia Perinatal Health Program. Fetal health surveillance: antepartum and intrapartum consensus guideline. J Obstet Gynaecol Can. 2007 Sep;29 (9 Suppl 4):S3-S6.

Case: 2012-N-6
OCC File: 2010-15028

Antenatal History

The mother of the deceased infant was a 26-year-old G2P1 with an EDD of November 9, 2010. Routine prenatal laboratory investigations and second trimester ultrasound were normal. A second trimester glucose challenge test was normal. She was GBS negative. Her antenatal course was uneventful.

She had had one previous term pregnancy delivered vaginally of an 8 lb female infant at 41 weeks in 2009. Her past medical health and family history were non-contributory.

Course in Labour and Delivery

The woman was admitted to the obstetrical unit of Hospital A on November 16, 2010 for induction of labour for postdates at 41 weeks gestation. The cervix was long and closed. A non-stress test (NST) was normal. Dinoprostone (Cervidil®) was inserted at 1600 hours and she was subsequently discharged home. She returned at 2300 hours in labour. At 2340 hours, the cervix was 2 cm dilated, 90% effaced with the vertex at spines-3. At midnight, the contractions were very intense and the cervix was 4 cm dilated. The dinoprostone was removed at 0010 hours and an epidural was placed at 0030 hours.

The external electronic fetal monitor showed late decelerations and the pattern persisted despite patient re-positioning, IV hydration and maternal oxygen administration. She was fully dilated at 0100 hours. An artificial rupture of membranes was performed for clear fluid and a fetal scalp clip was applied at 0107 hours. The vertex was occiput posterior at station +1. She began pushing at 0108 hours.

As there was an abnormal FHR tracing, the parents were advised that the baby needed to be delivered quickly and the use of forceps was discussed. The parents were informed of the increased risk due to the high station. The delivery summary indicated the application was midforceps while other references in the medical record indicated a high forceps application.

The forceps procedure began at 0114 hours. Despite the high station, descent was obtained with two pulls and moderate traction. It does not appear from the record that a rotation was performed. She was delivered at 0117 hours of a 3850 g female infant. Apgars were 1, 1, 3 and 7 at one, five, ten and twenty minutes. Venous cord gas was pH 7.1, PCO₂ 71 and BE -8.6. The heart rate was 80 bpm. Positive pressure ventilation (PPV) was administered and the heart rate was >100bpm at 0119 hours.

The baby remained flaccid with no respiratory effort. A "code pink" was called at 0122 hours and the obstetrician intubated the baby. Chest compressions were started at 0123 hours. The heart rate was again over 100 bpm at 0127 hours and the paediatrician arrived at 0128 hours. The heart rate remained over 100 bpm and at 0132 hours, the baby was making respiratory efforts. The ETT was noted to be in the esophagus and was removed. Multiple attempts to obtain blood and start an IV were unsuccessful. The baby was noted to be bleeding from the attempted IV sites.

Blood work at 0206 hours showed blood sugar 7.4, hemoglobin of 182, WBC 16.9 and platelets 83,000. The baby received positive pressure ventilation (PPV) through a mask before being placed on CPAP at 40 minutes of age. A chest x-ray at 0200 hours questioned developing Respiratory Distress Syndrome. At 0206 hours, the infant's pulse was 171, respirations 40 and O₂ saturation 97%. Oxygen was discontinued at 0213 hours and O₂ saturation was 98% on room air. An umbilical vein catheter was placed at 0250 hours and the baby was given a 40cc bolus of normal saline at 0325 hours. Umbilical catheter gas was pH 7.29, PCO₂ 34, HCO₃ 16 and BE -9.3. Hematoma at the site of forceps bruises was noted to be expanding. Blood culture and CBC were drawn and antibiotics were started.

A chest x-ray at 0400 hours showed worsening Respiratory Distress Syndrome. At 0420 hours, the children's hospital was contacted and a team arrived at 0508 hours. The baby was found to be pale and hypotonic. Fresh frozen plasma was started and the infant was transported to the children's hospital at 0550 hours.

Upon arrival at the children's hospital at 0605 hours, the baby was noted to be gasping. The baby was re-intubated and given a platelet transfusion in addition to fresh frozen plasma. The baby

had no suck or gag reflex, was noted to be posturing while being handled and was otherwise non-responsive. Bedside EEG was grossly abnormal with no electrical activity. A head ultrasound showed extensive right and third ventricle hemorrhages, right hemispheric hemorrhage, Doppler flow resistance indicative of high intracranial pressure and evidence of ischemic change. The EEG and head ultrasound findings were discussed with the parents and due to the poor prognosis, aggressive resuscitative measures were stopped. The baby died at 2200 hours on November 17, 2010.

Post mortem

Post mortem examination revealed:

1. Features of anoxic injury. Neuropathology showed generalized anoxic-ischemic encephalopathy, moderate to severe.
2. Features attributable to coagulopathy. The central nervous system showed hemorrhages at various levels and in various structures including intracerebral, intracerebellar and subarachnoid. There were cutaneous ecchymotic/petechial rashes identified. Submucosal hemorrhages were seen focally within the small intestine. The lungs showed patchy septal, pleural and parenchymal hemorrhage on histology.
3. Features attributable to birth trauma. A cephalohematoma measuring 4X2 cm was noted on the right parietal bone. There was a forceps impression on the skin in the right suboccipital and neck region with an underlying intramuscular and soft tissue hematoma. There was a subdural hematoma along the right tentorium.

The cause of death was anoxic brain injury with concomitant coagulopathy and hemorrhages.

Discussion

This baby died as a result of a combination of insults arising during labour and delivery and during the neonatal period.

Labour was induced electively at 41 weeks; this was not a compelling indication for induction. The fact that the cervix was

unfavourable adds further to the risk/benefit determination.

Labour onset occurred a few hours after the insertion of the cervical ripening agent. The contractions were strong and the labour advanced quickly, progressing from 2 cm at 2340 hours to full dilation at 0100 hours. The FHR tracing became abnormal around midnight and deteriorated further after the epidural was placed at 0030 hours.

The development of FHR abnormalities during the first 30 minutes after epidural analgesia is well recognized. Obstetrical care providers should consider the effect this may have on a fetus already demonstrating compromise on the FHR tracing. There is evidence that these abnormalities are due to uterine hypertonus and this would further compromise placental gas exchange already impaired by the intense contractions. Intrauterine resuscitation with repositioning, IV hydration and maternal oxygen were tried, but were unsuccessful.

Given the proposed mechanism of fetal compromise in this setting, a quick acting tocolytic such as nitroglycerin spray (Nitrospray®) or IV terbutaline could have been considered. It is recognized that these items are not always readily available in obstetrical units. The venous cord pH of 7.1 indicated some degree of fetal acid-base abnormality at birth, however the anoxic insult leading to the findings at autopsy may have occurred predominantly in the postnatal period.

Persistence of an abnormal FHR tracing necessitates delivery as quickly and safely as possible. Vaginal delivery, if deemed safe and feasible, is preferable as this would be the most expeditious route. As success cannot always be assured in this setting, simultaneous preparations should be made for Caesarean section. In this case, the obstetrician identified the need for immediate delivery and made a clinical determination that it was safe to proceed with a forcep procedure even though it was acknowledged that the high station increased the risks. The dictated note suggested the position was correctly identified and the procedure was not as difficult as anticipated given the station. The location of the forcep marks and the cranial trauma were not consistent with the narrative. The record does not indicate that preparations were being made for Caesarean section in the event that the forcep procedure was not successful.

Apgar score was 3 at ten minutes and the baby did not respond to initial resuscitation. The low score was due to low tone, poor response to stimulation and no spontaneous respirations; these are signs of serious central nervous system (CNS) depression. The respiratory effort was delayed to 15 minutes, also consistent with CNS insult. The ETT was noted to be in the esophagus, further compromising respiratory support.

There was no documentation of blood pressure, perfusion and cardiac output which was critical for assessment to guide treatment. The baby received only one bolus of sodium chloride which appears to be inadequate for a baby who was most likely hypotensive.

The coagulopathy further added to the baby's demise as a result of spontaneous bleeding and aggravation of the traumatic injuries. Thrombocytopenia was identified on CBC at 0144 hours, some 27 minutes after delivery. There are a number of causes of neonatal thrombocytopenia including, but not limited to, anoxia and DIC. The mother did not have a history of thrombocytopenia. Treatment measures for DIC were not initiated until approximately three hours after the CBC showed thrombocytopenia.

The team from the children's hospital was not notified until the baby was approximately three hours of age. This was a significant delay.

Recommendations

1. Obstetrical care providers are reminded that the indication for induction of labour must be compelling.
2. Obstetrical care providers are reminded that FHR abnormalities may occur shortly after the administration of epidural analgesia and to be prepared to provide intrauterine resuscitation, including the possible use of quick acting tocolytics, or timely delivery.
3. Obstetrical care providers are reminded that the initiation of epidural analgesia in the setting of FHR abnormalities may further compromise the status of the fetus.
4. Obstetrical care providers are reminded that preparations should be made simultaneously for Caesarean section when a forcep procedure is indicated for an abnormal FHR tracing.
5. Obstetrical care providers are reminded to have personnel skilled in neonatal resuscitation in attendance before delivery when fetal compromise is anticipated.
6. Neonatal caregivers are reminded to follow Neonatal Resuscitation Guidelines with particular attention to intubation and methods to ensure correct placement of endotracheal tubes.
7. Neonatal caregivers are reminded that response to treatment should be monitored with blood gases in addition to neurological assessment.
8. Neonatal caregivers are reminded that volume expansion may be crucial in the treatment of a compromised baby who fails to respond to initial resuscitation procedures.
9. Neonatal caregivers are reminded that calls to tertiary treatment centres should be placed as early as possible when dealing with a compromised neonate.

Case: 2012-N-7
OCC File: 2011-7385

Clinical History

The mother of the deceased infant was a 37-year-old gravida 1 with an EDD of July 10, 2011. She was followed by her family doctor in the family practice unit at Hospital A.

All appropriate prenatal blood testing was done including glucose challenge test at 28 weeks; all tests were normal. A second trimester ultrasound scan at 19 weeks showed normal anatomy. There was occasional irregular heart beat that led to fetal echocardiogram and consultation with pediatric cardiology. There was a diagnosis of occasional isolated premature atrial complexes. This was felt to be of little clinical significance. The caregivers were advised to monitor the fetal heart and only follow-up if there were increasing concerns, of which there were none.

There were frequent follow-up assessments that were documented on the Prenatal I and II records. A BPP was performed at 28 weeks gestation due to clinical assessment of large for gestational age. This, and a follow up at 34 weeks showing an estimated fetal weight (EFW) in the 73rd percentile, was otherwise unremarkable.

The woman was seen at her last prenatal visit on June 8, 2011 at 35 weeks. A GBS swab was done and was subsequently found to be positive. During the prenatal visits, the woman's blood pressure was borderline elevated, although never to a point where treatment would have been warranted.

The woman was admitted to Hospital A in the early morning of June 18, 2011 at 36 weeks and six days gestation due to premature rupture of membranes. Her blood pressure was 136/91. Pregnancy Induced Hypertension bloods were done and were normal. Intravenous penicillin was initiated due to her positive GBS status.

By approximately 1200 hours, five hours after admission, labour had still not been well established. An obstetrical consultation

was obtained for oxytocin augmentation of labour. Fetal heart tracings up until this point were normal.

Continuous external fetal monitoring was performed. The baseline fetal heart maintained in the 120-130 bpm range with normal accelerations and occasional short variable deceleration associated with contractions. At 2020 hours, there was a deceleration for approximately three minutes. The attending staff was called and the woman was assessed to be dilated 8 cm in a right occiput transverse position. At the time of assessment, the fetal heart had returned to normal and the oxytocin had been put on hold. From this point, the fetal heart baseline was in the 150 to 160 bpm range.

Over the next hour, there were recurrent late decelerations, but with recovery by the end of the contraction. From 2130 hours, the fetal heart pattern resumed a normal and reassuring pattern until approximately 2315 hours when recurrent deep decelerations were present.

Physician staff assessed the woman at 2320 hours, at which time there had been one deceleration with good recovery. The woman was now 8 to 9 cm dilated and still in a right occiput transverse position and asynclitic. The plan was to restart oxytocin however from that point, the fetal heart baseline raised from 150 to 180 bpm and there were recurrent deep decelerations despite holding oxytocin, IV fluids, and changes of position.

At 2340 hours an obstetrical consultation was requested.

At 2345 hours, the patient was still 8 to 9 cm dilated and external fetal monitoring indicated an abnormal fetal heart with a baseline of 190 bpm and decelerations down to 90 bpm. A Caesarean section was recommended due to abnormal fetal heart. The heart tracing from that point on, appears to have been a recording of the maternal heart.

The woman was taken into the operating room at 2351 hours. She had an epidural in place which was subsequently augmented. Upon commencing the Caesarean section, the fetal vertex was found to be wedged in the pelvis. The resident had difficulty with the attempted delivery, so the attending physician took

over. A request was made for nitroglycerin to be administered by the delivery anesthetist and another physician was asked to push up vaginally to elevate the head.

At 0002 hours on June 19, 2011, a 3104 g male infant with Apgars of 0 and 0 at one and five minutes, was born.

The neonatal staff was available at the time of delivery. The infant was pale, apneic, and flaccid with no movements noted and no heart rate auscultated. Resuscitation with bag and mask did not result in any response, so an ETT was placed. This did not result in any improvement so the tube was removed and repeat bag and mask ventilation commenced. The attending physician was able to reinsert an ETT at five minutes of age. At that time, chest compressions were initiated along with PPV and 100 % oxygen. Throughout this time, the heart rate on the monitor was 80 to 120 bpm, with no palpable pulse or auscultation of heart sounds. Epinephrine X 2 that was given through the ETT was ineffective.

An umbilical venous catheter was placed and there was no improvement with saline fluids or further epinephrine. Blood drawn from the umbilical venous catheter for blood gases revealed a PH of 6.83 and base deficit of 27 and lactate of 7.3. The umbilical cord gases showed a venous PH of 7.31, base deficit of 4, arterial PH of 7.13 and a base deficit of 10.

Due to a lack of response, the resuscitation was stopped at 0022 hours when the infant was 20 minutes of age.

Post mortem

Post mortem examination revealed:

Cerebral edema, grey and white matter; acute petechial hemorrhages in pleural and pericardial surfaces; normal male karyotype; placenta weight greater than 90 percentile for gestational age; circummarginate insertion of membranes borderline chorangiosis.

The fetal autopsy weight was 3089 g. The expected weight at 38 weeks gestation is 2603 g +/- 559 g. The placenta weight was greater than the 90th percentile for gestational age and

the fetal-placental weight ratio was less than the 3rd percentile for gestational age. The placental enlargement was much more pronounced than the increased fetal weight for gestational age.

The observed cerebral edema and petechial hemorrhages in the pleural and epicardial surfaces were felt to be consistent with fetal asphyxia as the cause of death.

Discussion

The pregnancy for this patient progressed without significant problems until premature rupture of membranes at approximately 37 weeks gestation. Earlier detection of an irregular fetal heart did not have any impact on the progression of the pregnancy or the events in labour. While the woman's blood pressure was mildly elevated at most prenatal visits, intervention was not felt to be necessary.

Labour progressed without concern until approximately 45 minutes prior to delivery. Prior to this time, there had been a couple of occasions of fetal heart late deceleration; however it had recovered to a normal pattern. From 2315 hours onwards, staff likely anticipated that the heart rate would recover as it had before.

There was timely consultation with obstetrical services. The time from consultation until commencement of Caesarean section was approximately 10 minutes. Although there was difficulty in delivering the baby due to an impacted vertex, this did not likely significantly contribute to the final outcome.

The infant was flat at delivery. The umbilical cord gases that were obtained do not reflect this status or the inability to resuscitate. Follow-up gases that were taken at 17 minutes of age are more representative of the actual condition of the infant. Response to the abnormal fetal heart was appropriate.

There was good documentation throughout the course of the pregnancy, during labour and in the antenatal period.

Recommendations

No recommendations.

Case: 2012-N-8
OCC File: 2011-12331

History

The mother of the deceased was a 21-year old G2P1. Her past medical history included the vaginal delivery of a 7 pound 8 ounce male infant at 39 weeks gestation in 2010. Her past medical history was otherwise unremarkable.

Prenatal testing was normal and her blood type was 0 negative. She was given RhoGAM® at 29 weeks. Her glucose challenge test was normal and a second trimester ultrasound scan done at 20 weeks revealed all normal anatomy and the placenta clear of the os. Antenatal II records revealed that she was followed regularly with appropriate evidence of fetal growth and no abnormality, apart from repeated 1 + protein, but she remained normotensive.

There was no documentation on the chart of prior involvement with Children's Aid Society.

The referral to Children's Aid Society was on April 11, 2011, at approximately 13 weeks gestation. At that time, the woman's partner was reported to have "repeatedly strangled and struck her, including striking her in the vagina and in the area between her chest and stomach." The woman was taken to hospital and it was reported that she had "scratches and scrapes" and "redness and fingerprints around neck." It was not felt that she needed any further medical care.

The woman's partner was charged with criminal offences for the domestic assault and was not to have any further contact with her. There was close involvement with Children's Aid Society throughout the pregnancy, mainly because the woman had another child at home.

There was no record of any further domestic assaults and it appeared that the woman and her partner subsequently reconciled.

The woman was admitted to hospital on September 21 at 37 weeks gestation with spontaneous labour. She was already 5 cm

dilated and due to her unknown Group B Streptococcus status, she was started on penicillin and an epidural was initiated.

Nursing notes describing events in labour were written as a late entry at 1800 hours on September 21, 2011. The notes recorded the events that occurred eighteen hours earlier, from 0010 hours on that date.

The first time of written indication of late recovery of the fetal heart was at 0530 hours.

At that time, the patient was 9 cm dilated with membranes intact. At 0550 hours, there was difficulty getting consistent monitoring "due to low fetal position."

At 0620 hours, it was noted that "FHR seems to be dropping difficult to get HR [heart rate] above 120." At 0630 hours, the supervisor was summoned. At 0635 hours, the family doctor was contacted and informed of heart rate decrease and indicated he would be attending. At 0700 hours, the doctor arrived and ruptured the woman's membranes.

At 0705 hours, the spontaneous delivery of a female infant occurred. The infant was limp and blue and there was no cry or evidence of pulse or respirations. The placenta was expelled at 0706 hours. There was no report of unusual bleeding.

Ventilation was initiated with bag and mask and CPR was initiated. There was successful intubation and repeated adrenaline via the ETT was given. Between 15 and 20 minutes post delivery, a heart rate was obtained by auscultation. There was difficulty with IV access, so an intraosseous line was obtained.

Contact was made with a neonatologist in a tertiary care centre. The neonatologist advised the care team to keep the infant cool, administer dopamine for blood pressure support and phenytoin if the baby seized, and to continue trying an umbilical vein. There was acknowledgement that the baby's status was extremely guarded. It was felt that it would be too long for an NICU team to come up from the tertiary care centre, so it was suggested that a hospital in a neighbouring province be contacted. The hospital from the neighbouring province was contacted and since they were unable to send a team, emergency air transportation was arranged through ORNGE (the Ontario provincial air ambulance service).

During the half hour that the physician was making arrangements for transportation, the infant was cared for by the anesthetist. The baby became more stable and the blood pressure remained reasonable, despite withdrawal of dopamine. The baby became quite jittery and was given phenytoin. On arrival of the ORNGE team, they were able to establish an umbilical venous catheter and the baby was set up on a ventilator and prepared for transfer to the out-of-province hospital. One of the attending physicians accompanied the transport team.

Cord gases were obtained for venous blood only with a PH of less than 6.8. The hemoglobin of the newborn was 182, ruling out an intrapartum fetal bleed.

The baby was transferred to a hospital in a neighbouring province. Despite the care provided, she was deemed to have a poor prognosis and was designated "do not resuscitate." The infant died on September 23, 2011.

Post mortem

An autopsy was not performed.

Placental pathology showed the mean weight for 37 weeks. There was a thin layer of adherent blood clot adjacent to one edge that did not indent the placenta. It was 13 x 5.5 x a few mm in thickness. The umbilical cord measured 31 cm in length. Only a short length of cord was left attached to the baby. Therefore, it was determined that this was a short cord.

Discussion

This pregnancy was complicated by a history of domestic abuse. Physical abuse was documented near the end of the first trimester. There is no evidence in the subsequent prenatal records however that this had any impact on the final outcome.

The mother and her partner appeared to have reconciled by the time the baby was born.

The pregnancy was otherwise normal up until labour. Fetal heart tracing started to have an abnormal appearance by 0140 hours. This gradually became more concerning.

A late recovery of the fetal heart was first acknowledged and recorded by the nurse approximately 1.5 hours prior to the delivery. Shortly after that, and until delivery, there was no consistent recording of the fetal heart. Despite that, the attending physician was not called until approximately a 1/2 hour prior to the delivery. The physician attended within 25 minutes and the baby was delivered shortly after.

Appropriate attempts were made for resuscitation and the baby was stabilized, though in an extremely critical state for transport.

The ORNGE team was present by 1023 hours and prepared the baby for transport. The baby was transferred out at 1216 hours.

Earlier intervention may have changed the course of events and had a significant impact on outcome.

Recommendations

1. Hospital A should ensure that all obstetrical care providers in their hospital have updated training in Fetal Health Surveillance in Labour.
2. Hospital A should perform a QCIPA review of this case.
3. Obstetrical care providers are reminded of the importance of complete, accurate, timely and detailed documentation.

Case: 2012-N-9

OCC File: 2011-13634

History

The mother of the deceased was a 33-year-old G2P0. This was an intrauterine insemination pregnancy with an EDD of December 13, 2011. She declined prenatal screening for aneuploidy. The second trimester alpha-fetoprotein was normal. The morphology ultrasound at 19 weeks was normal, however there was a large

fibroid. Prenatal blood work and glucose challenge test were normal. Her one previous pregnancy was an early loss at seven weeks gestation. She had a history of polycystic ovarian syndrome and uterine fibroids. There were no other medical or genetic risk factors. She denied smoking, drugs and alcohol consumption.

The pregnancy was complicated by several episodes of vaginal bleeding. She was diagnosed with a chronic abruption. Her hospital admissions included:

Date	Hospital	Notes
Aug 27-31, 2011	Hospital A	24.5 weeks gestation - normal hemodynamics, NST reactive, uterine irritability and cervix closed. - bedside ultrasound showed "globular appearance to placenta." - BPP was 8/8 and hemoglobin, platelets and fibrinogen were normal. - Kleihauer Betke test was negative. - received betamethasone. - August 30, 2011, the EFW was 699 g (in the 37th percentile) and the BPP was 6/8. - Doppler readings were normal.
Sept 28–Oct 4, 2011	Hospital A	29 weeks gestation - normal hemodynamics, NST reactive, uterine irritability, hemoglobin, platelets and fibrinogen normal - Kleihauer Betke negative - No further bleeding – daily NSTs normal - September 30, 2011 – EFW – 1159 g (in the 27th percentile), and the BPP was 8/8 - Doppler readings were normal - Abnormal placental morphology - GBS negative
October 6-7, 2011	Hospital B	30 weeks gestation (average ultrasound age was 29 weeks 6 days) - no weight or percentile - Notes indicate "correlates with gestational age," but "AC 2 weeks behind BPD" [abdominal circumference two weeks behind Biparietal Diameter]
October 10 –2011	Hospital B	30 weeks 3 days gestation - Admitted to hospital and monitored until delivery - no antenatal progress notes for any of Hospital B admissions (triage records only) - operative note indicated plan to "follow serially until 35 weeks" - surveillance from order sheets - daily NSTs - bleeding was noted
October 11, 2011	Hospital B	- BPPs ordered on October 11, 21 and November 1,, 2011 - Ultrasound hand-written reports - BPP – 8/8, EFW – 1576 g (no percentile given), BPD – 31 weeks 1 days, FL – 29 weeks 4 days

Maternal and Perinatal Death Review Committee 2012 Annual Report

Date	Hospital	Notes
October 16, 2011	Hospital B	- Bleeding was noted - bleeding on Oct 10 and 16 was mild to moderate in amount and associated with uterine cramping. There were no concerns about fetal wellbeing nor was there any evidence of maternal hemodynamic changes, anemia or coagulopathy.
October 17, 2011	Hospital B	BPP – 8/8, EFW – 1553 g (no percentile and no biometry on report)
October 25, 2011	Hospital B	- ultrasound – BPP 8/8, EFW – 1650 g (no percentile and no biometry on report) - notation of “slower growth”
October 28, 2011	Hospital B	- BPP 8/8 - undated report
November 1, 2011	Hospital B	- ultrasound - BPP – 8/8, EFW – 1938 (7th percentile), BPD – 33 weeks 5 days, AC – 31 weeks 2 days, FL – 31 weeks 2 days “dumpy placenta”

On November 2, 2011 at 34 weeks gestation, fetal testing was abnormal. The NST over two hours showed decreased variability and no accelerations. The Operating Room report indicated an ultrasound was done, but there was no documentation of this testing in a report or in the progress notes. The BPP indicated, “no reflex movement and no response to stimulation – in retrospect fetal movement related to seizure activity.” Notes indicated that the “baby appeared to be having seizure activity while the umbilical cord was being cut.”

An 1890 g male infant with head circumference 30.5 cm and length 43 cm was delivered by emergency Caesarean section. Apgars were 3, 5 and 5 at one, five and ten minutes. Resuscitation and intubation were required because of poor respiratory effort. Umbilical cord gases were normal (umbilical artery pH – 7.32, vein pH– 7.36). The baby had no spontaneous respirations, was noted to be jittery and heart rate was 60/min. Positive pressure ventilation (PPV) was given with 100% O₂ and heart rate increased to 100/min. The infant was intubated and placed on ventilatory support (rate 50, 15/5 on 40% O₂), but he remained very jittery and apneic. Perfusion was satisfactory. Abdomen was soft with no organomegaly. He had micropenis with hypospadias and both testes were palpated in the scrotum. He received one saline bolus. The blood sugar was 3.3, but dropped to 1.1 and he was given a 10% dextrose bolus and required a second bolus with blood sugar of 2.3. A 12.5% dextrose infusion was started.

Reassessment revealed that the infant had no spontaneous

respirations. He was cardiovascularly stable. Neurologically, he was obtunded and had persistent tremors; his pupils were dilated at 4 mm and fixed; his upper limb tone was increased and lower limbs were hypotonic.

The infant was oozing from venipuncture sites. Laboratory results indicated: normal CBC, electrolytes and venous blood gas was pH 7.13, bicarbonate 24, base deficit +8, INR/PTT were elevated at 2.33/96.6 respectively. Due to persistent jitteriness, seizures were suspected and the infant received two doses of phenobarbital. He was transferred to Hospital C, a children’s hospital, for further management.

At Hospital C, he remained apneic and neurological examination showed absence of gag reflex, absent suck and grasp and no corneal reflex. Upper limbs were hypertonic with deep tendon jerks, but lower limbs had absent reflexes. Cardiovascular system was stable.

A CT scan of the head showed diffuse, bilateral edema and split sutures. An MRI showed extensive bilateral restricted diffusion and abnormal lactate peaks on magnetic resonance spectroscopy, consistent with severe hypoxic ischemic encephalopathy. EEG was significantly abnormal and no seizures were captured.

The infant remained ventilator dependant, neurologically depressed and became anuric. An ultrasound of the abdomen showed acute tubular necrosis. An extensive genetic and

metabolic screen was undertaken including: chromosomes, microarray, 7 DHC, DNA, amino acid profile, lactate and pyruvate, free and total carnitine, acetylcarnitine with urine organic acids and ketones. The chromosomes were normal male.

The parents were informed of the serious findings and grave outcome, and agreed to withdraw treatment. The infant died after extubation on November 5, 2011 at three days of age.

Post mortem

An autopsy was not conducted.

Placental findings were consistent with chronic abruption including:

- subchorionic old hematoma involving the entire fetal surface;
- circumvallate placenta, complete circumferential with old marginal blood clot;
- hemosidrosis of membranes and chorionic plate.

Discussion

This infant was delivered by urgent Caesarean section at 34 weeks and three days gestation. The daily NSTs had been normal, however on the morning of November 2, 2011, the variability was decreased and there were no accelerations. The operative report indicated that the ultrasound report showed “no reflex movement and no response to stimulation.” The pediatric notes suggested that there may have been absent end diastolic flow in the umbilical artery. The baby was born with normal cord gases, but required intubation. The neonatal course was characterized by apnea, and signs of severe cerebral injury. The CT and MRI scans suggested a severe hypoxic ischemic insult. Based on the poor prognosis, medical intervention was withdrawn on day three of life. Autopsy was declined.

There did not appear to be an acute cause for the change in the fetal testing and the normal cord gases did not support an acute insult. The pregnancy was complicated by a chronic placental abruption, however all the testing of fetal wellbeing was normal. This does not rule out the possibility of a hypoxic insult that may have occurred earlier in the pregnancy causing a neurologic injury. However, the abnormal placentation and ultrasound assessment of the placental morphology suggest that there

may have been a chronic subclinical in-utero hypoxia. The ultrasound assessment of fetal growth and placental function was incomplete and poorly documented.

Alternatively, there may have been an underlying pathology that has not been identified. There was no autopsy and the results of some metabolic studies were not available.

The documentation of the ongoing assessment and plan of management were lacking. There were no progress notes during the patient’s admissions to Hospital B. It was not clear who was performing and reporting the obstetrical ultrasounds and it was not clear from the requisitions what assessment was being requested (i.e. growth, BPP, dopplers, etc.) and the documentation of the results was hand written with no dictated report.

The finding of significant tremors (described as “jitteriness” in the notes) is a very uncommon finding at birth. It can present hours after birth and is usually benign and self-limiting. It can be participated by hypoglycaemia or hypocalcaemia or be a sign of neonatal withdrawal from maternal use of certain drugs. However, it has been described in severe anoxic brain injury.

Recommendations

1. Hospital B should complete a review of the maternal care provided to the mother of the deceased infant, including all documentation, including, but not limited to, the availability of physicians notes.
2. Hospital B should complete a quality of care assessment of the ordering and reporting of obstetrical ultrasound.
3. Obstetrical care providers are reminded of the importance of umbilical artery doppler in the assessment of placental function in pregnancies complicated by fetal growth restriction.
4. The Regional Supervising Coroner should follow up with Hospital B regarding the issues identified. If, in the opinion of the Regional Supervising Coroner, systemic issues persist, the Regional Supervising Coroner should consider conducting a Regional Coroner’s Review with appropriate representatives of the hospital.

Case: 2012-N-10

OCC File: 2011-9570

Antenatal History

The mother of the deceased was a 22-year-old G1P0 with an EDD of August 2, 2011 based on early ultrasound dating. This was an unplanned pregnancy. She was referred by her family doctor for midwifery care in the first trimester. Routine prenatal laboratory investigations were normal. She declined prenatal genetic screening testing. Routine second trimester ultrasound was normal. Glucose Challenge Test was declined. GBS culture was positive.

The pregnancy was complicated by a presentation consistent with pyelonephritis at 30-31 weeks gestation, however a urine culture was negative. Symptoms were resolved with ceftriaxone. An obstetrical ultrasound done at the time was normal.

The mother's past medical history included asthma for which she used inhalers. She was a non-smoker. She had an appendectomy in 2000 and her family history revealed an episode of deep vein thrombosis in her father.

Course in Labour and Delivery

Labour started at 0900 hours on August 2, 2011 at 40 weeks gestation. The woman was assessed at home by her midwife at 0945 hours. There appears to be no documentation of this visit, other than what appears in the hospital chart.

The woman was admitted to Hospital A's obstetric unit at 1015 hours. The cervix was 4 cm dilated, fully effaced with the vertex at spines -2. Her membranes ruptured spontaneously for clear fluid at 1030 hours. She was given IV penicillin G 5 million units at 1035 hours. Contractions were strong every three minutes, lasting 60 seconds. At 1130 hours, she was 6-7 cm dilated and the vertex was at spines. At 1300 hours, the maternal pulse was recorded at 96 bpm. She received 2.5 million units of penicillin G IV at 1500 hours. At 1600 hours, the maternal pulse was recorded at 72 bpm. At 1815 hours, she was fully dilated. The FHR by intermittent auscultation (IA) was normal with a baseline of 140-150 until 1815 hours when the baseline changed to 115-120 bpm. There were no recorded decelerations or accelerations

and the woman was afebrile.

The woman started pushing shortly after reaching full dilation. At 1930 hours, she received another dose of 2.5 million units of penicillin G IV. Starting at 1940 hours, decelerations to 90 bpm from a normal baseline were noted. At 1950 hours, the baseline FHR changed to 100 bpm and at 1955 hours, was 110 bpm. There were no FHRs recorded after 1955 hours. There was a late entry in the chart indicating that the FHR was 90 bpm approximately two minutes before delivery.

The woman went on to spontaneously deliver a male infant at 2007 hours. The cord was tightly around the neck once. The baby was flat at birth. Apgars were 0, 0 and 0 at one, five and ten minutes. The arterial cord blood gas pH was 6.84 and BE -21. Chest compressions and PPV were started. The emergency room physician attended and continued the resuscitation. The baby was eventually intubated at 32 minutes on the third attempt. The children's hospital was consulted and passive cooling was started. The transfer team arrived at 2 hours 38 minutes. There was bleeding from IV sites. Subsequent blood work showed DIC.

After assessing the baby, the grave prognosis was explained to the parents. The option of palliative care was discussed, but the parents requested that the baby be transferred to the children's hospital for continued care. While at the children's hospital, the baby remained unresponsive. An MRI on day four showed severe hypoxic-ischemic encephalopathy involving the cerebral hemispheres and basal ganglia and intraventricular hemorrhage with mild ventriculomegaly. After discussion with the family, life sustaining measures were withdrawn on August 7, 2011.

Post mortem

Growth parameters were normal for the gestational age. There were no congenital anomalies.

There was multi-organ hypoxic-ischemic injury involving the entire brain, the liver and kidneys. There was evidence of meconium aspiration.

There were no significant placental findings.

Cause of death was hypoxic-ischemic encephalopathy complications of intra-uterine asphyxia of undetermined cause.

Discussion

This infant died from severe intrauterine asphyxia. There were no antenatal risk factors. Intrapartum FHR surveillance revealed that there was a decrease in baseline from 1815 hours until the baby was born. There were no recorded accelerations or record of fetal movement. There was a note of the heart rate being 90 with pushing, with recovery to 100-110 bpm. There were no FHRs recorded after 1955 hours.

The baby was flat at birth twelve minutes later. Acute total cord occlusion (as occurs with cord prolapsed) could result in such a severe asphyxial insult, but it is highly unlikely that a nuchal cord could produce such an outcome when the first decelerations were detected just twelve minutes before birth. A nuchal cord can cause repetitive variable decelerations on continuous electronic fetal monitoring. Over a period of time, such variables can develop into complicated variables (e.g. decelerations of <70bpm for >60secs, baseline tachycardia or bradycardia etc. - See JOGC September 2007, Volume 29, Number 9) which can be associated with fetal compromise. There was no evidence from the FHR recorded by IA of any repetitive decelerations earlier in the labour.

Concerns were raised by the parents that the midwives seemed to have difficulty auscultating the fetal heart for at least an hour prior to delivery. The midwives' record does not indicate any difficulties and FHRs were documented appropriately every five minutes during the second stage of labour up until the last twelve minutes. However, with descent of the presenting part, it can sometimes be more difficult to find the fetal heart as it comes near and under the symphysis pubis. This could have led to the perception that there was difficulty auscultating the fetal heart. It cannot be determined from this review whether the midwives were actually having difficulties or whether this was simply the perception. It would have been prudent nonetheless, to check the maternal pulse during the second stage of labour. If there had been any uncertainty, EFM should have been employed. If the FHR was abnormal during this time, recognition leading to timely intervention may have resulted in a different outcome.

Meconium aspiration occurs in utero due to an asphyxial event. The finding of meconium aspiration on autopsy is unexpected given that the amniotic fluid was clear at the time of membrane rupture and meconium was not observed during the labour or at delivery. An intrapartum event leading to meconium aspiration would be expected to be associated with an abnormal FHR. Again, there were no FHR changes detected by IA to suggest such an event.

Although the guidelines for IA were followed in terms of frequency, there is no mention of accelerations noted throughout the labour. The hospital's partogram form has a box to note decelerations only; there is no space to note periodic changes.

Fetal well-being assessment requires that there be at least two accelerations every 40-80 minutes, to be considered reassuring. In this case, documentation of fetal movement and/or digital scalp stimulation to elicit a response was absent. The absence of any narrative notes regarding the midwives' assessment of the progress of labour, as well as fetal well-being, makes it difficult to determine if in fact the midwives involved were concerned at any point throughout the labor and birth. If they were having difficulty obtaining the heartbeat, there was no note to indicate such. The decelerations to 90 prior to the birth were not commented on. It is therefore not known what measures, if any, were taken to expedite the birth of the baby or manage the deceleration with position change, oxygen etc. No consultation was made with an obstetrician at any point during second stage for abnormal findings with fetal heart monitoring.

There were no notes made by the midwives following the birth of the baby. No follow up or discharge from care was noted. Thorough and complete documentation is crucial, especially when there is an adverse outcome.

It should be noted that intermittent auscultation guidelines do not include listening during a contraction; the standard is to listen immediately following the contraction. If there is concern regarding recovery of the heart rate to baseline, then continuous electronic fetal monitoring should be commenced. There appears to be a period from 1815 hours until the birth

of the baby at 2007 hours where the FHR was not meeting the criteria of “normal.”

The review of this case proved to be difficult as there was minimal documentation.

Recommendations

1. Obstetrical Care Providers are reminded:

- of the importance of complete, accurate, timely and detailed documentation;
- of the importance of appropriate implementation of intermittent auscultation (IA) and of timely response to abnormal FHRs;
- of the Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline (JOGC September 2007, Volume 29, Number 9);
- of the importance of comparing maternal pulse with fetal heart tones during second stage of labor.

2. Hospital A should revise their fetal health partograph to include all periodic changes (e.g. accelerations).

3. The Regional Coroner should perform a Regional Coroner’s Review of the circumstances surrounding this death.

4. The SOGC should review the Fetal Health Surveillance in Labour Consensus Statement regarding intermittent auscultation (IA) to consider the absence of accelerations as an abnormal finding and to define baseline FHR changes.

Case: 2012-N-11

OCC File: 2011-3959

History

The mother of the deceased was a 31-year-old G3P1 with an EDD of March 29, 2011. Her past medical history included the delivery of an 8 pound 7 ounce male infant at term by spontaneous vaginal birth in 2007. She also had a previous elective termination.

Routine prenatal blood testing was normal. IPS showed a low risk of Down Syndrome. Glucose tolerance testing was normal and GBS screening was negative. Second trimester ultrasound scans were done at 19 and 21 weeks and both showed normal anatomy with the placenta clear of the cervical os.

Subsequent prenatal visits were documented on the Antenatal II record and showed the normal progress of the pregnancy. A BPP done on April 1 was normal with EFW of 3176 grams. Anatomy was not assessed.

The woman presented to hospital on the evening of April 1, 2011 at 40 weeks and 3 days gestation, in early labour with membranes intact, 5 cm dilated and fully effaced. She was admitted at 0015 hours on April 2 and followed with continuous fetal monitoring. Membranes were ruptured at 0141 hours and a fetal scalp clip was placed at 0156 hours. There were recurrent episodes of FHR deceleration, usually determined to be with contractions. The attending physician was at the bedside on a number of occasions. Fluid boluses, repositioning and scalp stimulation were used to improve the fetal heart and for evaluation.

The fetal heart tracing was difficult to interpret throughout most of the labour. However, from 0500 hours onwards, there appeared to be a more consistent pattern of late recovery of the fetal heart after contractions, until the time of delivery.

The woman was fully dilated at 0352 hours and started pushing from approximately 0400 hours until 0415 hours, but there was no descent. She was allowed to continue to labour and restarted pushing at 0608 hours. Because of concern about the

fetal heart, assisted delivery with a vacuum was recommended.

The woman was fully dilated with the fetal skull at spines +2 in the left occiput anterior position. There was significant 3 + caput over the fetal scalp when palpated like a tense bag of membranes. At 0648 hours, a Kiwi vacuum was applied with descent to crowning over one contraction and then a pop-off. The pediatric team arrived during the second application of the vacuum and the baby was delivered with the next contraction with the assistance of a right medial lateral episiotomy.

The infant was quite pale with no tone at the time of delivery and a tight nuchal cord was noted. Gases were taken and showed an arterial PH of 7.03, base deficit of 15 and venous PH 7.21 and base deficit of 9. The remainder of the mother's care was unremarkable.

Delivery occurred at 0650 hours and a "code pink" was called at 0651 when the neonatal heart rate was recorded by the labour and delivery nurse at less than 20 bpm; intubation was instituted. At 0653 hours, the neonatal heart rate was less than 10 bpm. Despite attempts at resuscitation, the baby remained with no tone or heart beat and continued to be pale. The pediatrician did not hear a heart beat at any stage, though acknowledged that a "nurse possibly heard heart beat at approximately 1 minute of age of less than 20 per minute." The code was stopped at approximately 17 minutes of age after no improvement of the newborn status.

Post mortem

The initial coroner's investigation determined that the death was likely due to intrauterine asphyxia. A forensic autopsy was not felt to be indicated.

The family requested that a hospital autopsy be conducted. The hospital subsequently arranged for a pediatric forensic post mortem at another facility.

The placental examination showed a term placenta with no significant abnormality.

Post mortem findings indicated that the newborn was a term male. The final pathological diagnosis included: centronuclear myopathy, severe liver disease, acute subdural and subgaleal hematoma, severe pulmonary hypoplasia, significant thymic hyperplasia and abnormal facies. The pathologist commented that the infant had the characteristic facies and muscle phenotype of myotubular myopathy. The pulmonary and hepatic pathology strongly suggested that this was an X-linked form of myotubular myopathy due to a mutation of the MTMJ gene. The intracranial and subgaleal hemorrhages were probably related to the vitamin K dependant coagulopathy and liver disease that could accompany the condition, which in turn would explain the large volume, acuity and entirely liquid state of the intracranial hematomas.

The death was attributed to intrapartum, intracranial and subgaleal hemorrhage, in the setting of infantile hepatic disease associated with myotubular myopathy. Contributing factors included severe pulmonary hypoplasia and small placental infarcts.

Genetic counseling was recommended and was offered to the family.

Discussion

This pregnancy appeared to be normal until the time of labour. Fetal heart tracing was difficult to interpret. However, over the 1.5 hours prior to birth, there appeared to be repeated late recovery of the fetal heart.

The arterial gases reflected this, however they do not appear to be low enough to account for the infant's condition at birth with vital signs essentially absent except for a presumed fetal heart of approximately 20 beats per minute shortly after birth.

The subdural and subgaleal hematomas were likely due to the congenital abnormalities as noted by the pathologist. As the vacuum delivery was done over a short period of time during two contractions, it is not likely that this contributed significantly to the findings at birth. The pop-off of the vacuum extractor is not unexpected with a large "caput."

Recommendations

No recommendations.

Case: 2012-N-12

OCC File: 2011-15754

Antenatal History

The mother of the deceased infant was a 34-year-old G2P1 with an EDD of December 21, 2011 based on early second trimester ultrasound. Routine prenatal laboratory investigations were normal. She was Rh negative and antibody screen negative. She received Rh immune globulin at 28 weeks. IPS was negative. GCT was abnormal and a two hour OGTT was normal. She was GBS negative. She was on citalopram during the pregnancy. Her antenatal course was uncomplicated.

She had one previous term pregnancy delivered vaginally of a 7 pound 1 ounce male in 2009. That pregnancy was complicated by gestational diabetes treated by diet alone. Her past medical history and family history were non-contributory.

Course in Labour and Delivery

At 0330 hours on December 19, 2011 at 39 weeks and five days gestation, the woman's membranes ruptured spontaneously. She was admitted to Hospital A at 0530 hours. The amniotic fluid was noted to have particulate meconium. The FHR was normal. Contractions were every seven minutes. At 0715 hours, there had been no change in the cervix, so oxytocin augmentation was ordered.

At 0730 hours, she was uncomfortable with the contractions. The baseline FHR was in the 140's with variable decelerations. The oxytocin was stopped and an epidural was started. She was assessed by the obstetrician at 0845 hours. The cervix was 4 cm dilated and 80% effaced. An order was given to restart the oxytocin in one hour if the EFM strip was normal.

At 0945 hours, there continued to be repetitive decelerations. The cervix was 5 cm dilated. Oxygen was given by mask at 8L/min.

At 1015 hours, a message was left for the obstetrician. The obstetrician returned the call at 1050 hours and after being updated, decided to come in to assess for possible Caesarean section.

The patient was assessed by the obstetrician at 1140 hours. The cervix was an anterior lip with the occiput posterior at spines +1. An attempt was made to have the woman try to push past the cervix, but the head remained high. The obstetrician's dictated delivery summary indicated that the decision was made to proceed to Caesarean section at this time, but the patient wanted to wait because the fetal heart had improved. A progress note indicated that the patient was exhausted and Caesarean section was offered.

Oxytocin was restarted at 1150 hours at 2mU/min and was increased to 6mU/min at 1215 hours. The patient was pushing at that time. At 1230 hours, the FHR tracing was interpreted as "reassuring." The oxytocin was increased to 8mU/min at 1245 hours and the decision was made to stop pushing and to restart pushing in one hour.

Pushing was re-commenced at 1330 hours. At 1354 hours, the FHR dropped and did not recover. The obstetrician was called at 1356 hours and attended at 1405 hours.

The woman went on to a spontaneous vaginal delivery at 1412 hours. There was a mild shoulder dystocia and the baby was pale and flaccid. Apgars were 1, 4 and 5 at one, five and ten minutes. Arterial cord gases were pH 6.77, PCO2 139, BE -17.1. Venous cord gases were pH 7.06, PCO2 62. Birth weight was 3917 g. Resuscitation was initiated by the anaesthetist and the emergency room physician was called. Bag and mask ventilation was provided. The baby was intubated and chest compressions commenced at three minutes of age. The heart rate was 100/minute after epinephrine was administered via the ETT. Passive cooling was initiated at 18 minutes of age. First seizures were noted at one hour of age and anticonvulsants were given.

Hospital B was called and their transport team arrived at 1545 hours. The baby was subsequently transferred to the children's hospital.

On December 20, 2011, neuroimaging showed evidence of diffuse ischemic-hypoxic changes and bilateral intraventricular hemorrhage. Acute tubular necrosis, DIC and transaminitis developed. On December 24, 2011, the grave prognosis was discussed with the parents. The baby was extubated and died on December 25, 2011 at 0615 hours.

Post mortem

There were no dysmorphic features identified at autopsy. There was hypoxic-ischemic encephalopathy with pontosubicular necrosis. The findings were consistent of global hypoxic injury.

The placenta showed meconium staining. There were nucleated red cells within the fetal vasculature, fetal vascular thrombosis, hemorrhagic endovasculitis and chorangiosis.

The cause of death was hypoxic-ischemic encephalopathy due to placental fetal vascular thrombosis and chorangiosis.

Discussion

This infant died due to an asphyxial peri-partum event. The antenatal course was uneventful. The mother presented in labour at term with ruptured membranes. Shortly after admission, particulate meconium was noted. FHR decelerations were noted during the labour and were described as variable in nature. At 1015 hours, the obstetrician was notified with regards to the decelerations and on assessment at 1050 hours, consideration was given to performing a Caesarean section. A Caesarean section was not performed at that time as the FHR tracing was felt to have improved and oxytocin augmentation was restarted. In the obstetrician's delivery summary, the decelerations during pushing in the second stage were described as occurring "with contractions and not recovering well." There were no cord complications noted at delivery although there was a mild shoulder dystocia. The time interval between delivery of the head and delivery of the shoulders was not documented. The cord gases showed a mixed metabolic and respiratory acidosis.

Review of the FHR tracing showed decelerations, a number of which were late in characterization beginning shortly after admission. Overall, the tracing was "atypical to abnormal" to becoming "abnormal" by SOGC criteria.¹ Although expert opinion may vary regarding the point at which the tracing indicates immediate delivery, augmentation of labour with oxytocin in this setting was contraindicated.

It cannot be determined with certainty by this review whether earlier delivery by Caesarean section would have resulted in a different outcome. At the time of presentation, there were signs suggestive of fetal compromise (e.g. particulate meconium and atypical FHR tracing) and placental findings suggestive of longer term antepartum insult were identified.

Recommendations

1. Obstetrical care providers are reminded of the SOGC classification of intrapartum FHR monitor tracings and the actions indicated for atypical and abnormal tracings.
2. Obstetrical care providers are reminded that the use of oxytocin is contraindicated in the absence of a normal FHR tracing.

References

1. Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline. Journal of Obstetrics and Gynaecology Canada (JOGC). Vol. 29, No. 9, September 2007.

Case: 2012-N-13

OCC File: 2010-17760

History

The 32-year-old mother of the deceased infant was pregnant for the third time with an EDD of February 2, 2011. The woman had one previous term delivery of a 7 pound 5 ounce infant after an uncomplicated pregnancy and vaginal birth. She also had a tubal pregnancy and salpingectomy. Her initial prenatal blood testing showed anemia with a hemoglobin of 98. IPS testing was done and showed her to be at low risk of Down syndrome. Her 20 week ultrasound was normal.

The woman was followed in shared care. On the morning of November 24, 2010, at 30 weeks gestation, she was seen by her family doctor and had a normal clinical examination. The woman reported decreased fetal movement, so a BPP was ordered for 1430 hours that day; the obstetrician was not informed.

The scan measurements were done and all growth parameters were appropriate for fetal age. The EFW of 1507 g was in the 45th percentile. The amniotic fluid index was in the 60th percentile. The Doppler S/D ratio was normal. The BPP score was 4/8 with 0 for breathing. Notes indicated that fetal movements were not adequate for a BPP score and that there was a moderate amount of fluid within the small bowel. The final summary of the radiologist indicated that "the first step would be to repeat this examination. Of course one always has the option of referral to a high risk centre as clinically indicated." This dictation was recorded the day after the BPP was conducted.

The technician who completed the ultrasound recognized that it was abnormal and concerning, and requested that the radiologist on duty be informed immediately. However, direct contact was not made and the radiologist only became aware of the abnormal result the following morning. The report was faxed to the family doctor as well as the obstetrician. The mother was instructed to go to the hospital for further assessment and consultation. The times that the films were read and when the attending physicians were contacted, could not be ascertained.

During the course of the coroner's investigation, it was

determined that radiologists were no longer available on site and that any results of a concerning nature were to be communicated with a radiologist located off-site.

The time the mother arrived at hospital is not noted on the record. It is noted however, that blood tests were taken at 1745 hours.

On arrival at the labour and delivery unit of the hospital, the woman did not have any history of vaginal bleeding, contractions, fever or leakage of fluid. The initial monitoring reportedly showed a fetal heart of 110 to 112 bpm with minimal to absent variability. This information could not be confirmed however as the strip was not available for review.

An IV was started and blood work was taken. Despite IV fluids, the fetal heart remained flat. A bedside ultrasound confirmed the fetal heart of 110 bpm with a maternal pulse of 70 to 90 bpm. After consultation with a maternal-fetal medicine specialist at another hospital, a decision was made to proceed with an emergency Caesarean section at the local hospital and that transfer or antenatal steroids were futile at this time. The children's hospital transfer team was also notified.

Upon delivery at 1902 hours, there was no movement or cry noted. The neonatal staff was in attendance and cord gases were taken. The placenta was removed for pathology and there was no sign of abruption. The balance of the procedure was unremarkable. Cord gases revealed an arterial PH of 6.87 with a base deficit of 20.3 and venous PH of 7.06 with a base deficit of 16.6. Apgars were 3, 5, and 5 at one, five, and ten minutes and birth weight was 1500 g.

The baby was initially stimulated and suctioned for thick meconium and PPV was applied. The baby's heart rate was above 100 bpm and improved with PPV to 160 bpm. The baby was intubated and stabilized. Initial gases following intubation and saturations improved to 93 -95% from 67%.

The baby was given BLES© (Bovine Lipid Surfactant) at approximately 35 minutes of age, but did not tolerate it. As a result, the baby was extubated, then reintubated. The children's hospital transfer team arrived at approximately 45 minutes of age and took over care. They were able to stabilize the airway

and manage breathing. Circulation continued to be a problem and despite multiple fluid boluses, the blood pressure remained very low. Follow-up gases showed normalization by three and half hours of age.

The baby continued to be completely apneic with no respiratory effort and minimal neurological response. The baby was quite hypotonic and there was significant concern about asphyxia and long term outcome. The parents were advised of the very poor prognosis and a decision was made to extubate the baby at approximately six hours of age. The baby died at 0315 hours on November 26, 2010.

Post mortem

Skull and cranial cavity: unremarkable, except that the brain was "mushy."

Heart and lungs revealed "dilated and tortuous ductus arteriosus with luminal thinning and ulceration." Mild left ventricular non-compaction. Sections of all organs were unremarkable.

The placenta was examined and revealed a parenchyma with hypovascular and edematous or fibrotic villi comprised approximately 50 % of the sample parenchyma. This extent of involvement could be expected to have adverse consequences for the fetus. There was also hypercoiling of the umbilical cord and marginal insertion of the cord.

These findings were suggestive as a possible etiologic reason for the villus pathology. In addition to these changes, the parenchyma showed villus abnormalities with dysmaturity, hypervascularity and excessive calcification. A specific cause accounting for the constellation of villus abnormalities of the placenta was not identified.

Discussion

This infant was born after an uncomplicated pregnancy where no concerns were identified until the day prior to birth. Due to maternal reporting of decreased fetal movement, a BPP was done. Although the BPP showed significant decrease in movement, this information was not directly or immediately communicated with the radiologist or the attending physician who had ordered the ultrasound. The attending physician also

did not conduct any follow up with the ultrasound unit.

It was not until the following day that the mother was subsequently advised to go to the hospital due to confirmation of the findings of decreased fetal activity. At that point, there was already evidence on initial monitoring of a compromised fetus. An emergency Caesarean section was appropriately done, however the newborn was extremely compromised at birth with low PH. Despite appropriate attempts at resuscitation and stabilization, the infant's condition never improved and she died a few hours after birth.

This mother and infant should have had urgent assessment in the obstetrical unit on the day when decreased movement was noted and confirmed with a BPP.

The delay of greater than 24 hours before the information was shared and an emergency Caesarean section was performed, likely contributed to the outcome. In addition, with the "shared care" arrangement, the family doctor should have informed the obstetrician or delegate of the concern regarding fetal activity as they would be the "most responsible physician" (MRP) for urgent care and delivery.

Recommendations

1. It is recommended that the physicians and healthcare providers at the ultrasound unit involved establish procedures to ensure direct and prompt communication of abnormal results and further assessment or action if urgent care is required.
2. Obstetrical care providers and staff in ultrasound units are reminded that when decreased fetal movement is suspected, there is a need for immediate assessment, communication of results and facilitation of further testing and management of care if necessary. There must be a method or procedure to directly and immediately communicate critical results to the most responsible physician or designate.
3. Technologists and radiologists interpreting biophysical profiles should have appropriate training and understanding of the implications of test results and appropriate responses and courses of action to follow.

Case: 2012-N-14

OCC File: 2011-13852

Maternal History

The mother of the deceased infant was a 32-year-old primagravida. She had an intravaginal insemination induced pregnancy on May 5, 2011. She was diagnosed with a diamniotic/dichorionic twin pregnancy with an EDD of February 2, 2012. An ultrasound, GCT and AFP test were normal. The EFWs were 412 g for Twin A and 750 g for Twin B. A BPP was 8/8.

Twin A subsequently had a fetal demise.

The woman went into spontaneous labour and received two doses of betamethasone prior to Caesarean section.

Neonatal history

The 1145 g female infant was born at 31 weeks and four days gestation following a Caesarean section for fetal distress. The infant had respiratory distress and chest x-rays showed evidence of Respiratory Distress Syndrome. Apgars were 2, 4 and 7 at one, five and ten minutes. The infant was intubated and given surfactant.

The infant was hypotensive and required saline bolus and a dopamine infusion. She was stabilized and extubated at 24 hours of age. Examination was normal. CBC and blood cultures were taken and antibiotics were started. At 48 hours, blood cultures were negative and the antibiotics were discontinued.

As apneic episodes and oxygen desaturations were noted, treatment with caffeine was initiated. The infant developed jaundice and was started on phototherapy. An umbilical line was placed and total parenteral nutrition (TPN) was started. Oral feeds were commenced on Day 3 with expressed breast milk. Feeds were gradually increased and tolerated well. Glycerine suppository was given for poor stooling and abdominal distension and subsequently watery stools were passed. The feeds were gradually increased and although a 13 cc aspirate was noted on Day Five, the feeds were subsequently tolerated. Premature formula was given, although the infant

received mostly expressed breast milk. Due to tachycardia, the caffeine dose was reduced and further episodes of apnea and bradycardia were noted on Day 7. A head ultrasound was performed and was normal. The caffeine dose was increased on Day 13 (December 20, 2011) due to further apneic episodes. In view of the spells, oesophageal reflux was considered, but no anti-reflux treatment was started.

The feeds were gradually increased and TPN was discontinued. There were increasing episodes of blood oxygen desaturations and apneic spells from 8 on December 21, 2011 to 18 on December 23, 2011. There was abdominal distension, but no aspirates were noted and an abdominal x-ray done on December 22, 2011 was normal. CBC and C-reactive protein were normal and stool for occult blood was negative. The infant was started on low flow oxygen on December 23, 2011 (Day 16). Blood culture was taken and antibiotics were started with vancomycin and cefotaxime. Oral feeds were held.

The infant became increasingly unwell; tachycardia (pulse 203) was noted and she appeared pale and limp. Frank rectal bleeding was also noted. She was placed on a ventilator and supported aggressively. Blood gases were acidotic with bicarbonate of 10, base deficit of 25 and a pH of 7.28.

Further resuscitation included a 45 ml saline bolus, 30 ml of fresh frozen plasma and dopamine infusion for low blood pressure. Meropenem was also added to her treatment.

A tertiary care centre was contacted and transfer was being arranged when the infant's condition became too unstable for transfer. A repeat x-ray showed NEC with intraluminal perforation, but no free air. Surgical consult was requested and a decision was made to insert an abdominal drain. Free air and perforation were confirmed.

The parents were updated regularly regarding the infant's grave condition. On December 24, 2011 (Day 17), a "code" was called at 0015 hours and despite aggressive resuscitation, including multiple doses of epinephrine, the infant succumbed at 0020 hours.

Post mortem

The blood and post mortem splenic cultures isolated enterococci.

The placenta showed evidence of hypoperfusion with:

- Small size
- Distal villous hypoplasia
- Decidual vasculopathy

The autopsy confirmed perforation, Hypoxic-Ischemic Encephalopathy and multiple pulmonary emboli.

Discussion

Necrotising enterocolitis is a leading cause of death in the NICU population. The incidence is 7-11% and is more common in babies under 1.5 kg and those who have been ill requiring a ventilator, treated for hypotension or transfused with blood. The mortality is between 15 – 30%. The pathogenesis is multifactorial and involves an over-reactive response of the immune system to an insult leading to increased intestinal permeability, bacterial translocation and sepsis.

Evidence-based strategies that may reduce the incidence of necrotising enterocolitis include promotion of exclusive breast milk feeding and probiotic administration.

This infant was a high risk due to her birth weight being below 1.5 kg and IUGR as well as requiring pressor agents and mechanical ventilation. Although she appeared stable, there were desaturations and apneic episodes from early on and these became more frequent. There were 4-6 episodes between December 10-15, 2011 and a significant increase after December 21, 2011. The feeds were not held until late on December 22, 2011.

If necrotising enterocolitis is suspected or diagnosed, holding the feeds and antibiotic treatment are recommended. Serial six-hourly abdominal x-ray can be helpful. In this case, although there were significant apneic spells, there were no other signs of necrotising enterocolitis and the disease was fulminant. The index of suspicion for sepsis (i.e. necrotising enterocolitis) should be very high although episodes of desaturations and apnea are common in healthy preterm infants.

It is not possible to determine if earlier implementation of the necrotising enterocolitis protocol would have changed the outcome.

Recommendations

No new recommendations.

Case: 2012-N-15

OCC File: 2012-2196

History

The mother of the deceased was a 15-year-old G1P0 who presented to hospital with abdominal pain in pregnancy. The Children's Aid Society (CAS) became involved in the first trimester when there were allegations made that the mother was smoking marijuana and drinking alcohol. Due to the CAS involvement at the time of death of the baby, the case was referred to the coroner's office.

There was minimal prenatal care. On October 5, 2011, the mother presented for her first prenatal visit in the first trimester. She denied use of any alcohol or street drugs. She did smoke cigarettes, but "not every day." The mother did not have any prenatal labs or dating ultrasound as was requested by her healthcare providers.

On November 10, 2011, the mother did not show for a prenatal visit. On November 14, 2011, the CAS contacted the prenatal clinic to advise them of their concerns about the mother using marijuana and alcohol while pregnant. At a prenatal visit on December 5, 2011, neither the laboratory tests nor the dating ultrasound were done. A pap test and swabs were offered, and refused. The mother did not show up for any other appointments. Attempts to contact her by telephone were hampered by a lack of telephone service; contact was also attempted through regular mail.

It was not until the mother was contacted by CAS and urged to have her investigations and prenatal visit done that she complied.

On January 5, 2012, an ultrasound gave an estimated gestational age of 21 weeks. The placenta was anterior and clear of the os. Fetal cardiac and facial structures were not well seen. The EDD was May 18, 2012. A repeat ultrasound was booked and the patient was notified.

On February 7, 2012, the mother presented to the secondary level centre with cramping; she was 9 cm dilated. No FHR tracings were completed. Upon assessment in the triage unit,

she had a large amount of blood per vagina. The FHR decreased to 80-90 bpm with contractions. Due to the clinical suspicion of abruptio placenta, the decision was made to proceed urgently to Caesarean section. The Caesarean was done under spinal anesthetic.

At the time of the delivery, the baby boy "emerged mildly depressed, but cried immediately." Apgars were 3 at one minute and 6 at five minutes. Weight was 930 g. Based on physical examination, the baby was estimated to be less than 27 weeks gestational age. Initially, he received CPAP, but then needed PPV and subsequently intubation. He was given surfactant, stabilized and transferred to a tertiary level NICU. Prior to transfer, blood cultures were obtained and IV antibiotics were started.

Cord gases were recorded as:

	Arterial:	Venous
pH	7.32	7.37
pCO ₂	53	44
pO ₂	19	-
HCO ₃	27	25
O2 Saturation	25	-
Base Excess	0.4	-

The CBC showed a neonatal hemoglobin of 148 (N: 135-195).

Upon admission to the tertiary level centre on February 7, 2012, the baby was changed to high frequency oscillation for increased CO₂. During the admission, a heart murmur and intraventricular hemorrhage were noted. On the night of February 15, 2012, the baby developed signs and symptoms of necrotizing enterocolitis (NEC) and his condition deteriorated. He had worsening pulmonary edema and metabolic acidosis. He developed multi-organ system failure. Despite numerous and significant efforts, he became bradycardic and unresponsive. The decision was made to withdraw efforts and provide palliative care. The baby died on February 16, 2012 at 1207 hours.

Cause of death was: pneumatosis intestinalis from NEC along with multiorgan system failure due to extreme prematurity.

Discussion

Although less than 1% of babies are born prior to 28 weeks gestational age, they have the most complications. Survival rates vary greatly and depend upon many other factors such as higher birth weight, female gender, prenatal corticosteroids and singleton birth. Placental abruption requiring urgent delivery by Caesarean section is a negative prognostic factor for survival. NEC is a serious complication of premature infants causing death.

Lifestyle factors that can affect preterm birth that may have impacted this case include: minimal prenatal care, smoking, drinking alcohol and using illegal drugs. (Reference: marchofdimes.com/baby/premature_indepth.html)

Recommendations

No recommendations.

Case: 2012-N-16

OCC File: 2011-11277

History

The deceased infant was born on August 3, 2011 at 35 weeks and two days gestation to a 33-year-old primagravida. The mother was healthy and had experienced an uneventful pregnancy until the day of delivery when she presented to her obstetrician with decreased fetal movements. Fetal assessments by NST and ultrasound were concerning, so an emergency Caesarean section was performed. After artificial rupture of membranes, the operative record indicated that the "amniotic fluid contains meconium."

The infant was born in good condition at 2047 hours and required no resuscitation. Apgar scores were 9 and 9 at one and five minutes respectively. Cord blood gases were normal. Birth weight was 2510 g which is appropriate for a gestational age of 35 weeks 2 days. There were no concerns until 2130 hours when the infant was noted to be "pale and motley [sic]," so the respiratory therapist was called to assess. The infant had normal vital signs, but was pale with mottled extremities. The NICU fellow was called. The infant had a "meconium spit-up" and after suctioning, she appeared to settle, so she was left with the parents with a plan to reassess in 10 minutes.

At 2245 hours, the infant looked pale, then vomited a large amount of "mec fluid." Glucometer testing showed a blood

glucose level of 1.7 mmol/L. Oral feeds were attempted, but the infant would not suck well. The respiratory therapist and fellow were informed and the fellow attended at 2320 hours. Only 5 cc of formula had been taken and repeat glucometer was still low at 2.4 mmol/L. The infant was admitted and transferred to the NICU resuscitation room for assessment and management. When an orogastric tube was placed, 24 cc of "dark green meconium-like gastric aspirate" was recovered.

On examination, the infant was described as inactive and mottled, but not pale. Vital signs were normal. Physical examination was otherwise normal, including the examination of the abdomen which was recorded as "soft, non-distended, bowel sound present, no masses." The admission problem list noted "prematurity, neonatal hypoglycaemia, ?fetal distress, rule out sepsis." A partial septic workup was performed and antibiotics were started. Intravenous glucose infusion was commenced and the infant was ordered not to receive anything by mouth. A chest X-ray was taken and she was transferred to the NICU for further management. CBC and blood gases were normal. The chest X-ray was normal, but the portion of the abdomen that was visible showed no gas beyond the duodenum and was subsequently reported as being suggestive of duodenal obstruction.

Overnight, she was fairly stable until around 0600 hours when she had a low grade temperature, developed mild tachypnoea and oxygen saturation decreased to 90-91 percent in room air. A nurse found that the infant's abdomen was tight and firm, that bowel sounds were diminished and that she had passed

meconium on several occasions. The fellow was notified and an abdominal X-ray was ordered. Again, there was evidence of obstruction at the level of the duodenum. The subsequent report from the radiologist stated that it "could be duodenal atresia, however malrotation cannot be excluded."

The staff neonatologist reviewed the case upon arrival in the morning and concurred with a probable diagnosis of upper bowel obstruction.

At 1023 hours, the children's hospital was contacted and advised of the situation. The infant was described as "stable" and the diagnosis was "rule out duodenal atresia." It was initially suggested that the abdominal X-rays be sent to the children's hospital for review by the surgeons.

At 1045 hours, the findings were discussed with the parents and they were advised that the infant was to be transferred to the children's hospital for assessment by the paediatric surgical service.

When the staff neonatologist at the children's hospital was informed of the call at 1125 hours, it was noted in the record that the "baby needs to come regardless of X-rays which have not yet arrived."

It is somewhat unclear from the call record, but it appears that the nurse at the originating hospital was informed at that time that the transfer was accepted and indicated that they would call when ready. A second entry at 1325 hours stated that the charge nurse was informed that the baby could be admitted.

The infant arrived at the children's hospital NICU at 1600 hours. Vital signs were normal although she was intermittently tachypnoeic. She was described as "pale and mottled." Her abdomen was soft with no distension and there was mild tenderness in the epigastric area. General Surgery assessed the infant as well and an urgent abdominal X-ray and upper gastrointestinal examination were ordered to rule out malrotation and volvulus. The infant was given a blood transfusion to improve tissue perfusion.

The abdominal X-ray revealed the same pattern as found at the originating hospital. Limited ultrasound and the upper GI examination were done at around 1800 hours and revealed

evidence of malrotation and possible midgut volvulus. The infant was taken for urgent laparotomy at 2000 hours and the surgeons found complete infarction of the small intestine due to midgut volvulus and malrotation.

The surgeons deemed that there was insufficient viable intestine to be able to sustain the infant, so the incision was closed and she was returned to the NICU for palliative care.

The parents were informed and agreed to withdrawal of life-sustaining medical therapy. The infant was extubated at 0138 hours on August 5, 2011 and passed away at 0253 hours.

Post mortem

Post mortem examination revealed:

- Malrotation of the bowel with congenital deficiency of the right meso-colonic attachment;
- Midgut volvulus with extensive acute hemorrhagic infarction of the small bowel;
- Stigmata of shock with generalized organ congestion and cortical lymphoid depletion of the thymic cortex;
- Periventricular leukomalacia, acute

Death was attributed to "acute small bowel infarction, due to midgut volvulus with congenital malrotation of the bowel."

The pathologist also commented that, "the neuropathological finding of acute periventricular leukomalacia and the degree of thymic cortical lymphoid depletion is suggestive of intra-uterine fetal stress."

Discussion

This infant died at two days of age from extensive small bowel infarction caused by malrotation and midgut volvulus. She was delivered by emergency Caesarean section slightly preterm at 35 weeks after the mother noted decreased fetal movements and an abnormal fetal heart pattern was detected. The infant was in good condition at birth, but exhibited signs of a proximal bowel obstruction shortly thereafter. After imaging studies were interpreted as showing evidence of bowel obstruction, she was transferred to the children's hospital and assessed by the general surgeons.

By the time the infant reached the operating room at the children's hospital, irreversible, extensive damage to the intestine had occurred and she died after a short period of palliative care.

Midgut volvulus due to malrotation is a rare, but extremely serious condition that can affect newborns. Most cases in children present in the first month after birth, including some in the immediate newborn period. Cases of in-utero volvulus have been described including some that have resulted in stillbirth. This diagnosis should always be considered in a neonate with bilious vomiting, especially when accompanied by signs of peritoneal irritation and/or circulatory collapse.

Midgut volvulus is considered a paediatric surgical emergency; any baby presenting with bilious vomiting should be referred urgently for assessment by a paediatric surgeon. While there are other less critical causes of persistent bilious vomiting such as duodenal atresia or stenosis, expeditious assessment, diagnosis and surgical treatment of a volvulus may be life-saving.

In this case, the infant presented with early and recurrent bilious vomiting/gastric aspirates with supportive radiological evidence of proximal bowel obstruction. However, she did not get to the paediatric surgical centre for evaluation and eventual laparotomy until approximately 16 hours after presentation.

The parents have raised concerns about the delay in recognition of this serious condition and the subsequent apparent lack of urgency in affecting transfer to the children's hospital. Once the infant reached the children's hospital, she was quickly assessed, investigated and operated upon, but the intestine was no longer viable.

It is clear that urgent laparotomy is the key to preserving intestinal viability after a volvulus has occurred. Recurrent bilious vomiting is the usual presenting sign that should trigger a set of investigations to rule in, or out, volvulus. This would usually begin with plain abdominal X-rays, but should progress to urgent upper gastrointestinal examination accompanied by paediatric surgical consultation. It is possible that, if things had moved along more quickly, surgery might have occurred while some viable bowel remained. However, it is also possible that the volvulus occurred in utero and that the bowel may have been unsalvageable for some time prior to the laparotomy.

The lack of fetal movement, abnormal fetal heart tracings and evidence of intra-uterine fetal stress on post-mortem may have been indicators that the volvulus had already occurred while the baby was still in utero. In addition, the amniotic fluid was described as meconium stained which, in retrospect, may have been stained with bile. If this were the case, it is unlikely that earlier intervention would have changed the outcome.

The team at the originating hospital undertook an internal review of this case in response to a letter from the Regional Supervising Coroner.

In response to the concerns identified by the Regional Supervising Coroner, the hospital identified several systems issues that may have contributed to the delay in transfer, including difficulties with obtaining copies of the images and problems mobilizing porters to affect the transfer. The hospital indicated that they have taken steps to improve these processes.

The recommendations made and implemented by the hospital were part of an internal Quality of Care Information Protection Act (QCIPA) review and are therefore not accessible to the MPDRC.

Recommendations

1. Neonatal care providers are reminded that bile-stained vomiting and gastric aspiration in a neonate is usually an indicator of significant intestinal pathology that should prompt urgent referral to a paediatric centre for surgical assessment. In such a setting, the presence of malrotation with midgut volvulus should be considered by conducting appropriate imaging studies and laparotomy if indicated.
2. The hospital involved should ensure that the internal review performed included an examination of the clinical presentation, interpretation of investigations and response by those clinically overseeing this case.
3. The transport team at the children's hospital should review the audiotape of the conversation between the referring physician at the originating hospital and transport team regarding advice and recommended action.

Case: 2012-N-17
OCC File: 2011-14825

Antenatal History

The mother of the deceased infant was a 27-year-old G1P0 who had a medical history that included a pituitary adenoma previously treated with surgery. She was hypothyroid controlled with levothyroxine 0.125 mg daily. She was GBS positive.

The mother began care with a midwifery practice (Practice A) and had two visits at 14 weeks and four days gestation and at 17 weeks. She then continued care with a second practice (Practice B) from 20 weeks onwards and had 12 prenatal visits. Routine prenatal laboratories were all normal, except the haemoglobin which was low normal. The 50 g OGCT was normal and the IPS was negative. Ultrasound at 7 weeks and five days, 11 weeks and 18 weeks and five days showed normal fetal morphology with low lying placenta.

A follow-up ultrasound at 26 weeks and six days showed posterior placenta clear of cervix and normal fetal growth was documented. The woman was considering a homebirth.

At 34 weeks and six days, she had reduced fundal height and an ultrasound indicated a fetus in the 10th percentile and BPP 8/8. At this visit, it was noted that she was GBS negative. She was instructed to commence daily fetal movement counts and advised when to call with concerns.

The midwife consulted with a maternal fetal medicine (MFM) specialist five days later and the woman was seen nine days after that; she was subsequently seen weekly by MFM until her induction at 40 weeks and six days. Documented informed choice discussions took place regarding the changing risk in the pregnancy and the need to birth in a hospital with a small for gestational age (SGA) or IUGR baby.

The following is a summary of the ultrasound results:

Date	Gestation	Amniotic fluid index (AFI)	Biophysical profile (BPP)	Umbilical artery Doppler	Percentile	Weight	Comments
Oct 13	34w6d	18.9 cm	8/8	Normal	10 th	1946g	SGA
Oct 20	35w6d	18.0 cm	8/8	Normal	10 th	2239g	SGA
Oct 27	36w6d		8/8	Normal		2412g	SGA
Nov 3	37w6d	15.3 cm	8/8	Normal		no weight	
Nov 9	38w5d	10.6 cm	8/8	Normal		2947g	No IUGR

Following the ultrasound on November 9 that indicated no IUGR, the next scan was scheduled for two weeks later.

The MFM discharged the woman back to midwifery care as the baby was demonstrating normal interval growth and symmetrical growth. Serial cervical stretches were commenced and a BPP was booked for 40 weeks 4 days. An ultrasound on November 22 at 40 weeks 4 days indicated:

Date	Gestation	Amniotic fluid index (AFI)	Biophysical profile (BPP)	Umbilical artery Doppler	Percentile	Weight	Comments
Nov 22	40w4d	14.6	8/8	Normal	5th	2858g	?IUGR

Course in Labour and Delivery and Postpartum

Induction of labour occurred at 40 weeks and six days for a fetus in the 5th percentile for growth. Induction with dinoprostone, oxytocin and epidural analgesia took place. There were approximately five hours of active labour, one hour of second stage labour and six minutes in third stage.

The baby was born in good condition at 0945 hours and required no resuscitation. Apgar scores were 9 and 9 at one and five minutes respectively. Cord blood gases were normal. Birth weight was 2750 g, which was at the 3rd percentile for weight according to the Fenton growth charts. Physical examination was otherwise normal. Breastfeeding was initiated and the baby did well. The infant was discharged home at four hours of age to be followed up by the midwife.

Over the next 24 hours, the infant breastfed every one to two hours and was stooling regularly. The midwife assessed her at home and reported that she was doing well. Later on, the mother reported that the infant was “cluster feeding” and “ate constantly” all day. The mother’s milk did not appear to have come in as she indicated that her breasts did not feel full and there was no milk dripping. Throughout the night, the infant seemed to be “fussy and gassy.” After a feed at 0130 – 0200 hours on November 26, she seemed to be in pain, gassy and hard to settle. The parents were up with the infant most of the night.

By morning, the infant had become quite lethargic and the mother called the midwife at 1100 hours to discuss her feeding concerns. It was felt that she may be dehydrated due to lack of intake and recommended that the parents get some formula to feed her as a supplement. They did so and reported back that she took a small amount of formula, but remained very sleepy. On the follow-up call, the parents reported that the infant may be looking a bit blue around the mouth. The midwife advised the parents to take the infant to the hospital and arranged for her to be seen by the paediatrician on call on the postpartum unit. The infant arrived at Hospital A at 1225 hours.

When the infant was first seen on the postpartum unit, she was yellow in colour and limp. She was rushed to the neonatal nursery and the paediatrician was paged. Intravenous access was secured and a drip with D10W commenced. Pulse oximeter was placed and the infant’s arterial oxygen saturation was found to be only 50%. The paediatrician arrived and immediately

intubated the infant, resulting in an improvement in saturation to 92%. PPV was commenced and the ETT was adjusted and secured. Several minutes later, the infant’s heart rate dropped and she was unresponsive with fixed, dilated pupils. Resuscitation was commenced with chest compressions, ongoing PPV and intravenous epinephrine.

There was a transient improvement in heart rate, but it dropped again. Some lip-smacking and abnormal posturing was observed and a loading dose of phenobarbital was given for presumed seizure activity. Further chest compressions and epinephrine were administered. This resulted in a sustained improvement in heart rate. The infant was connected to a ventilator and dopamine infusion was started. Antibiotics had been administered and two boluses of normal saline were given for poor perfusion.

Bloodwork taken shortly after the arrest at 1346 hours showed serum sodium of 146 mmol/L, Hb 189 g/L, WBC 12.8 with normal differential and slightly low platelets at 129. Arterial blood gas revealed a mild metabolic acidosis with pH 7.36, pCO₂ 30 mmHg, pO₂ 118 mmHg, HCO₃ 17 mmol/L and base deficit of 7 mmol/L. Bedside point-of-care glucose was 3.1 mmol/L, but the lab glucose was very low at 1.5 mmol/L. The children’s hospital transport team arrived at 1352 hours after having been notified of the infant’s condition and the need for transfer.

Upon arrival, team members described the baby as gasping with ventilation and unresponsive to pain or touch. She was mottled with poor perfusion. Her activity and responsiveness improved somewhat over the next half hour. A bedside glucose test showed a level of 2.1 mmol/L and a small bolus of D10W was given followed by an increase in the infusion rate. Follow-up glucometer check was 3.4 mmol/L. Another normal saline bolus was given for poor perfusion and transfer to the children’s hospital was commenced.

Upon admission to the NICU at the children’s hospital at 1850 hours, the infant had normal vital signs and good arterial saturation with minimal ventilation. She was breathing spontaneously. While she showed some response to handling, she was quiet with hypotonia and decreased reflexes. No gag or suck could be elicited. Initial bloodwork showed normal haemoglobin and WBC, slightly low platelets at 122, normal electrolytes and liver function tests. The lactate was slightly elevated at 5.7 mmol/L. Blood gases revealed a mild respiratory

alkalosis and she was successfully extubated shortly thereafter. No seizure activity was observed at this time.

Of note, the initial blood glucose was 1.5 by glucometer and 1.8 mmol/L from the lab. Another bolus of D10W was given and glucose infusion rate increased, but follow-up glucometer 30 minutes later was still low at 2.3 mmol/L. A glucagon infusion was started with subsequent improvement in blood glucose levels.

Later in the evening on the day of admission, the infant exhibited clinical seizures that were also detected electrically on the cerebral function monitor. She was treated with phenobarbital, phenytoin, midazolam and pyridoxine and the seizures finally came under control. She required re-intubation due to respiratory depression from the medications and remained ventilated on low settings.

An MRI was performed on November 27, 2011 and revealed extensive areas of restricted diffusion highly suggestive of hypoxic-ischaemic injury. The report also stated that the findings would be consistent with coexistent hypoglycaemic injury. Electrophysiologic studies revealed absent somatosensory and visual evoked potentials. The infant remained poorly reactive and seizures returned after some of the anticonvulsants were weaned.

The parents were counselled that there was evidence of extensive, severe CNS injury and that the prognosis was very poor. In consultation with the parents, a decision was reached to withdraw life-sustaining medical therapy. The ETT was removed on the morning of December 2, 2012 and the infant died two hours later.

Post mortem

The major findings included hypoxic-ischaemic injury felt to be a consequence of the cardiopulmonary arrest that precipitated hospital admission. No clear cause for the arrest was identified and no bacteriologic or metabolic explanation was apparent on ancillary testing.

Death was attributed to: Multi-organ hypoxic-ischaemic injury
due to: Cardiopulmonary arrest of undetermined cause
Potential contributory factor: Intrauterine growth restriction

Discussion

This infant died at eight days of age after withdrawal of life-sustaining medical therapy following evidence of severe CNS injury. She was born at term in good condition after an induction of labour for decreased fetal growth. She was discharged home at four hours of age and examination by the midwife at home the next day was considered normal.

Later that evening however, the infant became irritable and difficult to settle. By the next morning, she became lethargic and off-colour and was taken to hospital for assessment. Upon presentation, she was limp, cyanotic and unresponsive. After initial improvement with endotracheal intubation, she had what appeared to be a cardiorespiratory arrest and required extensive resuscitative efforts to revive her. Although her cardiorespiratory status subsequently improved, she had recurrent seizure activity and showed evidence of severe CNS injury that was confirmed by radiologic and electrophysiologic studies at the children's hospital. The prognosis was considered very poor and she died after withdrawal of life-sustaining medical therapy at eight days of age.

The cause of the infant's severe CNS injury is unclear. The pathologist performing the post mortem examination attributed it to hypoxia-ischaemia from the cardiopulmonary arrest the infant suffered after arrival at the hospital. However, the arrest was short-lived, blood gases taken shortly thereafter failed to reveal severe metabolic acidosis and there was little evidence of other end-organ damage. If hypoxia-ischaemia from an arrest were to cause CNS injury of the extent apparent with this infant, one would expect a much more deranged blood gas picture shortly after the event and evidence of collateral organ damage. The extent of the CNS injury is difficult to explain and the reason for the arrest remains unclear.

Acute life-threatening events in newborns are most commonly due to infectious, respiratory, cardiac or metabolic causes. An exhaustive work up performed to identify the cause in this infant's case was inconclusive. Sepsis was ruled out and there was no evidence of an inborn error of metabolism. Post mortem examination showed no evidence of congenital heart disease or pulmonary abnormality.

One plausible explanation for the infant's presentation and

subsequent course might be severe hypoglycaemia.

The infant was born small for her gestational age which is a known risk factor for hypoglycaemia. There was some suggestion that she did not receive much in the way of oral intake after discharge which could also increase the risk of hypoglycaemia. She was described as very irritable and difficult to settle on her second evening at home which could have represented signs of hypoglycaemia. Likewise, the lethargy exhibited the next morning could have been due to the same problem. When she arrived at hospital, her clinical condition could be explained by marked hypoglycaemia and the arrest could have been a consequence of a hypoglycaemic seizure.

The first blood glucose taken at Hospital A was very low at 1.5 mmol/L and remained low despite administration of intravenous glucose. The blood sugar did not normalize until a glucagon infusion was started at the children's hospital. Critical bloodwork taken at the time of a hypoglycaemic episode showed evidence of hyperinsulinemia. It is possible that prolonged, significant hypoglycaemia was the principal cause of the baby's severe CNS damage.

An MRI performed at the children's hospital showed extensive hypoxic-ischaemic injury and the report stated that there could be coexistent hypoglycaemic injury. While there are some patterns on MRI that are more strongly associated with hypoglycaemic injury than others, the patterns can be variable. Perhaps the best explanation for the clinical and radiologic picture is that the damage was caused by the combined effects of severe hypoglycaemia and hypoxia-ischaemia from the arrest.

Questions were raised about the early discharge from hospital of this infant and the postnatal management. The infant was born after an induction of labour for apparent decreased fetal growth and the infant did indeed plot out at only the third percentile for weight at birth. As such, she would be considered at risk for hypoglycaemia and postnatal glucose checks would be indicated. While most small for gestational age infants will have either no significant or only mild transient hypoglycaemia, some have a severe form, usually associated with hyperinsulinism. According to the College of Physicians and Surgeons of Ontario (CPSO) guidelines, small for gestational age infants should have their blood glucose monitored at two hours of age and then every three to six hours. For small for gestational age infants,

testing may be discontinued after 36 hours if feeding has been established and blood glucose levels remain at 2.6 mmol/L or higher.

As such, small for gestational age infants are not candidates for early discharge and need to be carefully assessed for the adequacy of their intake. This infant did not have her glucose levels checked in hospital or after discharge. There was some suggestion that her intake of milk may have been low. Although it is not certain that hypoglycaemia was the root cause of this infant's illness, it is possible that, if the recommended screening guidelines had been followed, significant hypoglycaemia would have been detected early, creating an opportunity for effective treatment and prevention of sequelae.

Recommendations

1. Paediatric care providers are reminded of the importance of recognizing infants at risk for hypoglycaemia and initiating appropriate screening protocols to detect significant hypoglycaemia.
2. Hospital A should create and implement processes for the detection of infants at risk for hypoglycaemia and the activation of appropriate screening protocols.

Case: 2012-N-18
OCC File: 2010-974

History

The mother was a 22-year-old G3P2 with an EDD of March 8, 2010 by dates and ultrasound at nine weeks gestation. The pregnancy was complicated by first trimester bleeding, nausea and vomiting requiring Diclectin®. She received Rh immune globulin at the time of the first trimester bleed. Medications in pregnancy included valproic acid 250 mg by mouth three times daily for treatment of epilepsy. Her IPS was negative for spina bifida. At her first prenatal laboratory investigations, her valproic acid levels were found to be sub-therapeutic. Her hemoglobin was 132 g/L. The 19 week anatomic ultrasound showed no neural tube defect.

Past obstetrical history was significant for two previous uncomplicated pregnancies with spontaneous vaginal deliveries at term.

On January 20, 2010 at 33 weeks 2 days gestation, she was seen in consultation by the obstetrics group located in her community. Her valproic acid dose was increased to 500 mg by mouth twice daily. A repeat dose of Rh immune globulin was also ordered. Prior to that time, her prenatal care was received at a high risk tertiary clinic.

On January 24, 2010 at approximately 1915 hours, she noted a gush of fluid per vagina. When she looked, it appeared to be a gush of blood and clots. She then started to have cramps, so an ambulance was called. The ambulance attendants noted a 'large amount of blood' loss. At 1942 hours, she arrived at the emergency department of Hospital A.

Upon arrival in the emergency department, she was noted be "shocky" and have a heart rate of 108 bpm, blood pressure of 112/68 mmHg, respiratory rate of 18 per minute and oxygen saturation of 100%. The FHR was 118 bpm. There was a large amount of blood per vagina and she was 4cm dilated. Contractions were two minutes apart, lasting 45-60 seconds.

Two IVs (one 18 gauge and one 20 gauge) were started and normal saline was infused 'wide open' through each, and immediate consultation with the referring centre undertaken. As Hospital A did not provide obstetrical services, arrangements were made for urgent transfer to Hospital B where obstetrical services were provided.

Upon arrival at Hospital B at approximately 2050 hours, the patient was "hemodynamically stable and the FHR tracing was normal." Dictated notes indicated that the patient showed "no signs or symptoms of severe anemia." Written documentation of blood pressure upon arrival could not be found in the records provided for review. The baseline FHR was 160 bpm with moderate variability. The bleeding was noted to be a "small amount" and she passed a "small clot." There was no documentation in the records provided regarding the total amount of intravenous fluid that had been administered.

The patient was "uncomfortable" with contractions and hemoglobin was noted to be 88 g/L. Her cervix was 4 cm dilated and 80% effaced. At 2143 hours, she was admitted to Labour and Delivery and an epidural was inserted for analgesia. There was no documentation regarding the time course of administration of the initial epidural medications. The infusion was started at a rate of 14 cc/hr. Maternal blood pressure was 109/57mmHg. Maternal heart rate could not be detected on the tracing strip and was not otherwise recorded on the records provided. At 2147 hours, the maternal blood pressure was 95/49 mmHg and at 2151 hours, the maternal blood pressure was 80/46mmHg. At 2153 hours, the maternal blood pressure was 84/40 and the IVs were both opened to administer a 500cc normal saline bolus.

At 2154 hours, just after the epidural, the FHR slowed to 60-80 bpm. At 2155 hours, the maternal blood pressure was 74/48 mmHg, maternal heart rate was 80-90 bpm and she was 5 cm dilated. Ephedrine was administered, along with oxygen and change of position. There was no documentation in the records provided regarding what route or dose was used for the ephedrine. At 2203 hours, the maternal blood pressure was 130/73 mmHg and the epidural infusion rate was decreased to 10 cc/hr. An artificial rupture of membranes was completed and

a fetal scalp clip applied. The FHR was 60-80 bpm and distinct from maternal heart rate. By 2210 hours, the FHR was atypical and decelerations were down to 60 bpm, lasting 40 seconds, without change in the cervical dilatation.

At 2230 hours, the decision was made to proceed to Caesarean section. The maternal blood pressure was 105/61 mmHg and additional medication was administered via the epidural to achieve surgical anesthesia. At 2239 hours, the patient was moved to the operating room and delivery took place at 2256 hours. The baby was flaccid at birth. At the time of the delivery, the placenta was delivered spontaneously with a large amount of clot behind the placenta.

Both the arterial and venous cord gas samples were clotted and could not be analyzed.

Neonatal course

The baby was born without heart beat or respirations. Apgars at one and five minutes were 0. Aggressive resuscitation was started at delivery as the team was in attendance. At seven minutes of age, the heart rate was in the 130s. The Apgar at 10 minutes of age was 3. The heart rate continued and spontaneous respirations were noted at 31 minutes of age. The infant was still limp and despite ventilation, colour and tone did not improve. The infant had a hemoglobin of 104 and was transfused.

The tertiary level NICU transport team was contacted at 0038 hours and arrived at 0400 hours. The infant arrested several times prior to, and after, the arrival of the transport team. The transport team repeated the intubation of the infant and colour improved. The infant arrested again and was resuscitated. The infant was unresponsive to pain. Repeated gases showed severe metabolic acidosis with low oxygen saturation. The infant could not be stabilized for transport. After extensive discussion with the parents, the decision was made to withdraw care. The infant died at seven hours and 48 minutes of age.

Post mortem

Pathology review of placenta was consistent with abruption.

Discussion

The incidence of placental abruption is 1:150. In about 1:800-1600 cases, the abruption is severe enough to result in death of the baby.¹ The exact cause of abruptio placenta may be difficult to determine. In this case, the mother did not have any known risk factors.

At the time of presentation, the gestational age was 33 weeks and six days. The FHR tracing was normal and the mother was "hemodynamically stable"; however, her hemoglobin was 88. The cervix was 4 cm dilated and she was contracting regularly. The decision was made to monitor the mother and baby continually and expect a vaginal delivery as she had had two previous uncomplicated term deliveries in four and six hours and this was a preterm infant.

An epidural for labour analgesia is common. However, it also results in a sympathetic blockade that may cause maternal hypotension. Given that the patient was hemodynamically stable (her BP was 109/57 and maternal heart rate not found in the documentation) at the time of epidural insertion, there was no absolute contraindication to epidural analgesia. However, the history of vaginal bleeding, as well as the maternal anemia noted at admission, were both indicators of potential hypovolemia which could aggravate hypotension induced by sympathetic blockade. The post-epidural sympathetic blockade made it more difficult to determine if the cause of the fetal bradycardia was maternal hypotension resulting from the side-effects of the epidural or from worsening abruption.

When the mother's condition worsened and the FHR tracing became abnormal, resuscitation was started. The abnormal FHR tracing persisted despite aggressive resuscitation efforts. The time from the bradycardia post epidural to delivery was 62 minutes. The time from decision to proceed to Caesarean section to delivery was 26 minutes. If fetal bradycardia is present and the time from decision to delivery is greater than 20 minutes, the neonatal outcomes are worse.²

Recommendations

1. In the context of abruptio placenta, obstetrical care providers are reminded that immediate delivery should be strongly considered at the first signs of maternal and/or fetal compromise. This intervention may be lifesaving.
2. Obstetrical care providers and providers of obstetrical analgesia/anesthesia should be aware that “the most common side effect of epidural or spinal anesthesia is hypotension with functional hypovolemia.”³ In a clinical scenario where maternal hypovolemia and/or hypotension are already present or likely, alternative forms of maternal analgesia may be warranted, and general anesthesia should be considered as an option for management of Caesarean section.
3. Hospital B should perform an Internal Review of this case.

References

1. ncbi.nlm.nih.gov/pubmedhealth/PMH0001903
2. bestpractice.bmj.com/best-practice/monograph/1117/treatment/step-by-step.html
3. ncbi.nlm.nih.gov/pubmed/14739801

Case: 2012-N-19

OCC File: 2012-4696

Antenatal History

The mother of the deceased infant was a 24-year-old G2P0 with an EDD of April 11, 2012 based on last menstrual period. Her prenatal care was provided by a midwife. Routine prenatal laboratory investigations were normal. She was Rh negative and antibody screen negative. She declined IPS testing. Glucose challenge test was normal. She was GBS positive and was not sensitive to clindamycin. She was allergic to penicillin.

In her past medical history, the mother had a dilation and curettage.

On April 15, 2012, the woman presented to her midwifery clinic after reporting leakage of fluid. The woman was assessed by her midwife for possible rupture of membranes. An indicator test for amniotic fluid was negative. The use of sterile speculum and assessment of ferning was not documented.

Course in Labour and Delivery

Labour onset began on April 17, 2012 at 1300 hours at 40 weeks and six days gestation. The woman was admitted to hospital under the care of her midwife at 1449 hours. Contractions were every two to three minutes, lasting 55-65 seconds and moderate intensity. The cervix was 3 cm dilated, 80% effaced and membranes intact. The FHR was 156 bpm. Maternal pulse was 113 bpm and white blood cell count was 27.7 with a few toxic granulations. The woman was afebrile.

An obstetrical consultation was obtained at 1640 hours for GBS prophylaxis. As the woman was allergic to penicillin and the GBS was resistant to clindamycin, she was prescribed vancomycin 1g IV then 500mg IV q12h [every 12 hours] in labour.

The woman became uncomfortable with her labour and an epidural was started at 1750 hours.

At 1805 hours, the cervix remained unchanged at 3cm dilation. Despite an effective epidural, a mild maternal tachycardia persisted. The FHR baseline remained in the 150-160's. There were sporadic FHR decelerations which responded to maternal oxygen and repositioning.

At 1832 hours, the contractions were noted to be every three to four minutes lasting 50-60 seconds with moderate/strong tone.

At 2013 hours, the woman was given acetaminophen for a headache. At 2043 hours, her temperature was 37.2°C.

At 2105 hours, an artificial rupture of membranes was carried out for scant clear fluid. The cervix remained 3cm dilated, 80% effaced with the vertex at spines-1. The baseline FHR was in the 160-170's.

At 2116 hours, the maternal temperature was 37.1°C. Labour parameters continued much the same until 0200 hours when the cervix had progressed to 5cm dilation, 80% effaced and the vertex at spines-1. There continued to be sporadic decelerations.

The obstetrician was consulted at 0034 hours on April 18, 2012 for variable FHR decelerations and failure to progress. The obstetrician attended at 0043 hours.

On examination, the cervix was unchanged at 5cm dilation and right occiput posterior position. A fetal scalp clip was applied and a 500cc bolus of fluid was given for concerns of mild dehydration.

At 0054 hours, the amniotic fluid was noted to be lightly meconium stained.

The woman was reassessed by the obstetrician at 0220 hours and oxytocin augmentation was ordered. Care was formally transferred to the obstetrician. Oxytocin was started at 0235 hours.

At 0242 hours, her temperature was 37.7°C. She was given acetaminophen and a fluid bolus.

Starting at 0244 hours, the decelerations became consistent with a late component. The FHR deceleration pattern became severe at 0314 hours. The oxytocin infusion was stopped at 0329 hours. The tracing was reviewed by the obstetrician. On examination, the cervix was 7cm dilated.

At 0336 hours, the cervix was 8cm dilated. The decision was made to proceed to Caesarean section. While preparations were being made in the operating room, another patient in the unit was attended by the obstetrician for vaginal delivery. That baby

was delivered by vacuum extraction at 0358 hours.

At 0404 hours, the obstetrician was called to the operating room "stat" for a FHR of 50 bpm. An emergency Caesarean section was performed under epidural anaesthesia and completed under a general anaesthetic.

The woman was delivered of a 3670 g female infant at 0422 hours. Apgars were 0 and 0 at one and five minutes. A "code pink" was called.

The arterial cord gas pH was 6.62 and the venous pH was 6.34. The baby was suctioned for thick meconium and intubated by the respiratory technologist who was in attendance and the paediatrician arrived at 0438 hours.

A heart rate was established at 0502 hours, but the baby remained bradycardic, hypotensive and unresponsive. After the paediatrician discussed, with the parents, the baby's grave prognosis, resuscitation was discontinued and the baby was pronounced deceased at 0705 hours.

Post mortem

At autopsy, overall growth parameters were normal for gestational age.

There was congenital hemorrhagic bronchopneumonia, heavy, involving all lobes of the lung.

Examination of the placenta revealed heavy acute chorioamnionitis with necrotizing funisitis. There was moderate meconium staining of the fetal membranes. No confirmation of bacterial organism was established.

A rectal swab was negative for GBS. A blood culture taken at the time of resuscitation showed no growth after five days. The cause of death was heavy ascending infection with florid hemorrhagic bronchopneumonia.

Discussion

This baby died from intrapartum asphyxia due to a severe intrauterine infection. Although post mortem cultures were negative for GBS, the presentation was consistent with GBS infection. The mother was known to be positive for GBS and was

treated according to protocol with vancomycin in labour due to her allergy to penicillin and GBS resistance to clindamycin.

Although antibiotic prophylaxis has dramatically decreased the incidence of neonatal GBS infection, it does not completely eliminate it. Although not clinically obvious, it is possible in this case that there was an established infection at the time the woman presented in labour. Non-specific findings for infection include the maternal tachycardia, the mild fetal tachycardia and the elevated maternal white blood cells with a left shift at the time of admission. The highest maternal temperature recorded during labour was 37.7°C although the woman did receive acetaminophen on two occasions.

If membrane rupture had occurred two days before labour, the risk of GBS infection would have been much greater. Assessment at the time and subsequent ARM in labour makes premature rupture of membranes unlikely. GBS infection is known to occur through intact membranes.

The FHR tracing became abnormal after 0244 hours. The nurse did not notify the obstetrician until 0316 hours. Even though attended by the obstetrician at 0316 hours, the oxytocin was not discontinued until 0329 hours. The decision to proceed to Caesarean section was not made until 0340 hours. There was a further delay as a result of the obstetrician's decision to proceed with the delivery of another labouring patient in the department.

An external review of this case was conducted by the hospital. The invited reviewers consisted of an obstetrician, a midwife and a nurse. The external review of this case concluded that the delivery should have been given priority and consideration should have been given to calling in the second on call obstetrician. The nurse and the obstetrician failed to recognize the severity of the FHR changes. The issues raised by the MPDRC were also identified by the reviewers.

Recommendations

1. Obstetrical care providers are reminded of the SOGC guideline on intra-partum fetal surveillance and the identification and management of an abnormal FHR tracing. (available online at: <http://sogc.org/wp-content/uploads/2013/01/gui197CPG0709r.pdf>)

Case: 2012-N-20
OCC File: 2012-5872

History

The mother was 33 years old, 5'8", 161 pounds and Rh negative. Her obstetrical history was G2P1. In 2007, she had a spontaneous vaginal birth at 39 weeks gestational age. There was difficulty conceiving in her next pregnancy with menstrual cycles being irregular, so clomiphene was used for ovulation induction.

There was no significant medical, family or surgical history documented in the file.

Antenatal History

The woman planned a hospital birth in the care of midwives. Care commenced at 10 weeks and four days gestation and consisted of 11 antenatal visits. Prenatal laboratory investigations were normal including IPS, public health screening and 50 gram OGCT. An ultrasound at 6 weeks and five days confirmed dates and FHR. Routine ultrasound at 13 weeks and four days gestation for nuchal translucency and at 19 weeks and one day for normal fetal morphology and a posterior placenta clear of the cervix. She received Rh immunoglobulin at 28 plus weeks. She was GBS negative at 36 weeks and two days gestation.

Course in Labour and Delivery and Postpartum

Labour commenced on May 13, 2012 at 38 weeks and five days gestation with spontaneous rupture of membranes at 1400 hours. The woman paged the midwife and made a plan to page again when regular contractions began.

At 1600 hours, her contractions began and at 1845 hours, she paged the midwife as the contractions were every two minutes for 50 seconds, and very strong. When she paged her midwife in active labour, she was told the hospital of her choice (where she was pre-registered), was not available to her. The alternative hospital offered was not optimal, and the parents discussed, and decided, to remain in the home.

The midwife arrived at the home at 1945 hours to find the woman 8cm dilated, soft 80% effaced, vertex at -2 spines, show on glove and no fluid noted. The issue of going to hospital was

re-addressed to ensure the parents were making an informed decision. The parents stated they would remain at home.

A second midwife was called and arrived with a student at 2015 hours. Contractions were every two minutes for three to 50 seconds and FHR was normal. The woman got into the tub and remained there for one hour, until 2115 hours when she left the tub with rectal pressure.

At 2145 hours, she was fully dilated with vertex at +2 spines and having spontaneous pushing urges.

At 2207 hours, the head was on perineum and FHR returned to normal on intermittent auscultation following a deceleration to 90 with fast recover to 140 bpm.

At 2209 hours, there was a spontaneous vaginal birth with a nuchal arm presenting after 24 minutes of pushing. The woman was delivered of a female weighing 2950g, 51 cm in length with a head circumference of 31 cm.

At 2210 hours, 10 IU oxytocin IM was administered and delivery of placenta and membranes occurred at 2215 hours.

A labial split and perineal tear was repaired and there was normal bleeding and an uneventful immediate postpartum period followed.

The baby girl had Apgar scores of 9 and 9 at one and five minutes. She latched and was nursing by 2255 hours with skin-to-skin care being done. The baby's temperature was 36.5°C at 2307 hours and 37.1°C 0015 hours with a normal newborn examination documented. Vitamin K and eye prophylaxis were administered.

At 12 hours of age, the midwife visited and documented a normal newborn female infant and a well postpartum mother. The baby was reported as being mucousy, but otherwise well. The baby's temperature was slightly low at 35.9°C.

The mother was concerned about keeping the baby warm, so she dressed her more warmly and warmed the bassinet prior to putting baby down.

The baby fed well during the day on May 14 and again at 0200

and 0500 hours on May 15, 2012. At the 0900 feed on May 15, the baby was not keen to latch or suck, even with stimulation. The parents noted, "it seemed like the baby had the hiccups and that the breathing seemed "rhythmic." The baby was "alert and looking around."

At 1000 hours on May 15, 2012, at approximately 36 hours of age, the mother found the baby to be breathing very poorly and to have a "yellow" colour. They immediately called 9-1-1 and the father commenced mouth-to-mouth ventilation under the direction of the Emergency Medical Services (EMS) dispatch until the EMS crew arrived at 1003 hours.

EMS records indicate that the baby was initially given blow-by oxygen by the fire crew. At 1005 hours, the EMS team described the infant as pale with some cyanosis around the mouth and distal extremities. She had the occasional agonal breathing, but was deemed to be in respiratory arrest. She was unresponsive and pupils were non-reactive. Respiratory support with bag and mask ventilation was commenced. Heart rate was measured at 80 beats per minute.

The baby was moved to the stretcher in preparation for departure to hospital but, when reassessed, her heart rate was found to have dropped to 40 beats per minute. Chest compressions were instituted and she was moved to the ambulance. She was intubated with a 2.5 ETT at 1016 hours, but despite appropriate placement and continuing CPR, her heart rate failed to improve. Correct ETT positioning was confirmed by auscultation and visualization.

One dose of endotracheal epinephrine was administered and an intraosseus catheter was placed. An infusion of normal saline was commenced and three additional doses of epinephrine were given via the intraosseus line.

At 1038 hours, the ambulance arrived at the hospital's emergency department after alerting staff of their impending arrival. The baby was being ventilated via the ETT and receiving cardiac compressions. Normal saline was infusing through the intraosseus line. A full medical and nursing team had been assembled to receive the infant. The baby was mottled and flaccid with fixed dilated pupils, no spontaneous movements and no audible heart rate. The ECG monitor showed "non-organized

PEA [pulseless electrical activity]" as there was a palpable pulse only with the external cardiac compressions. Six more doses of epinephrine and three boluses of normal saline were given and the ETT was changed to a larger size (3.5). Despite these efforts, vital signs could not be restored and she was pronounced dead at 1056 hours. The coroner and police services were notified as per protocol.

The baby's blood type was A negative so RH immunoglobulin was not required for the mother.

Post mortem

There was no examination of the placenta as this was a homebirth and the parents had disposed of it on the morning of the death.

The most important finding at autopsy was that of pulmonary hyaline membranes. This was mild and patchy, but found throughout both lungs. The causes of this are mechanical ventilation or an infective agent.

This baby did not receive mechanical ventilation. Blood and lung cultures were negative for GBS. The Gram stains from the lungs showed mixed bacterial growth including gram-negative rods which can produce an endotoxin. It was inferred that the endotoxin-producing bacteria reached the alveoli during the birthing process with a possible source being the bath water in which the woman was immersed during labour.

The cause of death was "perinatal infection by endotoxin-producing bacteria."

Discussion

This baby died at 1 ½ days of age after a sudden collapse at home. She had been born in good condition and had been feeding well until the morning of the arrest. She had been a bit mucousy and was slightly hypothermic on Day 1, but was otherwise felt to be normal. She presented with relatively rapid onset of poor feeding, abnormal respirations and ultimately a full respiratory arrest. Despite quick recognition and appropriate, rapid response from the parents and EMS, she could not be revived. Post-mortem examination revealed the unusual finding of hyaline membranes in the baby's lungs suggesting the death

most likely resulted from a perinatal bacterial infection.

Early-onset sepsis is not uncommon in newborns, occurring in approximately 1-2 per 1000 live births (Baltimore RS et al, Pediatrics 2001). Approximately half of these cases are caused by GBS while most of the rest are due to gram negative organisms such as E Coli. Most, but not all cases, are associated with risk factors such as prematurity, chorioamnionitis and/or prolonged rupture of the membranes. Presentation may be insidious or fulminant with almost all affected infants presenting within the first 48 hours after birth.

Although not classical in presentation or evolution, it is most likely that this baby's death was caused by early onset sepsis. Other causes of sudden collapse in a newborn were effectively ruled out and the detection of hyaline membranes in post-mortem lung samples is strongly suggestive of this diagnosis. While it is uncharacteristic to have such a rapid, downhill course and for the baby to be unresponsive to expeditiously applied resuscitative efforts, infection remains the most plausible root cause for her sudden deterioration and subsequent demise. Earlier detection and treatment may have changed the outcome but, with the relatively normal clinical appearance in the hours leading up to her collapse and the very fulminant course, this is unlikely.

Possible explanations for a baby's poor response to resuscitation include an incorrectly positioned ETT or use of high airway pressures leading to tension pneumothorax and pneumomediastinum. However, in this case, notations regarding the baby's ventilation indicate that those managing the airway checked and verified proper ETT position on several occasions. Regarding the pneumothoraces and pneumo-mediastinum, a malpositioned ETT (i.e. perforating the esophagus), or use of high pressures could result in extravasation of air, tension in the thorax and inability to effectively ventilate or oxygenate. However, there was no indication of technical difficulty with the initial intubation, evidence of proper chest excursion and air entry when assessed and no indication of laryngeal, esophageal or tracheal trauma on post-mortem examination.

While possible that extrinsic gas compressing the lungs or mediastinum may have impeded resuscitative efforts, the sudden collapse, poor condition even prior to resuscitative

attempts and underlying serious infection, makes it unlikely that the outcome could have been improved.

Concerns identified by parents

The parents of the deceased infant have raised several concerns about their daughter's care and the documentation of events. Their goal overall of this review is to improve their understanding of what befell their child and to see if any changes might be put in place to prevent a future occurrence. Responses to their concerns are arranged according to the source.

1. Pathologist's Report

- a. Several corrections have been requested to ensure that the account is factual.
- b. There is no record in the files pertaining to the blood test taken from the baby after birth. Presumably, the test being done was to determine the baby's blood type to determine whether an Rh immune globulin injection to the mother was indicated. Results of this test would have no relevance to the investigation of the baby's death.

2. Midwives' Records

- a. Parents were concerned about the incomplete documentation and possible failure to appreciate serious clinical signs, specifically being mucous and being slightly hypothermic. Both of these signs are common in newborns and not generally indicative of a pathological state. However, ongoing monitoring to ensure resolution is indicated. In this baby's case, it appears that she cleared her mucous after the earlier findings. Regarding the low temperature, it may have been prudent to suggest follow-up temperature checks but, in a baby who appears otherwise well, general advice on temperature maintenance would be considered appropriate.

3. EMS Records

- a. Parents expressed concern in this and the next section about the use of a size 2.5 ETT for resuscitation in this case. A 3.5 ETT would be the proper size for an average sized

term newborn and the reason for the choice of a 2.5 tube is unclear. However, a 2.5 tube should have been sufficient to adequately oxygenate and ventilate a baby of this infant's size even though it is not optimal for prolonged ventilation or if lungs are very stiff. It is unlikely that earlier placement of a 3.5 versus 2.5 ETT would have improved the outcome.

4. Hospital Records

a. The parents were concerned with the quality and detail of the records taken during their baby's short time in the Emergency Department at the hospital. While it is fair to say that some of the records are difficult to decipher and some details that might have been included are missing, overall, the records provide a good description of what transpired. It is clear that the baby was vital signs absent upon arrival and the notes indicate that the team applied appropriate, expeditious steps to try to revive her. Respiratory Technologist notes and a clear description of the intubation attempts were not part of the record provided for this review.

Additional comments: The pathologist and coroner questioned the possible role of water immersion in labour in the causation of this baby's infection.

Water immersion in labour is a widespread practice for non-pharmacologic pain relief in labour. Although there is a case series describing adverse outcomes, including sepsis after water immersion in labour, a cause and effect relationship remains tenuous. According to the most current Cochrane Review of water immersion in labour, "evidence suggests that it reduces the use of epidural or spinal anaesthesia. There is no evidence of adverse effects to the fetus/neonate or woman from labouring in water or waterbirth." (Cluett and Burns, Immersion in water in labour and birth (2009), Cochrane Database Syst Rev. 2009 Apr 15; (2):CD000111).

In this case, the mother spent one hour of her active labour in the bathtub of her home. She did not deliver in the water. She was known to be GBS negative at 36 weeks gestational age and had ruptured membranes for five to six hours prior to entering

the bathtub. The mother was not febrile prior to, or after the time spent in the bathtub. Given the short period of time spent in the bathtub, and time from tub to delivery, it seems unlikely that the water was the source of infection.

Recommendations

1. The Canadian Pediatric Surveillance Program (CPSP) initiative collecting data on early onset neonatal sepsis should consider adding information regarding maternal use of water immersion during labour and delivery.

Case Summaries: Stillbirths

Case: 2012-S-1

OCC File: 2011-5114

History

The mother was a 34-year-old Gravida 1 female. She and her partner had emigrated from North Africa. She worked as a "packager" and her partner was unemployed. The prenatal records indicated that there was poor social and familial support for the couple.

The mother was first seen in midwifery care on December 23, 2010 at 16 weeks and three days gestation. She had been seen by a family physician where she was diagnosed with and treated for a urinary tract infection. Ultrasound, initial bloodwork and

IPS tests were done. The woman also had a history of spotting.

The mother was 165 cm (5'4") and 46 kg (101 pounds) pre-pregnancy and had a normal blood pressure of 108/76 on her first visit. The results of testing were requested from the family doctor. There was a concern about recurrent urinary tract infections and in October 2010, she was treated for a significant growth of E. coli. A subsequent urinalysis on December 7, 2010 was normal. Urinalysis done on December 22, 2010 showed only 2+ blood, but was otherwise negative. The culture showed a growth of E. coli which was not considered to be significant, although she was given additional treatment. IPS testing was low risk for Down syndrome.

Serial ultrasound examinations were as follows:

Date	Gestation	Findings
Nov. 10, 2010	10 weeks 3 days	
Nov. 25, 2010	11 weeks 2 days	Normal NT
Dec. 29, 2010	16 weeks 6 days	Early for detailed scan - incomplete
Jan. 12, 2010	18 weeks 2 days	Placenta anterior, marginal previa, otherwise normal scan
Feb. 16, 2010		Placenta clear of cervical os
March 16, 2010	27 weeks 1 day	EFW of 921.3 (20thpercentile), head/abdominal circumference ration 1.19 (80thpercentile), AC equivalent to 25 weeks and 4 days, amniotic fluid volume normal.

On March 9, 2011, at 27 weeks gestation, her blood pressure was first elevated at 130/70. The symphysis fundal height (SFH) was 23, so an ultrasound was ordered at that time and performed a week later. The next office visit was on April 20, 2011 (6.5 weeks later) at 33 weeks and two days gestation. Her blood pressure was further elevated at 130/82 and the SFH was only 26. The medical notes indicated that the previously performed ultrasound was reviewed at that time and that further ultrasound assessment was not considered despite the smaller than expected interval growth.

On April 21, 2011, follow up with the woman indicated that her blood pressure was 128/80.

Until this time, there had been no protein or dipstick urine testing. Urinalysis and culture were ordered on April 21, 2011. There was no documentation on the chart indicating the reasoning for getting the urinalysis, monitoring the blood pressure, warning signs of fetal movement and pain bleeding, etc.

On April 27, 2011, the results of the urinalysis revealed 3 grams per liter of protein, but no other abnormality of the urine was noted and the culture was negative. The woman was contacted and advised to attend to the hospital for monitoring and consultation. The chart notes indicate that the woman had not been reporting any concerns and felt fetal movement earlier in the day and had been documenting fetal kick counts.

Shortly after arrival at the hospital, the woman was asked to provide a urine sample. At 1551 hours, an attempt was made to pick up the fetal heart and conduct a NST. Nursing assistance was requested and an obstetrician was subsequently called at 1555 hours because of concerns about a low fetal heart. Bedside ultrasound was done to confirm a fetal heart in the 80 range and by 1600 hours, a decision was made to proceed with a Caesarean section. The woman was taken to the operating room at 1608 hours. There were no maternal vital signs recorded pre-operatively.

The vital signs on the anesthetic record taken in the operating room showed an initial blood pressure of 160/110. It is recognized that this reading was taken during a very stressful time as the woman had been induced with a general anesthetic.

At 1614 hours, (14 minutes after the decision to proceed with a Caesarean section), a female infant weighing 1433 grams was delivered by low segment Caesarean section.

The placenta was removed manually and following the placenta, there was "a huge retroplacental clot occupying almost the entire placenta." The rest of the procedure was unremarkable. A pediatric team was in attendance and attempted to resuscitate the infant until 1641 hours.

The infant had Apgars of 0 and showed no signs of life after 25 minutes despite appropriate attempts at resuscitation. The arterial cord PH was 6.87 with a base deficit of 21.5.

Maternal blood testing revealed normal platelets, normal liver function tests and coagulation studies. Test results did not include a urinalysis even though the woman had provided a urine sample upon arrival at the hospital. The sample may not have been tested as the emergency Caesarean section took place shortly after arrival at the hospital.

The mother's subsequent care in hospital was unremarkable and her blood pressure did not require treatment until after discharge. Post partum pressures recorded in the recovery room were only mildly elevated.

Post mortem

An autopsy was not performed. The placenta was examined and indicated:

500 gram, large adherent clot 10 X 8 cm occupying 50 to 60% of the placental disc; marginal insertion of umbilical cord.

Discussion

This woman, in her first pregnancy, had appropriate initial assessment. There was concern about early urinary tract infections, however as the pregnancy progressed, this did not have further impact. At 27 weeks gestation, appropriate arrangements were made for an ultrasound due to a small SFH. From the chart, it is not clear when the results were reviewed by the caregiver. It was not until six weeks later during a visit with

the patient that the results were reviewed. During that visit, the blood pressure was elevated though still mild as compared to earlier in the pregnancy. Clinical estimate of fetal growth with an SFH of 26 at 33 weeks gestation may have raised concern that the growth of the fetus was falling off. The subsequent birth weight of 1433 gram (less than the 3rd percentile) at 34 weeks, confirms the findings.

A formal urinalysis and repeat culture revealed proteinuria. This was reviewed a week after it was detected. It cannot be determined if earlier review and consultation of the findings would have changed the subsequent outcome. On April 27, 2011, when the woman was seen in the hospital with significant fetal bradycardia, it was not clear whether she had been having any symptoms to suggest abruption that was subsequently found at the time of delivery. Blood tests relevant to pre-eclampsia were normal and there was no repeat urinalysis done. There was no recorded blood pressure prior to the time when anesthesia was being induced, so it is not clear whether a further significant increase of blood pressure had occurred in the week since she had last been seen in the clinic.

The emergency Caesarean section was performed expeditiously. While pre-operative vital signs may have been taken, these were not included in the charts provided for review.

An acute abruption, like in this case, can occur without antecedent symptoms. It is unclear whether the woman had any pain or perception of change of fetal movement. It is not known whether there had been a significant increase of the blood pressure over the previous week which would have been a predisposing factor to an abruption.

The attempt at resuscitation of the infant after delivery was appropriate although with Apgars of 0 and with significant acidosis as documented from the cord gases, there was no hope of recovery. The patient's subsequent course was unremarkable. Her blood pressure recorded in the recovery room was only mildly elevated and there was no need for antihypertensive therapy.

This woman did have mild pre-eclampsia. There was significant growth restriction which suggested placental insufficiency.

Elevated blood pressure in the week preceding delivery, as well as impaired placentation leading to growth restriction, would have been predisposing factors for abruption. However, as the abruption likely happened suddenly and without warning symptoms, closer surveillance (even ultrasound in the week prior) may not have altered the plan of care or altered the final outcome.

Recommendation

1. A Regional Coroner's Review should be conducted to review the care provided to this expectant mother. This review should consider: timing, communication between shared caregivers, monitoring of low symphysis fundal height (SFH) growth, flagging of follow-up appointments not attended, decreased fetal growth before hypertension, work/social issues (e.g. increased physical stress from standing and/or lifting and lack of social support networks), interval growth issues, appropriate and timely testing for proteinuria, and surveillance based on complete clinical picture.

Case: 2012-S-2
OCC File: 2010-9058

Antenatal History

The mother of this stillborn child was a 32-year-old G3P2 with an EDD of June 30, 2010. Her past obstetrical history included two previous uneventful pregnancies. The first was delivered of a 6 pound 7 ounce female at 38 weeks gestation in hospital. The second was delivered of a 6 pound 11 ounce female at 38 weeks at home. She had declined ultrasounds during the second pregnancy. Her past medical history and family history were non-contributory.

The woman came under midwifery care at 15 weeks gestation. Routine prenatal laboratory investigations were normal. She declined prenatal genetic testing and second trimester ultrasound although she stated that she would go for an ultrasound if clinically indicated. She declined a glucose challenge test. Her body mass index (BMI) at the initial visit was 34.

At her 32 week prenatal visit, the presentation could not be determined. She declined an ultrasound for this purpose. Her blood pressure was 160/78 with 2+ proteinuria. The management of pre-eclampsia was discussed.

At her 34 week prenatal visit, her blood pressure was 160/90 with 2+ proteinuria. Pregnancy induced hypertension (PIH) laboratory investigations and physician referral were declined. She also declined having her blood pressure checked twice a week.

At 35 weeks 1 day, her blood pressure was 145/85 - 160/90 mmHg. She agreed to a physician referral. The consultant recommended a "toxic profile" for diastolic blood pressure over 95.

At 35 weeks 5 days, her blood pressure was 145/85. The fetal presentation could not be confirmed on physical exam. A GBS culture was declined. Planned home water birth was discussed. She was advised of the need to go to the hospital if meconium was present in the amniotic fluid at membrane rupture.

She continued regular prenatal visits. She declined a BPP for post-dates at 41 weeks 1 day, but consented at 41 weeks and three

days gestation. The BPP scored 8/8. The fetal size was consistent with 37 weeks and one day. The risks of meconium and stillbirth associated with postdates was discussed.

At 42 weeks and one day, she declined another ultrasound and NST.

Course in Labour and Delivery

Membranes ruptured spontaneously at 1000 hours on July 18, 2010 at 42 weeks and five days for yellow meconium stained fluid. She was attended at home by her midwife at 1110 hours. As per the prenatal discussion, she was advised to proceed to the hospital because of the presence of meconium; she declined. She also declined antibiotic prophylaxis for unknown GBS status. The FHR was documented at 152 bpm with acceleration. Her contractions were irregular.

At 1210 hours, the meconium was noted to be thick. The FHR was 148 and 156 bpm. Transfer to hospital was again declined. The midwife left instructions to call when contractions became regular and to meet at the hospital if there was reduced fetal movement or bleeding.

The midwife called in at 2200 hours. Contractions had onset at 1700 hours and had been every four to five minutes since 2000 hours. She was again advised to go to the hospital because of the thick meconium and the risk of meconium aspiration.

The midwife attended the home at 2238 hours. The fetal heart was normal. The mother's BMI and labour positions caused some difficulty in auscultating the fetal heart. She got into her bathtub at 2310 hours having declined an assessment prior to entering the tub; she remained fully clothed. She was requested to get out of the tub at 0035 hours, but declined. She declined further FHR auscultation after 0015 hours and began to feel the urge to push. Further requests to get out of the tub were also declined.

At 0140 hours, she removed her clothes and at 0145 hours, with one push, she delivered into the tub of water. The baby was flat at birth. Apgars were 0, 0, 0 and 0 at one, five, 10 and 15 minutes. The cord was loosely around the neck once and there was a true knot in the cord.

The nose and mouth were suctioned for no meconium. PPV and CPR were commenced. The father was instructed to call for EMS. Resuscitation continued and EMS arrived at 0215 hours. The baby was transported to hospital and pronounced at 0234 hours. The parents and midwives were interviewed by police at the hospital and an autopsy was ordered.

Post mortem

The baby was a normally developed female weighing 3515 grams.

At autopsy, particulate meconium was evident within the alveoli. Lung culture grew heavy non-hemolytic streptococcus. The lungs showed evidence of being non-aerated. There was meconium staining of the fingernails.

There was recent, generalized and globally mild anoxic-ischemic encephalopathy with numerous foci of fresh perivascular hemorrhage.

The placenta weighed 446 grams.

There was a light growth of non-hemolytic Streptococcus. The membranes were meconium stained. There were no focal lesions in the parenchyma. A true knot in the cord was identified. There was no definitive evidence of myonecrosis of the umbilical vessels. Histologically there were scattered pigmented histiocytes. There was balloon change of the amniocytes and focal amnion sloughing and denudation. There was no evidence of villitis.

The cause of death was an anoxic-ischemic event recent due to or as a consequence of possible true knot in the cord.

Discussion

This baby died as a result of perinatal asphyxia causing fatal anoxic-ischemic encephalopathy. Although the true knot in the cord was identified at autopsy as a possible contributing factor, it was not tight and there was no stricture. Postdates pregnancy (>42 weeks), maternal hypertension and obesity are recognized as risk factors for stillbirth. Perinatal mortality (stillbirths plus

early neonatal deaths) for pregnancies >42 weeks is twice that at term. Meconium staining at autopsy and the placental weight below the 10th percentile are supportive of compromise due to postdate.

The midwives providing care in this case properly identified the concerns with postdate pregnancy. The client was repeatedly advised of the concerns, but the recommended tests of fetal surveillance and interventions were declined. These were well documented on the Antenatal 2 record, but the extent of the entries in this location made it extremely difficult to see the actual clinical picture of this woman. It may have been prudent for the midwives to have stayed with the client after membrane rupture to provide ongoing monitoring of the FHR, but the client's pattern of declining recommendations for monitoring and intervention were such that the outcome is unlikely to have been impacted.

Given the presence of meconium and the woman's refusal to go to the hospital, the midwives should have called the ambulance sooner in order to have the necessary resources to intubate the baby. Even if the paramedics had been able to intubate the baby, the outcome is likely to have not been impacted.

Recommendations

1. Obstetrical care providers are encouraged to use supplementary progress notes rather than excessive entries on the Ontario Antenatal Record 2.
2. Obstetrical care providers are reminded of the importance of adequate resources to provide endotracheal intubation if necessary when the amniotic fluid is meconium stained.

Case: 2012-S-3

OCC File: 2011-10126

History

The mother of this stillborn child was a 24-year-old G2P1 who presented in second stage labour at 36 weeks and four days gestational age on August 1, 2011. She was fully dilated with membranes intact and bulging. No fetal heart was auscultated. In the labour room, the membranes were artificially ruptured for

thick meconium. A fetal scalp clip was applied and no fetal heart beat was obtained. Twenty-two minutes after presentation, the woman had a spontaneous vaginal delivery of a macerated female weighing 5 pounds and 10 ounces. The placenta was retained and was removed manually in the operating room under general anesthesia. The diagnosis of placenta accreta was made.

The woman's prenatal course and interaction with the CAS is outlined in the following chart:

Date	Prenatal course	CAS involvement
March 10, 2011	First prenatal visit. Gestational age is 16 weeks. Mother admits to getting methadone 40mg once daily from the methadone clinic at the addiction centre. FHR is 164.	
March 11, 2011	Ultrasound gave estimated gestational age of 16 weeks and four days, placenta anterior and clear of os.	
April 12, 2011	Prenatal visit at 21 weeks gestational age. Treated with trimethoprim-sulfamethoxazole for persistent bacteriuria in pregnancy. Two previous prescriptions for amoxicillin 500mg by mouth, three times a day x seven days. The physician is uncertain if the patient took the amoxicillin or not.	
April 21, 2011	Ultrasound gave estimated gestational age of 23 weeks, placenta anterior and clear of os. Fetal anatomy was normal, gender not identified.	
May 5, 2011	No show for prenatal visit.	
May 11, 2011	No show for prenatal visit. Obstetrician sent note to nurse practitioner that mother had not shown for her last two prenatal visits. The obstetrician advised the nurse practitioner that mother had persistent bacteriuria and may not have been compliant with prescribed antibiotics. The obstetrician also advised the nurse practitioner that a notification was received from the addictions centre that the mother had tested positive for morphine and oxycontin.	

Date	Prenatal course	CAS involvement
May 13, 2011	Addiction treatment centre) worker notifies CAS starting in April 2011 that the mother started testing positive for oxycontin and cocaine. She had already admitted to using marijuana.	
May 17, 2011		CAS initial contact.
May 18, 2011	The addiction centre sent a response to a letter from the obstetrician. In this letter the obstetrician was notified that the mother had urine drug screens positive for cocaine and marijuana and that the addiction centre had notified CAS.	Family Risk assessment. Both mother and biological father have previous CAS involvement with previous children and personally.
May 19, 2011		Mother advised of supports and Healthy Baby Healthy Children and Nog (Native support) and she expressed openness to these. She agrees to continue to attend prenatal visits and the methadone clinic and gave CAS consent to speak to "collateral contacts." There were numerous attempts by CAS to contact the mother in person and by telephone. She was often not available for contact.
June 7, 2011	No show for prenatal visit. There is no further prenatal care until she presents in active labour on August 1, 2011.	

Discussion

In general, Children's Aid Societies encourage expectant mothers with a history of substance abuse who are the subject of ongoing child protection investigations, to seek ongoing prenatal medical care and other community services. Participation however, is voluntary and consensual. If the expectant mother has no other children in her care, then the CAS can open a file and encourage her to seek prenatal medical care and community supports. However, under Canadian law, the fetus has no rights until it is born. Thus, there is no recourse if the mother refuses care and there is no obligation for the obstetrician to report.

If the expectant mother has other children, then the focus of the CAS is the safety of those children. CAS cannot enforce

abstinence as a condition of custody; they focus on harm reduction and education as ways to mitigate risk to the children. For example, in this case, the CAS and the client (i.e. the mother) agreed that when she was "using", she would take her child to the grandparent's house. If abstinence were a condition of custody, the pregnant woman may be less likely to seek help if she fears the loss of her child(ren).

While health care providers in Canada have a legal responsibility to report any child in need of protective services, this obligation only applies once the child is born. Prior to the birth, health care workers should discuss the issue and obtain written consent from the pregnant woman if considering a CAS referral in the prenatal period.

Many women who use substances fear that their health care provider(s) will report them to the authorities. In response to this fear, many women may not seek medical assistance when needed. This situation is challenging to both health and social service providers.

The Centre for Addiction and Mental Health (CAMH) website is a valuable source of information and contains a summary of the Exposure to Psychotropic Medications and Other Substances during Pregnancy and Lactation: A Handbook for Health Care Providers.¹ This information can be found online at:

http://www.camh.net/Publications/Resources_for_Professionals/Pregnancy_Lactation/index.html

The Handbook suggests that, “health care practitioners who provide care to pregnant women with substance use and/or mental health problems can play a key therapeutic role in helping women move toward a better health outcome” and that “listening to women’s concerns and fears, and offering guidance and reassurance, can help providers be effective health care allies.” It is believed that “stigma and marginalization prevent many women with substance use and mental health problems from presenting to health care providers.” Women who do enter prenatal care are likely to “feel guarded and ashamed of their substance use and/or mental illness.” Understanding the stigma of mental illness and substance use and responding in a non-judgmental way will encourage women to be more involved in their own treatment.²

Effective communication is essential to establishing a positive relationship between the health care provider and the pregnant patient. The nature and context of the communication can affect how a woman responds to the advice of her health care provider. It has been found that, “a provider’s choice of words, demeanour and attention can make it easier for a woman to listen and communicate, and to act on new knowledge.”³

Research has found that two significant factors that lead to successful change in a person’s life include how the client (i.e. pregnant woman) interprets her experience of the therapeutic relationship (e.g. a health care provider’s non-judgmental attitude, warmth, respect and caring) and the techniques and skills utilized by the health care provider.

Additional information can be obtained in the SOGC guideline entitled, “Substance Use in Pregnancy” which can be accessed at: <http://www.sogc.org/guidelines/documents/gui256CPG1104E.pdf>

Recommendations

No recommendations.

References

1. http://www.camh.net/Publications/Resources_for_Professionals/Pregnancy_Lactation/index.html
2. http://www.camh.net/Publications/Resources_for_Professionals/Pregnancy_Lactation/index.html
3. Ibid

Case: 2012-S-4
OCC File: 2009-3416

History

The mother of this stillborn child was a 38-year-old woman who had two normal pregnancies in 1990 and 1992. A tubal ligation was performed in 1992 and reversed in 2005. She had a third normal term pregnancy in 2007.

The medical history was not completed on the Antenatal record. From the emergency department records, the mother was a smoker and suffered from asthma.

The EDD was March 6, 2009 based on the last menstrual period. The ultrasound on August 19, 2008 showed a crown-rump length of 12 weeks 4 days. The IPS was normal. The morphology ultrasound at 24 weeks was normal; however there was an anterior placenta previa. Both the first and second trimester ultrasound suggested a due date one week earlier. The ultrasound results were not documented on the antenatal forms.

The fetus was thought to be in a breech position at 34 weeks. An ultrasound showed normal fetal growth, a vertex position and a fundal placenta. The pregnancy was complicated by shortness of breath and she was seen three times in the emergency department. The woman was admitted for worsening asthma at 41 weeks.

On March 16, 2009, she was admitted at 41.5 weeks gestation for induction of labour. Prostaglandin gel applied at 1400 hours was used to establish labour.

At 1645 hours, she had an artificial rupture of membranes with clear amniotic fluid seen.

At 1730 hours, she received morphine and dimenhydrinate. The FHR was monitored continuously and was normal.

At 1840 hours, there was a sudden deceleration followed by a large amount of vaginal bleeding and at 1843 hours, the

physician was called. It was difficult to document the FHR thereafter, but there appeared to be persistent bradycardia.

At 1850 hours, the physician arrived and a fetal scalp clip applied. Notes indicated that "no real FHR [FHR] was heard."

The 3822 g male infant was delivered by emergency Caesarean section at 1912 hours. A complete abruption was evident. The infant did not have a measurable heart rate and did not respond to resuscitation. Apgar scores were 0, 0 and 0 at one, five and 10 minutes. A cord hemoglobin was normal and maternal Kleihauer Betke test was negative. Cord gases do not appear to have been done.

Post mortem

An autopsy was not conducted and no placental pathology was available for review.

Discussion

The mother was delivered by emergency Caesarean section at 41 weeks and three days gestation following a complete placental abruption. The induction process to that point was uneventful and the FHR normal. The only identifiable risk factor was smoking. The first and second trimester ultrasounds suggested a due date one week earlier so the gestational age may have been 42.5 weeks at the time of induction.

No post dates surveillance was completed. This may or may not have any bearing on the abruption that occurred and final outcome.

Recommendations

1. Obstetrical care providers reminded of the importance of confirming the menstrual dates with the gestational age on ultrasound assessment.
2. Obstetrical care providers reminded of the importance of post dates fetal surveillance.

Case: 2012-S-5

OCC File: 2011-11967

Antenatal History

The mother of this stillborn child was a 28-year-old G2P1 with an EDD of September 21, 2011. She had one previous term pregnancy delivered vaginally at 42 weeks of an 8 pound 13 ounce female in 2008.

The woman received her obstetrical care from her family doctor. Ultrasounds at 15 and 20 weeks were normal. Routine prenatal laboratory investigations were normal. IPS was negative and glucose challenge test was "within normal limits."

Systems review and family history were non-contributory.

Course in Labour and Delivery

At 0915 hours on September 23, 2011, at 40 weeks and two days gestation, the woman called the hospital to report decreased fetal movement. The Obstetrical Telephone Advice Record indicates the reason for the call was decreased fetal movement, but does not indicate what instructions were given. It is not clear from the record at what time the woman arrived at the hospital.

There is an electronic fetal monitoring strip on the record that indicates a start time of 1026 hours and continuing until 1224 hours. The woman was seen by her family doctor at 1245 hours. The cervix was closed and posterior and the vertex was unengaged. The obstetrician on call was consulted for a non-reassuring NST. The woman was seen by the obstetrician who reviewed the FHR tracing. A contraction stress test using nipple stimulation was ordered. The EFM was restarted at 1247 hours. Contractions resulted from the nipple stimulation, but the FHR tracing was still not reactive. The obstetrician recommended that the family doctor proceed to induction and to keep the patient nil per os [NPO], or without food/water. The attending family doctor signed out and another family doctor was assigned.

Oxytocin was started as per the obstetrician's order at 1402 hours and FHR decelerations were noted at 1415 hours. The

oxytocin was stopped at 1445 hours and the incoming family doctor was called. The patient was re-positioned, given O2 by mask and the IV opened. The FHR was recorded as 120 bpm, then accelerated to 150 bpm. The oxytocin was restarted at 1515 hours and the FHR baseline was 128 bpm, with accelerations to 150 bpm, but with continued decreased variability. The oxytocin was discontinued at 1530 hours and the family doctor reviewed the FHR tracing at 1538 hours. On examination at 1553 hours, the cervix was 2 cm and thick. During the examination, the FHR dropped to 80 bpm.

The obstetrician was called at 1610 hours and was informed that there was fetal bradycardia and late decelerations. The patient was prepared for the operating room. The obstetrician attended at 1620 hours and meconium was noted on examination at 1630 hours. The woman was transferred to the operating room at 1640 hours.

Under general anaesthesia, she was delivered of a stillborn male infant weighing 4198 g at 1703 hours. Resuscitation attempts were terminated after 10 minutes.

Cord arterial (labelled venous) pH was 6.80. Cord venous (labelled arterial) pH was 7.00, and BE -18.

Post mortem

Post mortem examination revealed growth parameters that were greater than for gestational age.

Examination of the brain revealed florid pontosubicular necrosis, acute petechial hemorrhages and subacute microinfarcts of the brainstem and cerebellum.

Placental pathology showed overcoiling of the umbilical cord, moderate meconium staining of the fetal membranes, fetal thrombotic vasculopathy, chorangiosis and chronic villitis.

The cause of death was stated as hypoxic ischemic brain injury, acute and subacute due to severe fetal thrombotic vasculopathy of the placenta. Chorangiosis of the placenta was identified as a contributing factor.

Discussion

This stillborn died as a result of perinatal asphyxia. The post mortem examination report determined that this was due to severe fetal thrombotic vasculopathy of the placenta.

The mother of the stillborn presented to hospital at 40 weeks and two days gestation with decreased fetal movement. Electronic fetal monitoring was initiated and was interpreted as "non-reassuring." Review of the tracing shows the presence of decelerations which subsequently became repetitive late decelerations with nipple stimulation and deteriorated further with labour induction. The changes seen on the admitting NST in the clinical setting of perceived decreased fetal movement warranted immediate delivery.

Although some degree of fetal compromise existed at the time of presentation to the obstetrical unit at the hospital, it cannot be determined by this review whether a Caesarean section shortly after admission would have changed the outcome.

Recommendations

1. Obstetrical care providers are reminded of the SOGC Guidelines "Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline."¹
2. Obstetrical care providers are reminded that oxytocin is contraindicated in the absence of a normal FHR tracing.
3. The committee recommends that the Regional Supervising Coroner (RSC) follow up with the hospital regarding the issues identified. If, in the opinion of the RSC, systemic issues persist, the RSC should consider conducting a Regional Coroner's Review with appropriate representatives from the hospital.

References

1. Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline. Journal of Obstetrics and Gynaecology Canada (JOGC). Vol. 29, No. 9, September 2007.

Case: 2012-S-6

OCC File: 2011-7884

History

The mother of this stillborn child was a 23-year-old gravida 1 with an EDD of August 6, 2011. She was on synthroid for hypothyroidism. Her health history was otherwise unremarkable. She was referred to a consultant obstetrician at approximately 13 weeks gestation and was followed regularly. The prenatal records show a normal course. Prenatal testing was normal and she was Rh positive. IPS was not done. A second trimester ultrasound scan at 20 weeks showed normal anatomy and anterior placenta clear of the cervical os.

She was last seen at 34 weeks gestation and there was a normal assessment.

On June 29, 2011, the mother was the passenger in a vehicle that was involved in a collision where the airbags were deployed. EMS transported the woman directly to the labour and delivery unit at the hospital for assessment.

A note later in the day reported that there was "a large reddened area noted from the right shoulder to about the heart level from the seat belt, with a small amount of skin scraped off." The obstetrician noted that the mother had stable vital signs, but had been complaining of generalized pain and aches, had a tender abdomen, and had a small area of blood on her pants. Pelvic examination revealed a closed cervix with a small amount of dark red blood on the examining glove. It was noted that the fetal heart was present and that the tracing was normal for the gestational age.

The fetal heart monitor tracing was started at 1151 and continued until 1427 hours. There was no indication of monitoring of fetal movement or contractions. The tracing had multiple areas where there was no pick-up of the fetal heart. For the first hour, the tracing showed variability, though subsequent monitoring showed a flat pattern and many areas where it was not picked up. There were no accelerations of fetal heart seen on the monitor.

Initial blood testing revealed a white cell count of 15.5, hemoglobin of 124 and normal platelets. INR was 1.12 and other coagulation studies were normal. Liver function tests showed an AST of 88 (normal less than 31); alkaline phosphatase, alanine aminotransferase (ALT) and lactate dehydrogenase (LDH) were normal. She was confirmed to be B positive with no antibodies. Kleihauer Betke screen collected at 1400 hours was positive, indicating an equivalent of 25 ml of fetal blood.

An obstetrical ultrasound and BPP performed at 1224 hours revealed a live fetus approximately 35 weeks gestation with normal growth parameters. The placenta was anterior and the cervix closed and long. The fetal heart was 133. BPP score was 6/8 with 0 for tone. No abruption was seen.

At 1515 hours, the woman was routed to the emergency department in order to rule out any other injuries as it was felt that obstetrically, she was stable. There was no indication of communication between the obstetrical and emergency staff. It is not known if any obstetrical staff accompanied the woman to the emergency department. While in the emergency department, she was triaged as CTAS 3 (i.e. urgent – to be seen by physician within 30 minutes). Vital signs recorded at 1520 hours in the emergency department were normal. There was no indication on the records of a FHR.

The notes made in the emergency department indicated that the woman "states no fetal movement" and that she was sent down there for a C-spine and knee X-ray series. She was in a neck collar and board since the motor vehicle collision earlier in the day.

The woman was in the emergency department for approximately three hours. When she was returned to the labour and delivery unit, there was difficulty in finding the fetal heart. At 1744 hours, an ultrasound indicated a fetal heart of 34. There was no remark made of the placenta. It was explained to the woman that the fetus was in a dire condition and that a Caesarean section was suggested.

The Caesarean section was started at 1800 hours under general anaesthetic. At 1816 hours, a stillborn female infant

weighing 2490 g was delivered. It was noted that “a moderate large amount of clots were obtained which indicated a severe placental abruption.” Apgars were 0 and there was no fetal heart or signs of life. The pediatric staff in attendance was unable to resuscitate the infant. Cord gases were not done, nor were a post mortem or placental examination.

The woman’s further course in hospital was unremarkable.

Discussion

The mother of the stillborn was involved in a motor vehicle collision where she suffered significant bruising over her chest as a result of deployment of air bags. With an anterior placenta, that would increase the risk of injury. Initial assessment in the obstetrical unit at the hospital where she was taken revealed a tender abdomen and evidence of blood on the glove of the examining hand. The continuous monitoring was not a normal pattern. The monitor did not suggest an active fetus. BPP profile early in the assessment indicated 0 for tone. There was no ultrasound evidence of an abruption at that time however it is clear that this subsequently evolved. The ultrasound had been done shortly after admission, about approximately three hours prior to going to the emergency department; an abruption was likely evolving over that time period.

The patient should have been reassessed with regard to the monitoring tracings, BPP findings, positive Kleihauer Betke test and physical exam (and in particular, her abdomen), prior to being sent to the emergency department. This reassessment may have helped to determine that the fetus was in a compromised state. If she was determined to be stable, in a case with a high probability of abruption occurring, an obstetrical nurse should have gone and supervised continuous monitoring.

Even if further continuous monitoring had continued in the emergency department, the final outcome may not have changed, as an abnormal tracing had not raised any alarm previously.

Recommendations

1. The hospital involved should review the management of trauma to pregnant patients and the communication and collaboration between the obstetrical and the emergency departments.
2. Obstetrical care providers are reminded of the potential subtle nature of the evolution of an abruption with abdominal trauma.

Case: 2012-S-7

OCC File: 2011-15690

Case Summary

The mother of this stillborn child was a 29-year-old G1P0 healthy woman with a history of asthma controlled with salbutamol. She had a BMI of 25 and gained 37 pounds in the pregnancy. Prenatal laboratory investigations were normal. IPS was not completed. At 12 weeks gestational age, an ultrasound showed a small subchorionic hemorrhage. Routine ultrasounds were negative. An OGCT was negative and a GBS swab was negative.

On November 20, 2011 at 0230 hours, the mother presented in early labour at 40 weeks and six days gestational age. Contractions were every 8-10 minutes, there was "some bloody show," her cervix was 1-2 cm dilated and the FHR was auscultated in the 130's bpm.

At 1130 hours, the contractions were every five to six minutes, cervix was 2-3 cm and FHR still in the 130's.

At 1630 hours, the contractions increased and the woman presented to obstetrical triage for analgesia. On admission, an external fetal monitor tracing at 1730 hours had a baseline of 140 bpm, moderate variability, no accelerations and there were occasional, uncomplicated, variable decelerations of up to 25 bpm lasting less than 20 seconds. There was no documentation of FHR response to fetal scalp stimulation. Maternal heart rate was not documented. Intermittent auscultation in labour was initiated.

At 1845 hours, there was an artificial rupture of membranes for clear fluid. The FHR was documented at 130 to 150 bpm and maternal pulse of 107. Accelerations were documented throughout labour on the partogram.

At 2115 hours, the FHR was "down to 118" and it "recovered to 140 with maternal change of position" to left lateral. Intermittent auscultation was continued. There was no documentation of maternal heart rate until 2215 hours when it was recorded as 84 bpm and FHR 145 bpm.

At 0015 hours on November 21, 2011, the woman was fully dilated, vertex at spines and occiput posterior. She began pushing.

At 0025 hours, the FHR was documented at 130s to 150s.

At 0158 hours, the FHR was auscultated at 70 bpm. The woman was repositioned and the FHR was 110 bpm.

At 0200 hours, the FHR was 90 bpm and the external fetal monitor was applied. The tracing was reported to have a FHR of 88-150 bpm and "patchy."

At 0205 hours, the obstetrician on call was consulted for a FHR of 90 bpm. A fetal scalp clip was applied. There was "significant artifact" and thus the external monitor was reapplied.

At 0220 hours, a second scalp clip was applied. The tracing was determined to be "patchy" and a third scalp clip was applied. The heart rate detected was then determined to be maternal. A real time scan was performed and no fetal cardiac activity was seen. Since "the midwives had been quite convinced that the FHR had been 130-140 consistently until it dropped to the 70s to 90s," the obstetrician decided to proceed to a stat Caesarean section.

The patient was taken to the operating room at 0237 hours and a general anesthetic was administered. The procedure started at 0240 hours. The electrosurgical unit did not function and it was not until a third machine was subsequently brought in that a functioning unit was found.

The infant was delivered by Caesarean section at 0252 hours. Forceps were applied as the "vertex was wedged and there was a soft tissue dystocia." The infant was a male weighing 2840 g with Apgars of 0, 0 and 0.

Resuscitative efforts included PPV, intubation, chest compressions and two doses of epinephrine.

The pediatrician note indicated that the obstetrician mentioned a "tight" cord around the neck at the time of delivery.

Post mortem

The placenta showed a stage 1 maternal inflammatory response (acute chorionitis) and stage 2 fetal inflammatory response (acute phlebitis and acute arteritis of one artery with one of the umbilical arteries showing segmental nonocclusive fibrin thrombi attached to the intima).

The cause of death was determined to be acute bronchitis/bronchiolitis with early pneumonia due to amniotic infection syndrome.

Discussion

The mother presented in early labour on November 20, 2011. She was appropriately seen and assessed not to be in active labour at that time. She was not admitted to labour and delivery until labour was active; this is consistent with the standard of care. Avoiding admission to hospital until active labour has been shown to decrease intervention rates in labour.

The family was concerned because the demise occurred so close to delivery and thus they were questioning the care received from the midwives.

Intermittent auscultation "is an appropriate technique for fetal surveillance when a woman experiences a healthy pregnancy and birth."¹ In this review, there were no risk factors in pregnancy prior to admission in labour that would contraindicate intermittent auscultation. Recommendation 10 of the SOGC Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guidelines states, "Admission fetal heart tracings are not recommended for healthy women at term in labour in the absence of risk factors for adverse perinatal outcome, as there is no evident benefit."²

In this case, throughout labour, intermittent auscultation was appropriately performed and accelerations were documented.

When the FHR was noted to be 90 bpm at 0205 hours, the obstetrician was called. The obstetrician attended by 0210 hours. The patient was fully dilated, at spines and occiput transverse. There was no documentation of the maternal heart

rate. There were difficulties in making the decision to proceed to Caesarean section and delays in starting the Caesarean section.

There was no documentation of the rationale not to try a vaginal delivery assisted by vacuum or forceps. Non-reassuring fetal status is an indication for operative vaginal birth³ and could have been attempted in the labour room to expedite delivery. The time of delivery of 0252 hrs was 47 minutes after the FHR was noted to be 90 bpm.

The post mortem examination determined the cause of death to be amniotic infection syndrome. Amniotic infection syndrome is defined as inflammatory lesions of the placenta, cord, membranes and fetal organs (especially the lungs). It is a very difficult diagnosis to make as these tissues can be contaminated both before and after birth. Amniotic infection syndrome is the cause of death in 5% of stillbirths and an accessory lethal factor in another 5%.⁴

Recommendations

1. Obstetrical health care providers are reminded to document maternal heart rate to ensure differentiation from FHR.
2. Obstetrical health care providers are reminded that operative vaginal birth should be considered as an alternative to Caesarean Section in the setting of non-reassuring fetal status assuming the prerequisites are present and there are no contraindications, as per the SOGC Clinical Practice Guidelines for Operative Vaginal Birth (No. 148, August 2004).

References

1. JOGC, Vol 29, Number 9, September 2007, p. 31
2. JOGC, Vol 29, Number 9, September 2007, p. 33
3. SOGC, Vol 148, August 2004, p. 748
4. Pediatrics, Vol. 25, No. 3, 1960, p.436

Case: 2012-S-8

OCC File: 2011-15360

Antenatal History

The mother of this stillborn child was a 32-year-old G8P3 with an EDD of December 13, 2011 by first trimester ultrasound. Routine prenatal laboratory investigations, IPS and second trimester ultrasound were normal. An OGCT was abnormal and the oral glucose tolerance test was diagnostic of gestational diabetes. The mother was referred for diabetic education, diet and blood sugar monitoring. Her care was transferred by her family doctor to an obstetrician at 34 weeks. Prior to her transfer, the family doctor documented her blood sugar levels at her prenatal visits on the Antenatal Record 2.

The woman had three previous vaginal births. The first was delivered in 1998 of a 2100 g male at 32 weeks gestation. The pregnancy was complicated by threatened preterm labour at 26 weeks gestation. Her second pregnancy was delivered in 2002 at 35 weeks gestation of a 2950 g female. That pregnancy was complicated by gestational diabetes. The third pregnancy was delivered of a 3350 g male infant at 37 weeks gestation. The pregnancy was complicated by gestational diabetes requiring insulin. She also had three spontaneous abortions and a pregnancy termination. Because of the past obstetrical history of preterm labour, she was treated with progesterone.

The mother smoked approximately six cigarettes a day and at the beginning of this pregnancy, her weight was 266 pounds (height of 4 feet, 10 inches), for a BMI of 55.7.

She was first seen by the obstetrician on November 4, 2011 at 34 weeks gestation. Weekly NST and BPP were arranged. At her subsequent weekly prenatal visits, her blood sugars were not documented other than a note on November 30, 2011 at 37.5 weeks indicating the blood sugars were ``sl hi`` and on December 9, 2011, at 39.5 weeks indicating ``sugars starting to be erratic``. At her last prenatal visit on December 9, 2011 she was booked for induction on December 12, 2011.

Course in Labour and Delivery

The woman presented to hospital on the morning of December 12, 2011 for labour induction. She was placed on an external monitor. When seen by the obstetrician, she was told that he had reconsidered the situation and because her blood sugars were high, he was going to arrange for induction in a nearby, regional hospital.

An appointment was made for her to be seen at the regional hospital on December 13, 2011 at 1800 hours. Later in the day on December 12, 2011 however, she presented to the obstetrical unit at the regional hospital because she had not felt the baby move for a couple of hours. The fetal heart could not be detected and fetal demise was confirmed on ultrasound by the obstetrician on call.

Labour was induced and an artificial rupture of membranes was performed for a small amount of fluid. There was some question as to whether there had been a possible rupture of membranes earlier in the day. Labour became well-established and the woman delivered a stillborn male infant weighing 2936 g at 0303 hours on December 13, 2011.

Post mortem

The autopsy did not reveal any abnormalities and the cause of the stillbirth could not be determined.

The placenta weighed 589 g. There were three areas of recent infarct, the largest measuring 3.5 X 2.5 X 1.0 cm. The amniochorion was unremarkable other than minute areas of lymphocytic infiltrate.

Discussion

This case involved an infant that was stillborn at term following a pregnancy complicated by gestational diabetes. The gestational diabetes was treated by diet alone and blood sugar monitoring. The mother's blood sugar control after 34 weeks gestation cannot be determined by this review. A note by the obstetrician

provided after the delivery indicated that the patient did not bring her blood sugar diary to her prenatal visits.

Fetal surveillance was heightened during the last five weeks of the pregnancy by weekly NST and BPP. The BPP of November 24, 2011 was normal. The BPP of December 2, 2011 scored 6/8 with 0 for amniotic fluid. Note was made of the umbilical cord Doppler ratio being elevated at 3.0 and compared with an ultrasound of November 7, 2011, there was a suggestion of an overall lag in growth. The antenatal record does not indicate any consideration for intervention as a result of this report.

The BPP of December 8, 2011 again reported a score of 6/8 with 0 for amniotic fluid. No other parameters were reported on. Again, the antenatal record does not indicate any consideration for intervention.

On December 12, 2011, the woman presented for a scheduled induction of labour. Her blood sugar was 13.4 at that time. A FHR tracing was started at 0825 hours and she was seen by the obstetrician at 0845 hours; a decision was made to cancel the induction and make arrangements for induction at the regional hospital because of the high blood sugar. The FHR tracing was completed at 0848 hours and the woman was discharged at 0855 hours.

A late entry note by the obstetrical nurse on December 13, 2011 for December 12, 2011 indicates that the obstetrician was "aware of tracing, NST normal with 2 accelerations in 20 min increasing by 10-15BPM lasting 10-15 sec, good fetal mvt noted." Upon review by the MPDRC, this tracing is in fact, abnormal.

In summary, this was a high risk pregnancy due to gestational diabetes. This resulted in heightened surveillance of the pregnancy. The blood sugar reporting and monitoring was poor. This, combined with the need for insulin in the previous pregnancy, should have prompted a referral to an endocrinologist. Although the baby was not large for dates, high blood sugars may have played a role in the stillbirth.

Abnormalities in the fetal surveillance testing began to appear

on the BPP of December 2, 2011, at 38 weeks gestation. In the setting of a high risk pregnancy at term with poor blood sugar control, plans should have been made for delivery at that time. Having gone on to the booked induction date of December 12, 2011, the abnormal FHR tracing on admission that day should have prompted proceeding with immediate delivery.

It cannot be determined by this review what intervention and subsequent outcome may have resulted if the obstetrician had been informed about the FHR tracing.

Recommendations

1. Obstetrical care providers are reminded of the importance of appropriate monitoring of blood sugar levels in gestational diabetes to determine if insulin is required.
2. Obstetrical care providers are reminded to consider referral to an endocrinologist if there is any question about adequate blood sugar control.
3. Obstetrical care providers are reminded of the increased risk of fetal morbidity and mortality associated with oligohydramnios as documented on ultrasound.
4. Obstetrical care providers are reminded of the criteria for interpreting non stress test (NST) results.
5. Primary Health Care Providers should consider early referral to an obstetrical care provider when a pregnancy is considered high risk.

Case: 2012-S-9

OCC File: 2012-2467

Clinical History

The mother of this stillborn child was a 34-year-old G2A1 with an EDD of March 18, 2012. This pregnancy was conceived with in vitro fertilization and intracytoplasmic sperm injection. The father was a 56-year-old with a history of hepatitis C.

The mother was seen in prenatal care from approximately 13 weeks gestation. She had all routine prenatal blood tests and all were normal. Although she had hemoglobin E trait, she had a normal hemoglobin level. Arrangements were made for IPS, however a final IPS result was not available for review. A 12-week ultrasound revealed a normal nuchal translucency. A second trimester ultrasound scan and a one-hour OGCT were both reported as normal.

The mother's past health history included a first trimester spontaneous abortion; she was otherwise healthy. In the first trimester of this pregnancy, she had nausea and vomiting requiring Diclectin®; the symptoms settled down by the second trimester.

She was followed by an obstetrician for the duration of her pregnancy and the prenatal records showed an unremarkable course with the last recorded visit at 35 weeks and four days with GBS screening at that time.

She presented to the triage area of the Family Birthing Centre at Hospital A at 2100 hours on February 27, 2012 with a history of decreased fetal movements since 1200 hours that day. A NST was performed for 1.5 hours. It is not clear whether the patient had been asked to indicate times of movement as there were no movement indications on the NST record. There was normal baseline variability however there was no obvious FHR accelerations normally associated with movement.

Bedside ultrasound was done and clinical notes indicated "ample movement demonstrated, normal AF, placenta anterior." The patient was sent home after these tests with a plan for a BPP

the next morning (gestational age of 37 weeks and two days). The following day, on February 28, 2012, the BPP revealed that the amniotic fluid volume (AFV) was "subjectively increased" however a four quadrant AFV was high, but within normal limits. The measurements were within normal with an EFW of 3239 grams (59th percentile). BPP score was 6/8, 0 for fetal breathing and cord dopplers were normal. A NST was done after the BPP and with continued monitoring, it was noted that the "NST improved, few accels, moderate variability, increased fetal movement three times in past 10 minutes." Upon review of the NST, there were no fetal movements with accompanying fetal heart accelerations seen on the strip.

The patient was discharged home and the plan was for her to come back the next day, February 29, for another NST.

A NST was conducted from 1437 until 1619 hours on February 29, 2012. During that time, the trace had poor variability, no accelerations and no indication of fetal movement was recorded. After the testing, a decision was made for admission to labour and delivery and a plan for artificial rupture of membrane and oxytocin induction of labour. The patient was initially reluctant to have artificial rupture of membranes.

She was admitted at approximately at 1715 hours and an artificial rupture of membranes was performed at 1800 hours. Oxytocin was started. There was copious amniotic fluid and it was meconium stained.

The tracing continued to be flat. A first deceleration occurred just before the artificial rupture of membranes was performed at 1800 hours; a scalp clip was placed at that time. She had definite deep and prolonged decelerations around 1900 hours which appeared to recover, however she continued to have recurrent decelerations and the oxytocin was shut off at 2005 hours. The patient consented to a Caesarean section at 2020 hours and the monitor was taken off. She was taken to the operating room at approximately 2035 hours.

There was no further monitoring available until the time of delivery. The patient was in the operating room at 2045 hours and at 2050 hours, the anesthesiologists were "told to proceed

with spinal as obstetrician doing another delivery.”

Surgery was started at 2103 hours and at 2108 hours, the male infant was born by low segment Caesarean section. Neonatal staff attended to the infant. The balance of the procedure was unremarkable.

The infant had Apgars of 0, at one, five and 10 minutes.

Initial suction below the cord revealed meconium. Ventilation was initiated with bag and mask as there was no respiratory effort. As no heart rate was detected, chest compressions were started. Intubation occurred at 2112 hours with initial epinephrine via ETT by 2116 hours.

Despite continued ventilation and chest compression, no vital signs were obtained, and resuscitative efforts were stopped at 2121 hours.

Cord gas that had been taken revealed a PH of 6.95 with a base deficit of 12.

Post mortem

Post mortem findings revealed a 37 week gestation stillborn male that was anatomically normal. There was evidence of acute mode of intrauterine fetal death. The placenta was within normal weight for gestation. There was evidence of marked umbilical cord overcoiling and areas of marked cord narrowing; the cord diameter varied from .6 to 2.1 cm. Normal male cytogenetic microrray result was obtained. Testing for routine newborn screening was done and was negative.

The cause of death was noted to be acute perinatal asphyxia due to acute umbilical cord compression (cord wrapped twice around the leg) in a setting of an overcoiled cord with areas of cord narrowing.

Discussion

The family of the stillborn had expressed their concerns regarding the care provided at Hospital A and the subsequent

investigations conducted by the Office of the Chief Coroner.

In particular, the family was concerned with comments made by obstetrical care providers to the mother when she indicated on February 27, 2012 that she had not felt fetal movement for a duration of time. The family indicated that although staff had acknowledged and commented on the the lack of movement, no action was taken to the non-reactive NST strips.

The following day, on February 28, the mother returned to hospital for a BPP. The BPP was reported as “normal” although the score acknowledged a two point decrease for fetal breathing. While this result is common and often without significance, it was noted that during the NST, the mother could not feel movement and though there was appropriate baseline variability, there were no accelerations that one would expect to see with an active fetus. The staff was reassured and advised the patient to come back after mid day on February 29, 2012.

The monitoring strip from 1430 hours on February 29, 2012 was abnormal and remained so until around the time of the artificial rupture of membranes at 1800 hours when the first significant deceleration was noted. At 1900 hours, there were clearly deep and prolonged decelerations. There was a continued delay in decision making of an urgent need for delivery. When that decision was made and the patient was transferred to the operating room, there was no evidence of any further monitoring; delivery occurred approximately 30 minutes later. There is the possibility that the outcome may have been different with an earlier delivery. Even though it may not have made a difference in the outcome, fetal monitoring should have resumed as soon as the patient was transferred to the operating room until it was time to prepare for the surgery.

This case demonstrates the need for obstetrical care providers to acknowledge and respond accordingly when patients indicate a significant decrease in fetal movement. The decrease in movement, combined with two non-reactive NSTs (although not distinctly “abnormal”) and a period of abnormal FHR, were indications for a more urgent response.

Recommendations

1. Obstetrical care providers are reminded of the following recommendations pertaining to fetal health surveillance from previous reviews conducted by the Maternal and Perinatal Death Review Committee (MPDRC):

Year	Type	Recommendation
2006	Stillbirth	Fetal well-being should be assessed without delay when patients present with decreased fetal movements.
2006	Stillbirth	Obstetrical care providers are reminded that a non-reassuring FHR tracing should prompt another test of fetal well-being, such as fetal scalp sampling, or expedited delivery.
2009	Neonatal	Obstetrical care providers are reminded of the importance of identifying and acting on abnormalities in fetal heart monitoring.
2010	Neonatal	Obstetrical care providers are reminded of the criteria for an abnormal FHR tracing and the action required as defined by SOGC Intrapartum Fetal Surveillance Guidelines published in September 2007
2011	Stillbirth	It is recommended that electronic fetal monitoring be continued while preparations are made for emergent Caesarean section.

Chapter Four: Lessons Learned from MPDRC Reviews

Labour and delivery is a unique area of medicine. Obstetrical care providers are actually providing care to two patients simultaneously – the mother and the fetus. When there is a change in a measured parameter, such as heart rate, it is important to determine with which patient the change is associated – the mother or the fetus. Identifying and documenting changes based on which patient is being assessed is sometimes challenging, but is essential to providing effective and thorough obstetrical care to both the mother and the fetus.

To address this challenge, the MPDRC recommends that consideration be given to establishing a standard location on EFM strips or partogram documents to clearly and visibly differentiate between maternal and fetal vital signs. By doing so, a change in either value can be easily identified and assessed.

Appendix A: Summary of Recommendations - with Identified Themes 2012 MPDRC Cases

Maternal Case Reviews

Case #	Rec #	Theme	Recommendations
1			No recommendations
2			No recommendations
3			No recommendations

Neonatal Case Reviews

Case #	Rec #	Theme	Recommendations
1	1	Quality	Those overseeing the operations of the NICU at the tertiary neonatal centre should perform a thorough review of this case and the parents' complaints.
1	2	Communication/ documentation	Health care providers are reminded that the coroner's office should be contacted when family members appear to have concerns about the care provided.
1	3	Medical	Neonatal health care providers are reminded of the possible adverse effects of red blood cell transfusion including Transfusion-Related Acute Gut Injury (TRAGI) and the need to limit transfusions to situations in which there are clear indications. Consideration should be given to placing a transfused patient "nil per os" (NPO) [nothing by mouth] for several hours before and after the transfusion.
1	4	Quality	The Committee recommends that the Regional Supervising Coroner (RSC) follow up with the hospital regarding the issues identified. If, in the opinion of the RSC, systemic issues persist, the RSC should consider conducting a Regional Coroner's Review with appropriate representatives from the hospital.

Maternal and Perinatal Death Review Committee 2012 Annual Report

Case #	Rec #	Theme	Recommendations
2	1	Diagnosis and testing - EFM	Obstetrical care providers are reminded that: a) Application of a fetal scalp clip should be considered when it is difficult to monitor fetal heart in labour; It is important to ensure that the heart rate being monitored is fetal and not maternal. This is especially important when there are abrupt changes in the fetal heart rate tracing. (Reference: Simpson, Kathleen, "Avoiding Confusion of Maternal Heart Rate with Fetal Heart Rate during Labour". American Journal of Maternal Child Nursing: July/August 2011 - Volume 36 - Issue 4 - p 272) b) Efforts should be made to proceed to "emergency" Caesarean section in a timely manner; c) When the fetal head is known to be well down in the pelvis and difficult delivery is anticipated, consideration should be given to manoeuvres that will facilitate delivery in order to decrease the risk of trauma to the fetal head at delivery; d) If intubation is deemed to be necessary but cannot be successfully performed, assistance should be sought from another skilled operator in the use of laryngeal mask airway (LMA); e) When active resuscitation is required and severe acidosis and encephalopathy are present, immediate consultation with a pediatrician at the referral centre or tertiary centre should be undertaken for consideration of total body cooling and transfer.
		Policy and procedure	
		Medical	
		Medical	
		Medical	
2	2	Quality	A Regional Supervising Coroners Review should be conducted on the circumstances surrounding the death of this infant.
3	1	Medical	Obstetrical and neonatal care providers are reminded of the risk of subgaleal hemorrhage (SGH) with vacuum extraction, forceps or difficult delivery.
3	2	Communication/ documentation	Obstetrical care providers are reminded of the need to thoroughly document operative interventions.
3	3	Diagnosis and testing	Obstetrical care providers are reminded to obtain cord blood gases at delivery.
3	4	Communication/ documentation	Neonatal care providers are reminded to document a complete and full newborn assessment on initial examination in a timely fashion.
3	5	Policy and procedure	The hospital should review/establish a protocol for monitoring newborns delivered by vacuum extraction, forceps or difficult delivery.

Maternal and Perinatal Death Review Committee 2012 Annual Report

Case #	Rec #	Theme	Recommendations
3	6	Policy and procedure	The hospital should review/establish its policy regarding indications for the attendance of a paediatrician at delivery.
3	7	Policy and procedure Communication/ documentation	The hospital should review its policy for communicating critical laboratory results.
3	8	Quality	The hospital should perform a quality of care review of this case.
4	1	Policy and procedure	Prenatal ultrasound providers are reminded of the SOGC and American Institute of Ultrasound Medicine (AIUM) Guidelines requiring full second trimester morphology assessments and reporting.
4	2	Diagnosis and testing	Mothers who have suffered fetal or neonatal loss due to cardiac abnormalities should be offered fetal echocardiography in subsequent pregnancies.
5	1	Policy and procedure	Obstetrical care providers are reminded of guidelines concerning the length of the second stage of labour.
5	2	Communication/ documentation	Obstetrical care providers are reminded of the need to clearly document the rationale for a plan of management that deviates from the guidelines.
5	3	Policy and procedure	Obstetrical care providers are reminded of the guidelines for the identification and management of abnormal fetal heart rate patterns.
5	4	Diagnosis and testing	Obstetrical units are reminded to obtain cord blood gases at the delivery of compromised neonates.
5	5	Quality	The Committee recommends that the Regional Supervising Coroner (RSC) follow up with the hospital regarding the issues identified. If, in the opinion of the RSC, systemic issues persist, the RSC should consider conducting a Regional Coroner's Review with appropriate representatives from the hospital.
5	6	Diagnosis and testing	The parents of this infant should be advised to seek genetic counseling regarding the possibility of Cystic Fibrosis.
5	7	Quality	Hospital A should perform an Internal Review of this case.

Maternal and Perinatal Death Review Committee 2012 Annual Report

Case #	Rec #	Theme	Recommendations
6	1	Medical	Obstetrical care providers are reminded that the indication for induction of labour must be compelling.
6	2	Medical	Obstetrical care providers are reminded that fetal heart rate abnormalities may occur shortly after the administration of epidural analgesia and to be prepared to provide intrauterine resuscitation, including the possible use of quick acting tocolytics, or timely delivery.
6	3	Medical	Obstetrical care providers are reminded that the initiation of epidural analgesia in the setting of fetal heart rate abnormalities may further compromise the status of the fetus.
6	4	Medical	Obstetrical care providers are reminded that preparations should be made simultaneously for Caesarean section when a forcep procedure is indicated for an abnormal fetal heart rate tracing.
6	5	Policy and Procedure	Obstetrical care providers are reminded to have personnel skilled in neonatal resuscitation in attendance before delivery when fetal compromise is anticipated.
6	6	Policy and Procedure	Neonatal caregivers are reminded to follow Neonatal Resuscitation Guidelines with particular attention to intubation and methods to ensure correct placement of endotracheal tubes.
6	7	Medical	Neonatal caregivers are reminded that response to treatment should be monitored with blood gases in addition to neurological assessment.
6	8	Medical	Neonatal caregivers are reminded that volume expansion may be crucial in the treatment of a compromised baby who fails to respond to initial resuscitation procedures.
6	9	Communication and Documentation	Neonatal caregivers are reminded that calls to tertiary treatment centres should be placed as early as possible when dealing with a compromised neonate.
7			No recommendations
8	1	Education/Training	Hospital A should ensure that all obstetrical care providers in their hospital have updated training in Fetal Health Surveillance in Labour.
8	2	Quality	Hospital A should perform a QCIPA review of this case.

Maternal and Perinatal Death Review Committee 2012 Annual Report

Case #	Rec #	Theme	Recommendations
8	3	Communication/ documentation	Obstetrical care providers are reminded of the importance of complete, accurate, timely and detailed documentation.
9	1	Quality	Hospital B should complete a review of the maternal care provided to the mother of the deceased infant, including all documentation, including, but not limited to, the availability of physicians notes.
9	2	Quality	Hospital B should complete a quality of care assessment of the ordering and reporting of obstetrical ultrasound.
9	3	Medical	Obstetrical care providers are reminded of the importance of umbilical artery doppler in the assessment of placental function in pregnancies complicated by fetal growth restriction.
9	4	Quality	The Regional Supervising Coroner (RSC) should follow up with Hospital B regarding the issues identified. If, in the opinion of the RSC, systemic issues persist, the RSC should consider conducting a Regional Coroner's Review with appropriate representatives of the hospital.
10	1	Communication/ documentation	Obstetrical Care Providers are reminded: a) of the importance of complete, accurate, timely and detailed documentation;
		Diagnosis and testing	b) of the importance of appropriate implementation of intermittent auscultation (IA) and of timely response to abnormal fetal heart rates;
		Policy and procedures	c) of the Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline (JOGC September 2007, Volume 29, Number 9);
		Diagnosis and testing	d) of the importance of comparing maternal pulse with fetal heart tones during second stage of labor.
	2	Policy and procedure	Hospital A should revise their fetal health partograph to include all periodic changes (e.g. accelerations).
10	3	Quality	The Regional Coroner should perform a Regional Coroner's Review of the circumstances surrounding this death.

Maternal and Perinatal Death Review Committee 2012 Annual Report

Case #	Rec #	Theme	Recommendations
10	4	Policy and procedures	The SOGC should review the Fetal Health Surveillance in Labour Consensus Statement regarding intermittent auscultation (IA) to consider the absence of accelerations as an abnormal finding and to define baseline fetal heart rate changes.
11			No recommendations
12	1	Policy and procedures	Obstetrical care providers are reminded of the SOGC classification of intrapartum fetal heart rate monitor tracings and the actions indicated for atypical and abnormal tracings.
12	2	Medical	Obstetrical care providers are reminded that the use of oxytocin is contraindicated in the absence of a normal fetal heart rate tracing.
13	1	Policy and procedures	It is recommended that the physicians and healthcare providers at the ultrasound unit involved establish procedures to ensure direct and prompt communication of abnormal results and further assessment or action if urgent care is required.
13	2	Policy and procedure	Obstetrical care providers and staff in ultrasound units are reminded that when decreased fetal movement is suspected, there is a need for immediate assessment, communication of results and facilitation of further testing and management of care if necessary. There must be a method or procedure to directly and immediately communicate critical results to the most responsible physician or designate.
13	3	Diagnosis and testing	Technologists and radiologists interpreting biophysical profiles should have appropriate training and understanding of the implications of test results and appropriate responses and courses of action to follow.
14			No recommendations
15			No recommendations
16	1	Medical	Neonatal care providers are reminded that bile-stained vomiting and gastric aspiration in a neonate is usually an indicator of significant intestinal pathology that should prompt urgent referral to a paediatric centre for surgical assessment. In such a setting, the presence of malrotation with midgut volvulus should be considered by conducting appropriate imaging studies and laparotomy if indicated.

Maternal and Perinatal Death Review Committee 2012 Annual Report

Case #	Rec #	Theme	Recommendations
16	2	Quality	The hospital involved should ensure that the internal review performed included an examination of the clinical presentation, interpretation of investigations and response by those clinically overseeing this case.
16	3	Communication/ documentation Transport	The transport team at the children's hospital should review the audiotape of the conversation between the referring physician at the originating hospital and transport team regarding advice and recommended action.
17	1	Diagnosis and testing	Paediatric care providers are reminded of the importance of recognizing infants at risk for hypoglycaemia and initiating appropriate screening protocols to detect significant hypoglycaemia.
17	2	Policy and Procedures	Hospital A should create and implement processes for the detection of infants at risk for hypoglycaemia and the activation of appropriate screening protocols.
18	1	Medical	In the context of abruptio placenta, obstetrical care providers are reminded that immediate delivery should be strongly considered at the first signs of maternal and/or fetal compromise. This intervention may be lifesaving.
18	2	Medical	Obstetrical care providers and providers of obstetrical analgesia/anesthesia should be aware that "the most common side effect of epidural or spinal anesthesia is hypotension with functional hypovolemia." ³ In a clinical scenario where maternal hypovolemia and/or hypotension are already present or likely, alternative forms of maternal analgesia may be warranted, and general anesthesia should be considered as an option for management of Caesarean section.
18	3	Quality	Hospital B should perform an Internal Review of this case.
19	1	Policy and Procedure	Obstetrical care providers are reminded of the SOGC guideline on intrapartum fetal surveillance and the identification and management of an abnormal fetal heart rate tracing. (available online at : http://www.sogc.org/guidelines/documents/gui197CPG0709r.pdf)
20	1	Other	The Canadian Pediatric Surveillance Program (CPSP) initiative collecting data on early onset neonatal sepsis should consider adding information regarding maternal use of water immersion during labour and delivery.

Stillbirth Case Reviews

Case #	Rec #	Theme	Recommendations
1	1	Quality	A Regional Coroner's Review should be conducted to review the care provided to this expectant mother. This review should consider: timing, communication between shared caregivers, monitoring of low symphysis fundal height (SFH) growth, flagging of follow-up appointments not attended, decreased fetal growth before hypertension, work/social issues (e.g. increased physical stress from standing and/or lifting and lack of social support networks), interval growth issues, appropriate and timely testing for proteinuria, and surveillance based on complete clinical picture.
2	1	Communication/ documentation	Obstetrical care providers are encouraged to use supplementary progress notes rather than excessive entries on the Ontario Antenatal Record 2.
2	2	Resources	Obstetrical care providers are reminded of the importance of adequate resources to provide endotracheal intubation if necessary when the amniotic fluid is meconium stained.
3			No recommendations
1	1	Diagnosis and testing	Obstetrical care providers reminded of the importance of confirming the menstrual dates with the gestational age on ultrasound assessment.
4	2	Diagnosis and testing	Obstetrical care providers reminded of the importance of post dates fetal surveillance.
5	1	Policy and procedures	Obstetrical care providers are reminded of the SOGC Guidelines "Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline."
5	2	Medical	Obstetrical care providers are reminded that oxytocin is contra-indicated in the absence of a normal fetal heart rate tracing.
5	3	Quality	The Committee recommends that the Regional Supervising Coroner (RSC) follow up with the hospital regarding the issues identified. If, in the opinion of the RSC, systemic issues persist, the RSC should consider conducting a Regional Coroner's Review with appropriate representatives from the hospital.

Maternal and Perinatal Death Review Committee 2012 Annual Report

Case #	Rec #	Theme	Recommendations
6	1	Quality	The hospital involved should review the management of trauma to pregnant patients and the communication and collaboration between the Obstetrical and the Emergency Departments.
6	2	Medical	Obstetrical care providers are reminded of the potential subtle nature of the evolution of an abruption with abdominal trauma.
7	1	Communication/ documentation	Obstetrical health care providers are reminded to document maternal heart rate to ensure differentiation from fetal heart rate.
7	2	Medical	Obstetrical health care providers are reminded that operative vaginal birth should be considered as an alternative to Caesarean Section in the setting of non-reassuring fetal status assuming the prerequisites are present and there are no contraindications, as per the SOGC Clinical Practice Guidelines for Operative Vaginal Birth (No. 148, August 2004).
8	1	Diagnosis and testing	Obstetrical care providers are reminded of the importance of appropriate monitoring of blood sugar levels in gestational diabetes to determine if insulin is required.
8	2	Medical	Obstetrical care providers are reminded to consider referral to an endocrinologist if there is any question about adequate blood sugar control.
8	3	Medical	Obstetrical care providers are reminded of the increased risk of fetal morbidity and mortality associated with oligohydramnios as documented on ultrasound.
8	4	Diagnosis and testing	Obstetrical care providers are reminded of the criteria for interpreting non stress test (NST) results.
8	5	Medical	Primary Health Care Providers should consider early referral to an obstetrical care provider when a pregnancy is considered high risk.

Maternal and Perinatal Death Review Committee 2012 Annual Report

Case #	Rec #	Theme	Recommendations
9	1		Obstetrical care providers are reminded of the following recommendations pertaining to fetal health surveillance from previous reviews conducted by the Maternal and Perinatal Death Review Committee (MPDRC):
		Diagnosis and testing	Fetal well-being should be assessed without delay when patients present with decreased fetal movements. (2006 Stillbirth)
		Diagnosis and testing	Obstetrical care providers are reminded that a non-reassuring fetal heart rate tracing should prompt another test of fetal well-being, such as fetal scalp sampling, or expedited delivery. (2006 Stillbirth)
		Diagnosis and testing	Obstetrical care providers are reminded of the importance of identifying and acting on abnormalities in fetal heart monitoring. (2009 Neonatal)
		Diagnosis and testing	Obstetrical care providers are reminded of the criteria for an abnormal fetal heart rate tracing and the action required as defined by SOGC Intrapartum Fetal Surveillance Guidelines published in September 2000. Obstetrical care providers are reminded of the criteria for an abnormal fetal heart rate tracing and the action required as defined by SOGC Intrapartum Fetal Surveillance Guidelines published in September 2007. (2010 Neonatal)
		Diagnosis and testing - EFM	It is recommended that electronic fetal monitoring be continued while preparations are made for emergent Caesarean section. (2011 Stillbirth)



Questions and comments regarding this report may be directed to:

Maternal and Perinatal Death Review Committee

Office of the Chief Coroner
25 Morton Shulman Avenue
Toronto, ON
M3M 0B1