



CONDUCT COUNTS!

CASE OF THE MONTH

Most of the cases highlighted by the CRTO relate to “conduct unbecoming a professional” as the vast majority of investigations are about poor behaviour or communication. In fact, less than 20% of CRTO cases include allegations related to clinical care.

This month, however, the CRTO is publishing a report forwarded by the Office of the Chief Coroner on a Maternal and Perinatal Death Review Committee investigation. The Committee looked into the actions that contributed to the death of an infant 20 days after birth. Specifically, the Committee considered the diagnostic ultrasound imaging that failed to detect congenital heart disease, the difficulties resuscitating the infant, and delay in transferring the patient. The Committee makes several recommendations that are particularly relevant for RTs practising in the areas of L&D, NICUs and transport.



RESOURCES

[A Commitment to Ethical Practice](#)
[CRTO Standards of Practice](#)

PROFESSIONALISM

“Professionalism” or professional conduct is a term often used to describe the behaviours that are expected of individuals who hold a certain role in society. A “professional” is typically someone who has obtained skills that are recognized as requiring specific, intensive training and who applies those skills in a position impacting others (e.g., engineer, lawyer, RT, PT, MD, etc.). Professionals are often held to moral, ethical and legal standards because of this potential impact.



MATERNAL AND PERINATAL DEATH REVIEW COMMITTEE

This document is produced pursuant to section 15(4) of the Coroners Act, R.S.O. 1990, c. 37, on the basis that it is to be used for the sole purpose of a Coroner's investigation, and not for any litigation or other proceedings unrelated to the Coroner's investigation. Moreover, the opinions expressed herein do not necessarily take into account all of the facts and circumstances surrounding the death. The final conclusions of the Coroner's investigation may differ significantly from the opinions expressed herein.

Neonatal Death

Date of death: August 15, 2015.
OCC file: 2015-10389 (MPDRC 2016-N-10)
Age: 20 days

Records Reviewed:

- Coroner's Investigation Statement
- Medical records from Hospital A , Children's Hospital A and Children's Hospital B
- Obstetrical ultrasounds
- Post mortem examination report
- Correspondence from the parents

Antenatal History

The mother of the deceased infant was a 36-year-old G₄P₂ with an estimated date of delivery of August 3, 2015 confirmed by early ultrasound. Routine prenatal laboratory investigations were normal and integrated prenatal screening was negative.

Her past obstetrical history included a spontaneous abortion in 2008 and a twin pregnancy in 2009 delivered at 36 weeks by Caesarean section for malpresentation.

The past medical history was non-contributory and family history was negative for congenital heart disease.

On March 3, 2015, a fetal morphology assessment was performed at 18 weeks' gestation. The report indicated growth parameters of 18 weeks' gestation. There was no placenta previa. The anatomy was normal, however the four-chamber view of the heart, outflow tracts and spine were not well seen. A follow-up ultrasound was suggested in 1-2 weeks. The follow-up ultrasound on March 17 2015 was a limited ultrasound of heart and spine. The report indicated "morphology completed" and "boxes" were checked indicating the four-chamber heart, outflows and spine where normal.

The mother also underwent an ultrasound on June 2 at 31 weeks' gestation for fetal growth. The ultrasound report indicated that the baby was in breech presentation. Growth parameters and the amniotic fluid volume were normal. The biophysical profile (BPP) was 8/8.

Glucose challenge test (GCT) was normal and she was Group B streptococcus (GBS) negative.

Identified risk factors on the antenatal record were trial of labour for vaginal birth after Caesarean (VBAC) and a BMI 43. Originally the plan was to VBAC, but a repeat Caesarean section was scheduled for July 27, 2015.

Course in Labour and Delivery

The mother was admitted to Hospital A on July 26, 2015 at 38 weeks 6 days gestation, in early labour after contractions onset at 0200 hours. On examination at 0525 hours, a bedside ultrasound confirmed vertex presentation. Amniotic fluid volume was noted to be markedly increased. The cervix was 1cm dilated, 50% effaced with the presenting part at spines -1. A decision was made to proceed with a trial of labour and she was admitted at 0610 hours.

At 0800 hours, contractions were stronger, but the cervix was unchanged at 1cm. The fetal heart rate (FHR) was baseline 140 bpm with minimal variability, no accelerations and no decelerations. Despite strong contractions, there was no change in the cervix and the decision was made to proceed to repeat Caesarean section.

At 1436 hours, under spinal anaesthesia, she was delivered of a 3675 g female infant. A large amount of amniotic fluid was noted on entry into the amniotic cavity. There were no cord complications and no meconium. The infant had spontaneous cry, pallor, and was hypotonic. Apgars were 1, 5, 6 and 5 at one, five, 10 and 20 minutes. Cord gases were venous pH 7.21 BE -6.3 and arterial pH 7.13 and BE -6.7.

At 1437 hours, the infant was suctioned for secretions. Her heart rate dropped to 90 bpm and positive pressure ventilation (PPV) was started with room air. The heart rate dropped to 70 bpm at two minutes of age and FiO₂ was increased to 30%.

At 1439 hours, her heart rate dropped to 50 bpm and FiO₂ was increased to 100%. A code pink was called.

At 1440 hours, chest compressions were initiated and a #1 laryngeal mask airway (LMA) was inserted by the anesthetist and respiratory therapist.

At 1441 hours, when the infant was five minutes of age, the pediatrician attempted to intubate the infant and was unsuccessful.

At 1443 hours, the heart rate dropped to 50 bpm and there was very poor respiratory effort. Chest compressions were restarted and continued with 100% oxygen.

At 1444 hours, the heart rate was up to 80 bpm and compressions were discontinued. The infant remained cyanotic and had poor respiratory effort. She was transitioned to continuous positive airway pressure (CPAP) of 5 cm with 100% oxygen.

At 1446 hours, an orogastric tube was placed and 12 ml of secretions were aspirated. One minute later, the heart rate was 130 bpm with oxygen saturation of 80%.

At 1448 hours, the oxygen saturation dropped to 73% and there was intercostal indrawing and grunting. The infant remained on CPAP.

At 1459 hours, an attempt was made to discontinue CPAP and start free flow with 100% oxygen. The infant was placed back on CPAP due to a drop in the oxygen saturation to 79% and a respiratory rate of 32 breaths per minute. Her blood pressure was 57/48, mean of 52 and she remained pale and cyanosed.

A chest X-ray was done at 1515 hours when the infant was 39 minutes of age. The X-ray showed slight deviation of trachea, minimal aeration of left lung and cardiomeastinal silhouette appeared enlarged.

At 1520 hours, pre and post oxygen saturation was measured and showed 89% and 72% respectively. This was consistent with intra-pulmonary shunting.

At 1529 hours (at 53 minutes of age), a peripheral IV was inserted and a 30 ml bolus of saline was given. The infant remained flaccid, cyanosed with O₂ saturation of 76%.

At 1536 hours, a second bolus of 30 ml of saline was given. There was slight improvement with a heart rate of 150 bpm and a respiratory rate of 60 breaths per minute. The oxygen saturation again dropped to 74%.

At 1600 hours (at one hour and 24 minutes of age), a second intubation attempt was planned with rapid sequence induction (RSI).

At 1608 hours, fentanyl 6 mcg, atropine 0.6 mg, and succinylcholine 4 mg was given over two minutes.

At 1611 hours, intubation was attempted. On auscultation, there was poor air entry on the right and none on the left.

At 1612 hours, heart rate dropped to 50 bpm and oxygen saturation was 67%. Placement of the tube was uncertain, so it was removed and positive pressure ventilation (PPV) was started.

By 1615 hours, heart rate was up to 80 bpm and oxygen saturation was 70%.

At 1623 hours, after a third attempt, the respiratory therapist successfully intubated the infant. The infant was now one hour and 47 minutes of age.

A minute later, the infant's heart rate again dropped to 58 bpm; chest compressions were restarted. The heart rate rose to 126 bpm, but the infant continued to be cyanosed (71%). Blood glucose was low at 2 mmol and 10% dextrose was administered.

At 1635 hours, a capillary gas was done and showed: pH 6.8, pCO₂ 115, (capillary refill was 4.5 seconds). A call was placed for the transport team from Children's Hospital A. The call was delayed due to intubation.

A repeat chest X-ray at 1636 hours showed improved aeration of the left lung with no evidence of pneumothorax effusion or consolidation.

At 1647 hours, the heart rate was 159 bpm and oxygen saturation was 81% and capillary refill time was three seconds. A complete blood count showed: Hgb of 196, WBC 18.3, neutrophils 7.69%, and platelets were 73. Blood culture was sent.

At 1715 hours, the transport team from Children's Hospital A arrived. The request was to transfer the infant for respiratory distress and to rule out hypoxic-ischemic encephalopathy and heart disease.

At 2000 hours, the infant was further stabilized and transported to Children's Hospital A.

At 2020 hours, the infant arrived at Children's Hospital A. She was noted to be cyanotic on 100% O₂ with poor perfusion. She was acidotic with elevated serum lactate levels and had acute renal and liver injury. An echocardiogram demonstrated a small patent ductus arteriosus (PDA), pulmonary atresia, hypoplastic right ventricle, dilated and poorly functioning left ventricle with compacted myocardium and aortic stenosis. She was given prostaglandins with good effect and inotropic support was initiated. There was gradual improvement in left ventricular function.

On July 27, 2015, a head ultrasound revealed patchy increased echogenicity in the posterior periventricular white matter suggestive of edema. There was also evidence of intraventricular hemorrhage.

On day three of life, the infant was transported to Children's Hospital B. She remained hypotensive and requiring inotropic support.

On July 30, 2015, the infant underwent angiography and aortic balloon dilation. The left anterior descending coronary artery was found to be narrowed. Improvement over the next few days led to weaning off epinephrine on July 31 and norepinephrine on August 2 with improving lactate levels. The plan was for heart transplantation if a heart became available.

On August 12, 2015, the infant's lactate level rose. An echocardiogram showed a dilated hypertrophied left ventricle. The left ventricular ejection fraction was 41%. Cardiac ischemia was identified.

On August 15, 2015, the infant went into cardiac arrest and CPR was initiated. She did not respond to resuscitative efforts and given the severe underlying heart disease, CPR was stopped after 13 minutes. The infant died at 1957 hours on August 15, 2015.

Post Mortem Examination Report

The autopsy was restricted to the heart only.

Autopsy confirmed the unusual combination of pulmonary atresia with intact ventricular septum together with aortic valve stenosis. The aortic valve was thickened and bicuspid. There was marked difference between the right and left main stem coronary arteries. The right main coronary artery was enlarged with a diameter of 4mm whereas there was severe stenosis of the left main coronary artery measuring 1mm in diameter. The heart was enlarged weighing 66 g, approximately three times normal with a very small right ventricular cavity and a very large left ventricular cavity.

There was marked ischemic damage to the left ventricular myocardium with atrophy, fibrosis and calcification of the left ventricular anterior and posterior papillary muscles. There was also diffuse subendocardial fibrosis.

It was commented on that the combination of pulmonary atresia/intact interventricular septum with aortic stenosis was particularly unusual and that in most instances, obstruction to both the left and right ventricular outflow tracts in embryonic/fetal life results in intrauterine death.

Pathology report on the placenta was unremarkable other than a mild increase in weight (i.e. 686 g vs. expected 426-611 g).

Summary

This infant died 20 days after birth from complications of congenital heart disease. The congenital heart disease was not diagnosed antenatally and thus was unknown to the care providers at Hospital A.

The infant was severely depressed at birth and a code pink was called. Difficulties were encountered during the resuscitation with two failed attempts and finally successfully intubated on third attempt at two hours of age. Her ventilatory support was sub-optimal and very labile oxygenation with O₂ saturation dropping to 40% despite 100% inspired oxygen. The infant also had frequent bouts of bradycardia with heart rate down to 58 bpm requiring repeated cardiac compressions.

Although there were difficulties in resuscitation, a call to Children's Hospital A was not placed for two hours, resulting in delayed stabilization of the infant. Although cyanotic heart disease was suspected, treatment with prostaglandin was not considered.

The infant was transferred to Children's Hospital A where testing revealed acidosis, acute renal and hepatic injury. Inotropic support was given. An echocardiogram revealed numerous congenital heart abnormalities and the infant was transferred to Children's Hospital B at three days of life where aortic valve balloon dilatation resulted in some clinical improvement.

The infant was on the cardiac transplant list, but on August 12, her condition became more unstable. The tenuous nature of her circulation both in terms of maintaining coronary perfusion and balance between systemic and pulmonary blood flow was problematic. She was noted to have more ischemic episodes as indicated by ST segment changes and she arrested on August 15, 2015.

Discussion

Review of ultrasound images

The assessment of the March 3, 2015 ultrasound image of the heart and spine was not optimal. The four-chamber heart and outflows tracts were not adequately seen. This was correctly reported and a follow up was recommended.

On the March 17, 2015 ultrasound assessment, the heart and spine were checked off as “normal.” Upon review of the images however, the technical quality of the heart was poor. The transverse section of the fetal chest was very small, occupying less than ¼ of the image. The depth adjustment was too deep. The four chamber heart view was not well seen. The left outflow track view appeared to be seen, but the right outflow track was not well seen.

Detection of congenital heart disease

Congenital heart disease is a leading cause of infant mortality, with an estimated incidence of about 4–13 per 1000 live births. Structural cardiac anomalies are among the abnormalities most frequently missed by prenatal ultrasonography. Detection rates can be optimized by performing a thorough screening examination of the heart including the four-chamber view and the outflow tract views.

The requirement for a complete second trimester ultrasound to include a four-chamber view of the heart and the cardiac outflow tracts¹ is to screen for congenital heart disease. Despite this, current screening approaches continue to fail to detect a significant percentage of major forms of congenital heart disease. These structures were not seen on the original second trimester ultrasound on March 3. A follow up ultrasound was requested because of this and the repeat on March 17 reported a normal four-chamber view and outflow tracts. There was no indication of technical difficulties obtaining the necessary views. The severe anomalies in the outflow tracts in this infant raise the question as to whether these should have been detected at the second trimester ultrasound.

The International Society of Ultrasound in Obstetrics and Gynecology (ISUOG) Practice Guidelines relating to sonographic screening examination of the fetal heart recommends that “cardiac images should be magnified until the heart fills at least one third to one half of the screen. The cine-loop feature should be used to assist the real-time evaluation of normal cardiac structures.”² The requirements for an adequate four-chamber view and assessment of outflow tracts are outlined.

¹ “Content of a Complete Routine Second Trimester Obstetrical Ultrasound Examination and Report” SOGC Clinical Practice Guideline JOGC March 2009.

² ISUOG Practice Guidelines (updated): sonographic screening examination of the fetal heart Ultrasound Obstet Gynecol 2013; 41: 348–359

If severe heart anomalies associated with a high risk of intrauterine death had been diagnosed prenatally, the management options would have included consideration for termination of the pregnancy or arrangements for delivery at a tertiary care centre with expertise in the management of congenital heart disease. Ultimately heart transplant would be necessary for survival. It cannot be determined from this review whether antenatal detection would have changed the outcome.

Recommendations

To Sonography Canada, Ontario Association of Radiologists, Ontario Association of Medical Radiation Sciences, Ontario Society of Diagnostic Medical Sonographers, Ontario Association of Radiologists, Society of Obstetricians and Gynaecologists, Ontario College of Physicians and Surgeons and Ontario Medical Association:

1. Obstetrical imagers, both technologist and radiologists, are reminded of the technical requirements and imaging guidelines for evaluation of the fetal heart in the second trimester.

To the College of Physicians and Surgeons and College of Respiratory Therapists,

2. In a level 2 NICU, ability to intubate and provide appropriate respiratory support is crucial. Care providers need to be up to date on the process of intubation and ventilation.

To the College of Physicians and Surgeons:

3. Early communication with the tertiary care centre is recommended for guiding the treatment and expediting transfer.