Rare but Fatal Complication of Umbilical Venous Catheterization

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A B S T R A C T

Umbilical venous catheterization in neonates is an intravascular infusion route for resuscitation and maintenance fluids, blood and blood products, parenteral nutrition, and hypertonic solutions that can be used as an alternative when peripheral venous access is not possible. When used, special precautions should be taken and guidelines followed to prevent rare but often fatal complications.

Key Words. Cardiac Tamponade; Umbilical Venous Catheter; Complication; Newborn

Introduction

Cardiac tamponade secondary to central venous catheterization is rare, but potentially lethal. To avoid this complication, the catheter tip should be prevented from entering the right atrium and its position should be checked periodically by chest X-ray.1,2

Cardiac tamponade should be suspected in any patient with a central venous catheter whose condition deteriorates suddenly.1,3 We present 2 infants who had similar problems but with different outcomes as a result of high index of suspicion of cardiac tamponade. Immediate evacuation of the pericardial fluid can be life saving and can greatly influence the outcome as exemplified by our cases. This article will highlight the importance of confirming and reconfirming the position of the catheter tip with anteroposterior and, if possible, lateral radiographs, timely diagnosis, and prompt therapeutic intervention.

Case Report 1

A 3-day-old girl, second of twins who was born at 32 week of gestation weighing 1620 g, was admitted to the neonatal intensive care unit as a case of nonimmune hydrops secondary to twin-to-twin transfusion. Umbilical arterial and venous catheters were inserted. Normal position of umbilical lines was confirmed by chest and abdominal X-ray. She did not have significant pleural effusion but needed abdominal paracentesis to relieve massive ascites. Echocardiogram confirmed the presence of persistent pulmonary hypertension but absence of pericardial effusion. The baby was ventilated by high-frequency ventilation and inhaled nitric oxide therapy from admission and needed inotropic support of dopamine at 10 µg/kg/minute. She improved dramatically and by day 2 was on very minimal high-frequency ventilation settings, off inhaled nitric oxide and dopamine therapy.

Eight hours later, she died suddenly following an unexplained collapse, profound bradycardia, and failure to respond to aggressive resuscitation. Postmortem pericardiocentesis revealed 30 cc of straw-colored infusate. However, analysis of the pericardial fluid showed the same composition as dextrose solution in the umbilical venous catheter (UVC).

Case Report 2

A full-term baby girl with a birth weight of 2975 g had meconium aspiration and Stage I hypoxic ischemic encephalopathy secondary to severe birth asphyxia. An umbilical venous catheter (5 Fr gauge-polyvinyl chloride catheter) was inserted during resuscitation at the delivery room. The position of umbilical line at the level of T8 was confirmed by chest and abdominal X-rays at the neonatal intensive care unit (Figure 1). The baby improved satisfactorily and remained stable. By 32 hours of age, she was in room air with flow of 1 L/minute by nasal cannula and renal dose of dopamine 2 µg/kg/minute.
At about 36 hours of age, she had a sudden unprovoked episode of profound apnea and bradycardia associated with marked hypotension and unrecordable SaO2. Aggressive respiratory and cardiovascular resuscitation failed to elicit significant overall improvement. Air entry was normal and chest transillumination did not show evidence of pneumothorax. Ultrasound of the heart was performed in the absence of immediate access to echocardiogram and confirmed our initial suspicion of cardiac tamponade. Urgent pericardiocentesis was performed and 35 cc of straw-colored fluid was removed. Analysis of the infusate showed high glucose level of 72 mmol and calcium of 0.93 mmol, which has the same composition as the intravenous fluid being infused through the UVC. She recovered within a few minutes and achieved normal perfusion, heart rate, oxygen saturation, and blood pressure. Follow-up chest X-ray showed a displaced UVC at the level of T6 (Figure 2). It was pulled out by 2 cm to level of T8. Echocardiogram performed after stabilizing the patient still demonstrated the presence of moderate pericardial effusion and the UVC tip just entering the left atrium (Figure 3). The umbilical venous catheter was removed altogether. Two days after the event, she was extubated to room air, and follow-up echocardiogram 3 days later showed complete resolution of the pericardial effusion.
Discussion

The use of UVCs for hyperalimentation has been increasing in the neonatal period. Pericardial effusion and pericardial tamponade, although rare in newborns, are known complications of central venous catheterization. Few cases of cardiac perforation secondary to central venous catheters have been reported. This complication may be overlooked and the outcome can be detrimental. The variation in reported incidence and time to presentation is partly related to the many risk factors associated with this complication. The exact cause of the relatively bloodless pericardial effusion remains in question. However, it has been postulated that the primary etiology of cardiac perforation is inappropriate placement or migration of the catheter within the heart.

It is theorized that movement of the head and neck, respiration, and even the normal cardiac cycle are associated with catheter migration. Direct trauma during insertion also predisposes to vascular or endocardial damage and perforation. The perforation can be caused either by direct puncture or from endothelial damage by the hypertonic solutions, and high-pressure infusions. It may be secondary to a direct perforation that self-seals. Several authors, however, have speculated that perforation is secondary to endocardial damage from repeated contact of the UVC tip with the myocardial wall, resulting in thrombus formation and adherence of the central venous catheter to the myocardium. The angle that the catheter tip forms with the wall of a vein or cardiac chamber is also thought to be an important factor responsible for vessel trauma. When the tip lies at a more perpendicular angle to the wall, there is an increased chance of direct trauma and erosion.

Hyperosmolar fluid in direct contact with the endocardium can cause osmotic injury. Such damage may cause thrombus formation and myocardial necrosis, resulting in leaked fluid accumulation in the pericardial space. Fluid transmurally diffuses across the myocardium, or the central venous catheter erodes into the pericardial space, resulting in a pericardial effusion that predominantly does not contain cellular elements. It has also been speculated that rapid injection of contrast or hyperosmolar agents at high pressure may increase the risk of perforation. The use of radiography to determine the catheter tip position has been evaluated by a number of authors. The optimal position for the catheter is at the junction of the inferior vena cava and the right atrium with the tip visible between the 8th and 9th thoracic vertebrae. Ades et al. have shown that UVCs properly placed in the right atrial/inferior vena cava junction, as demonstrated by echocardiography, were located at a wide range of vertebral bodies (T6–T11).

The wide variability in atrial size and position with redundancy of the atrial septum in newborns with otherwise normal cardiac anatomy may explain why radiographic landmarks do not correlate with intracardiac anatomy. Malposition has been defined either as a catheter tip visible above the 7th vertebra with significant potential for migration, or as a position below inferior vena cava and right atrial junction with potential intra- or extrahepatic complications. The UVC tip should remain outside the cardiac silhouette but still within the vena cavae (approximately 1 cm outside the cardiac silhouette in premature infants and 2 cm in term infants).

The experience with these 2 cases confirms the importance of determining the position of the catheter tip before use, and of periodically ascertaining the position of the catheter tip by using anteroposterior as well as lateral radiographs. The radiation burden in this group of neonates is low and the benefits of diagnostic radiographs far outweigh any potential radiation risks. Pericardial effusion should be suspected, confirmed if possible, and aspirated immediately in any baby receiving intravenous fluid or parental nutrition via a centrally placed venous catheter and presenting with acute circulatory collapse that is not responding to normal resuscitation measures. Cardiac auscultation can easily identify muffled heart tones. If this complication is suspected, time should not be wasted on radiographic confirmation, arterial blood gas sampling, echocardiography, or any other diagnostic procedure. The infusion must be stopped and an attempt made to aspirate the fluid from the pericardial space by pericardiocentesis.

Conclusion

This report illustrates the need to maintain a high level of awareness about catheter tip position, even when preventive practices suggest it is sited correctly. One must adhere to guidelines regarding indications and techniques and be aware of the possible complications, their prevention, and management.

Cardiac tamponade must be considered in any patient with an indwelling central venous catheter.
who shows evidence of clinical deterioration, however vague the symptoms and signs. Resuscitation using the catheter should be avoided until cardiac tamponade has been excluded. It is mandatory to periodically ascertain the position of the catheter tip by radiographs. Although cardiac tamponade is a serious emergency, it is a treatable condition provided one has a high index of suspicion.

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