Learning from errors

Parenteral nutrition – ascites with acute renal failure as a complication from an umbilical venous catheter in an extremely low birth weight infant

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Summary

Umbilical venous catheters (UVCs) are frequently used in the neonatal intensive care setting and play a crucial role in the management of extremely low birth weight (ELBW) infants. One very rare complication reported is parenteral nutrition (PN) ascites secondary to vessel perforation or hepatic erosion by PN at the tip of malpositioned UVCs with various hepatic lesions. We describe a case of early onset PN ascites with no obvious associated hepatic lesion but complicated by pre-renal acute renal failure in an ELBW infant with the tip positioned between the 10th and 11th thoracic vertebrae.

BACKGROUND

The use of umbilical venous catheters (UVC) was first reported by Diamond in 1947,¹ for exchange transfusions in neonates with erythroblastosis fetalis. In addition, it provides intravascular access for the administration of intravenous fluids, parenteral nutrition (PN), drugs, transfusions and central venous pressure monitoring especially for the management of extremely low birth weight (ELBW) infants.² However, the use of UVCs is not without its risk as improper tip positioning is associated with complications that may lead to morbidity and occasionally mortality.³ Ideally, the tip should be positioned at the junction of the inferior vena cava and right atrium (ie, just above the diaphragm). Seguin *et al*² reported a 92% initial success rate for UVC placement, but only 68% were reported to be just above the diaphragm.

We describe a case of early onset PN ascites with pre-renal acute renal failure (ARF) on day 2 of life in an ELBW infant from a UVC which on plain abdominal radiograph appeared to be just below the diaphragm (between the 10th and 11th thoracic vertebrae). The procedure was performed by a paediatric training doctor with previous experience in inserting central lines. Although the tip of the UVC was noted to be just below the diaphragm, it was thought to be safe for infusing PN.

Early recognition and subsequent appropriate management led to resolution of both complications without any long-term sequelae. This case highlights a potential life-threatening complication secondary to malpositioned UVC tip leading to a sudden, unexplained ascites with ARF in an ELBW infant.

CASE PRESENTATION

A male infant was born by spontaneous vaginal delivery at 26 weeks' gestational age with birth weight of 930 g. The infant was born in good condition with Apgar scores of 4, 7 and 9 at 1, 5 and 10 min of age. He was electively

intubated, given prophylactic surfactant, and extubated 2 h later to nasal continuous positive airway pressure at 5 cm H₂O in room air. A double lumen UVC (Vygon, 4.0F) was inserted on second attempt. The first attempt had no flow back and hence was removed. A similar size UVC was placed without resistance and with good back flow on second attempt. The UVC tip was seen just below the diaphragm between the 10th and 11th vertebra on the abdominal anteroposterior radiograph (figure 1). Although not optimal, the position was thought to be acceptable and PN in 10% dextrose was commenced.

Over the next 8–12 h, the abdomen was noted to be shiny and distended with scrotal oedema. This was followed by decreased urine output and biochemical features suggestive of pre-renal ARF (increasing serum potassium, urea and creatinine at 36 h of age). The peak serum potassium, urea and creatinine of 8.4 mmol/l, 19.7 mmol/l and 223 µmol/l, respectively, were noted between 46 and 67 h of life (table 1). Although, central venous pressure monitoring would have been helpful, it is not considered in this case. He was given a fluid challenge with frusemide, which transiently improved his urine output (from 0.3 to 1.6 ml/kg/h). He also received intravenous salbutamol, and dextrose with insulin infusion for hyperkalaemia but he never showed any electrocardiographic changes. A diagnostic ascitic tap was performed at 28 h under ultrasound guidance, which revealed a yellow straw-coloured fluid. Ascitic fluid testing revealed glucose of >110 mmol/l (>20 g/l or >2000 mg/dl). This was subsequently confirmed on laboratory biochemical analysis to be consistent with the PN. A diagnosis of PN ascites was confirmed.

As part of his workup, he had an abdominal and renal ultrasound, which showed ascites extending into the scrotum. Both kidneys were normal in size, and appeared echogenic with poor corticomedullary differentiation. Both renal veins and arteries were patent on Doppler examination. The liver and the gall bladder appeared normal on

Figure 1 Anteroposterior plain radiograph showing UVC tip at T10 position.

Table 1 Daily fluid intake, serum electrolytes, urea, creatinine (mmol/l) and urine output

Day of life	Fluids (ml/kg/day)	Na (mmol/l)	K (mmol/l)	Urea (mmol/l)	Creatinine (μ mol/l)	Urine (ml/kg/h)
D1	90	138	5.7	6.7	85	4.2
D2	120	136	7.4	11.8	129	0.3
D3	160	135	7.2	16.9	193.7	4.5
D4	180	135	8.2	19.4	222	6.0
D5	180	134	5.9	16.5	177	7.2
D6	180	127	6.1	14.7	146	
D7	180	132	5.6	13.6	118	

ultrasound. The length of the indwelling portion of the umbilical line was checked from the umbilical stump (done twice a day) as well as on plain radiograph. Both showed it to be in the same position as immediate after insertion (figure 2). This ruled out the displacement or spillage of the UVC.

OUTCOME AND FOLLOW-UP

The UVC was removed and replaced with a percutaneous intravenous central catheter. The renal functions and electrolytes improved slowly over the next couple of days and normalised by day 10 of life. The ascites resolved over

the course of the next 3 days on repeat ultrasound scans. During this episode, there was no deterioration in his respiratory parameters or his liver functions and his mean blood pressure (non-invasive) and blood sugars remained normal. There was no periventricular leucomalacia noted on cranial ultrasound. He was followed up for neurodevelopmental assessment until 3 years of age. His neurodevelopmental outcome at 3 years of age was normal.

DISCUSSION

In our ELBW infant, a double-lumen UVC catheter was used, which does not have an increased risk of malposition

Figure 2 Plain abdominal radiograph showing UVC at the same level as postinsertion and features of ascites. This radiograph was taken just before the UVC was removed.

as compared with single lumen UVCs.⁴ The tip was seen at the level between the 10th and 11th thoracic vertebra, which was not the perfect position. Ideally, the UVC tip should be in the inferior vena cava at or above the level of the diaphragm. This normally corresponds to the level between the 7th and 9th thoracic vertebrae on a radiograph. The majority of UVC tips sited below this level were found to be within the liver.⁵ In all the reported cases of PN ascites as a complication of UVC, the tips were in a suboptimal position below the diaphragm, overlying the liver and lying between 10th and 12th vertebrae similar to our infant.^{6–10}

The tip position in our infant was confirmed on an AP view of a plain radiograph. Use of ultrasound/echocardiography has been found to be superior to plain radiographs. As well as avoiding radiation injuries, it avoids re-positioning of the patient and allows visualisation in relation to cardiovascular structures.^{5 11 12} In fact when using radiographs for confirming the catheter tip, lateral views may be better than AP views in confirming the tip position.¹³

Roughly between 8 and 12 h postinsertion, abdominal distension was noted, which gradually increased with deteriorating renal functions. In reported literature, the mean age of presentation of symptoms was 4 days postinsertion.^{6–7 14} The very early presentation in our case

makes it unique from all other presentations reported in literature.

Intraperitoneal extravasation of PN or PN ascites has been cited as one of the complications associated with malpositioned UVC, which may lead to vessel perforation or liver capsule disruption as a result of hepatic necrosis.⁶ The exact mechanism of PN ascites in our infant remains unknown, although hepatic erosion seems the most likely explanation. Our infant remained haemodynamically stable throughout and his haemoglobin concentration was maintained throughout. The above observation and the nature of the ascitic fluid aspirated on paracentesis suggest direct perforation of the umbilical vein vessel wall very unlikely. Of the seven cases reported by Coley *et al*,⁷ none had changes in haemoglobin concentration but contrast studies through the UVCs in six patients showed abnormal liver parenchymal staining, indicating venous perforation and hepatic injury, with three showing obvious peritoneal extravasation. Paracentesis was performed in seven patients, and five revealed cloudy fluid high in glucose, triglycerides and protein consistent with PN similar to our infant. Four patients, who had ultrasound examinations, revealed intrahepatic location of the UVC catheter tip, with heterogeneous distorted hepatic parenchyma indicative of hepatic damage. At the time of perforation, changes

in haemoglobin concentration or alterations in liver function test were not observed.

In another report, Lim-Dunham *et al*⁸ identified four neonates with erosion of the UVCs into hepatic parenchyma over a 2-year period. All four had abdominal distension within 9 days of UVC placement, and in all the cases, the tip was located below the diaphragm and superimposed over the liver on plain AP radiograph. Ultrasound of all patients showed intraparenchymal liver lesions with an echogenic rim and hypoechoic centre. Ascites was identified in all four with fluid proven to be PN by paracentesis in two patients. In all these cases, the intrahepatic lesions and the ascites resolved without any long-term complications.

In our case, the ultrasound showed normal liver architecture and the liver function tests remained normal throughout. Early recognition and subsequent appropriate management may have prevented liver damage in our case. Ultrasound examination of all cases of PN ascites identified before day 4 of UVC use in the literature, including ours, did not show any hepatic lesions indicating the possible association between the duration of catheter use and development of hepatic necrosis.⁸⁻⁹ The mechanism leading to the hepatic necrosis is unclear. It is thought to be due to the nature of the PN formulations (hypertonic and alkaline with a high osmolality), which come in contact with the hepatic parenchyma at the tip of the UVC leading to tissue necrosis and subsequent erosion.⁷⁻⁸ The longer duration of exposure of the PN may probably lead to hepatic necrosis.

The treatment of PN ascites from hepatic erosion is primarily by the removal of the UVC with or without paracentesis as clinically indicated. In all reported cases, the ascites and other related complications including hypotension, focal liver parenchymal damage and ARF due to hypovolemia, all resolved on prompt identification and removal of the UVC.⁷⁻¹⁰⁻¹² In our case, both the ascites and ARF resolved 3 days after removal of the UVC.

In conclusion, it is important to remember that unexplained ascites with ARF or hypotension may be a complication of a malpositioned UVC. Early diagnosis and management of this rare complication is essential for a favourable outcome.

Learning points

- ▶ UVC tips must be optimally placed at the inferior vena cava/right atrial junction just above the diaphragm, between the seventh and ninth thoracic vertebrae as seen on a plain radiograph.
- ▶ Ascites, though rare, is a serious complication of using UVCs.
- ▶ Successful outcome of the infants with PN ascites depend on early recognition and appropriate management. Neonatal staff using central lines in routine clinical practice must be made aware of this rare complication.

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Competing interests None.

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