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Parenteral Nutrition Ascites: Calamity from an Umbilical Vein Cannula

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Abstract

Umbilical vein catheterization is considered a critical requirement in preterm neonates for the administration of fluids and parenteral nutrition. However, inadvertent migration and malposition are known to cause complications that are often life-threatening. We describe a neonate with parenteral nutrition-associated lipid ascites owing to extravasation from an umbilical vein cannula. Fatality was averted due to prompt recognition and paracentesis that was therapeutic and confirmed the diagnosis as well.

Keywords
- umbilical vein cannula
- parenteral nutrition
- ascites

Introduction

Neonatal intensive care practices have indeed improved survival and outcomes. One of the downsides comes from need for invasive procedures. Umbilical vein cannulation (UVC), due to its ease of insertion and maintenance, is one of the commonest procedures conducted on preterm infants. Many adverse events have been described; therefore, the physician needs to be constantly on the lookout for complications. These venous lines are known to migrate even after the position has been initially confirmed by radiology and ultrasound.[1] We describe a life-threatening peritoneal leak from a displaced cannula. It was recognized and corrected promptly.

Case Report

Baby T, one of twin, was delivered at 29 weeks gestation with birth weight of 1250 g. Early rescue surfactant was administered for respiratory distress syndrome, and she was extubated to continuous positive airway pressure. A 5 Fr UVC was inserted soon after birth to provide parenteral nutrition (PN) as per unit policy. Shukla formula was used to calculate length of insertion at 6.5 cm.[2] Position was confirmed by radiography at body of T8 vertebra, at the level of diaphragm (►**Fig. 1**). Recurrent apnea warranted reintubation on day 3 of life. On day 4, perfusion concerns were noted that were corroborated with metabolic acidosis on blood gas measurements. She developed rapidly progressive abdominal distension, oliguria, hyponatremia, and hypotension. Late onset sepsis and necrotizing enterocolitis were suspected, antibiotics were upgraded, and circulation was supported by fluid bolus followed by inotropes. Worsening was associated with tense abdominal distension. Abdominal X-ray showed migrated UVC tip and ascites. Fluids were adjusted for anuria and deranged kidney function. Portable ultrasound showed large ascites, and scattered nonuniform hyperechoic texture in hepatic segments III, IV. UVC tip was visualized in left branch of portal vein with thrombosis (no flow on color Doppler) and was removed. The hepatic veins and inferior vena cava showed normal flow. In view of tense ascites, peritoneal tap was done, and 150 mL of milky ascitic fluid was drained. Biochemical analysis of the obtained milky intraperitoneal fluid revealed glucose (188 mg/dL) and triglycerides (1,688 mg/dL). The infant was diagnosed as having intraperitoneal extravasation of PN infusate from UVC. Within hours of therapeutic tap, the clinical status on all fronts improved dramatically, and supports could be weaned off rapidly. Full enteral feeds were achieved by day 13 of life. The infant was discharged home at 35 weeks postmenstrual age. This twin has mild development delay and early signs of spastic diplegia.

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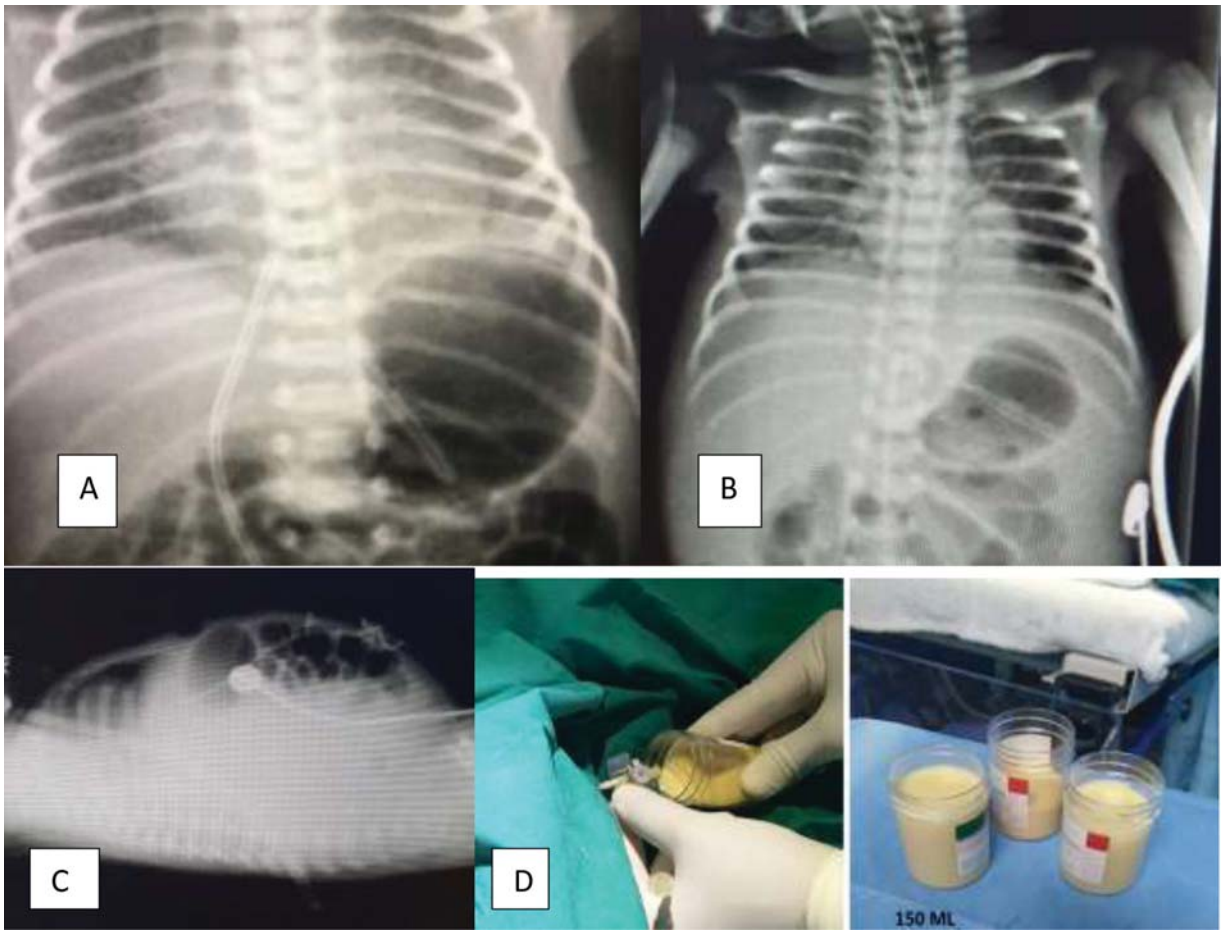

Fig. 1 (**A**) Umbilical vein cannula tip seen just above the diaphragm, at the level of body of T8 vertebra. (**B**) Anteroposterior view showing migration from previous position (in ►**Fig. 1**) to tip lying within hepatic shadow and ascites. (**C**) Lateral view showing ascites with bowel pushed anteriorly. (**D**) Therapeutic paracentesis—milky (lipid; 150 mL) ascites noted was drained.

Discussion

We describe a potentially fatal complication of UVC, averted due to prompt recognition and correction. UVC in neonates is most often easily and seamlessly inserted even by less experienced personnel. Correct depth of insertion and method of confirmation of placement are subjects of much controversy. Usually clinical methods used to estimate insertion lengths are based on birth weight or shoulder-umbilical length.[2] However, the commonly used formulae are not reliable in preterm infants. Incorrect tip placements can have devastating results.[3] Shrinking of Wharton jelly with time, movement of lungs and diaphragm, dynamics of blood flow, inadvertent movement with each manipulation for hub access can all lead to catheter migration—both upward and downward.[4] Ability of X-ray to correctly assess position of tip has a low sensitivity (around 60%) as compared with ultrasound with more than 90%.[5] Ultrasound conducted by trained neonatologists seems to correlate well with scans done by cardiologists with accuracy rate as high as 0.81 on receiver operating characteristic curve.[6]

Hepatic injury from UVCs has been widely reported. These range from incidentally detected ultrasound findings of hypoechoic mass with an echogenic rim alone; to severe clinical deterioration (as in our case).[7] In a large series of 1,081 neonates, nine developed severe hepatic injury.[8] All had the UVC malpositioned within the liver circulation leading to vessel perforation by the catheter or hepatic lacerations. Abdominal distension/ ascites, shock, and renal hypoperfusion are due to subcapsular extravasations/hemorrhage that rupture into the peritoneal cavity. Measurement of intrabdominal pressures (IAP) would have been ideal to classify as abdominal compartment syndrome (ACS). ACS in children is defined as sustained elevation in IAP $>$10 mm Hg with associated new/ worsening organ dysfunction.[9] This is measured intravesically with a bladder catheter in situ that has been connected to a pressure transducer. One mL/kg of saline is instilled, 30 to 60 seconds is allowed for detrusor relaxation, and then the pressure is measured (after zeroing) at the end of expiration in supine position. In our case, due to the life-threatening emergency, the intensive care team sought to drain the fluid as soon as confirmed by ultrasound. Hence, actual measurements of intravesical pressure could not be done. Intravascular absorption of fluid from third spaces can result in severe hyponatremia. Therapeutic abdominal paracentesis and need for surgical interventions have been described as well.[10] Biochemical analysis of tapped fluids and comparison to parenteral infusates help differentiate congenital chylous effusions from iatrogenic injuries. In most situations, the UVC tip is found to

be low, below the diaphragm and within the liver shadow in the X-ray.[11]

The position of UVC needs to be reassessed periodically for migration. Prompt use of ultrasound helps determine hepatic injury and delineation of cause for deterioration related to UVC complications. UVC position within the hepatic shadow, or in vessels below the ductus venosus, seems to increase likelihood of problems.

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Conflict of Interests
None declared.

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