

Sterile hepatic abscess due to umbilical venous catheterization

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A preterm infant with isolated fetal ascites was admitted to the neonatal intensive care unit due to the appearance of respiratory distress at birth. An umbilical venous catheter (UVC) was inserted. Abdominal ultrasonography (US) showed localization of the catheter tip in the portal vein. It was removed and replaced with a newer one. UVC tip location was confirmed with X-ray. His condition had been improving until he worsened suddenly on the sixth day of life. US showed hepatic abscess and intraabdominal hemorrhage derived from the malpositioned UVC. A drainage catheter was inserted to the abscess and paracentesis was applied. Practitioners should be cautious about any signs of UVC complications, even if true localization of the catheter tip is proven at the first application. Furthermore, if it is difficult to decide whether the catheter tip is in the right location, confirmation with US can be considered.

Key words: umbilical vein catheterization, complication, hepatic abscess, newborn.

Since their first use for replacement transfusion in the treatment of erythroblastosis fetalis, umbilical venous catheters (UVCs) have been used commonly in neonatal intensive care units (NICUs) for drawing blood samples, measuring venous blood pressure, and administering fluids, nutrition and medications, blood products, and exchange therapies in sick neonates¹.

Several complications related with UVC, such as thrombosis, embolization, hemorrhage, abscess, arrhythmias, effusions, portal hypertension, renal vein perforation, necrotizing enterocolitis and perforation of the colon, sepsis, cardiac tamponade, and ischemic injury of the extremities, have been reported previously²⁻⁸.

Liver abscess is an uncommon but life-threatening event in the neonatal period³. If the UVC has been positioned improperly, the endothelium of the hepatic vessels can be damaged during infusion of parenteral nutrition or other hypertonic solutions. Abscess formation will be easier in the culture medium provided by the necrotic tissue and parenteral nutrition.

Herein, we report a preterm infant with isolated fetal ascites who developed liver abscess and intraabdominal hemorrhage derived from a malpositioned UVC.

Case Report

A preterm male infant, with birthweight of 3180 g, was born at 33 weeks gestation by repeat cesarean section due to premature labor to a 30-year-old mother, gravida 5, para 5. Apgar scores were 6 and 6 at 1 and 5 minutes, respectively. Prenatal ultrasonography (US) showed an accumulation of fluid in the peritoneal cavity, but not in other body cavities. The mother's parvovirus polymerase chain reaction (PCR) and cytomegalovirus antibodies were negative.

The infant was placed on nasal continuous positive airway pressure for respiratory distress. Hours later, he was intubated because of deterioration in his respiratory status. A UVC was inserted to infuse parenteral nutrition and for blood sampling. The catheter tip was seen just below the diaphragm. Complete blood cell count, peripheral blood smear, C-reactive protein, and blood culture were performed for sepsis work-up, and empiric ampicillin and gentamicin treatments were started according to our NICU policy. Yellow-colored clear ascitic fluid (100 ml) was drained from the abdominal cavity. Many lymphocytes were seen on the microscopic examination. There was no bacterial growth in the fluid culture. Abdominal US was performed to evaluate the

ascites. No anomaly in the gastrointestinal or genitourinary system accompanying the fetal ascites was seen, but the UVC tip was observed to be in the right portal vein with thrombosis. The catheter was removed and replaced with a newer one. At approximately two days of life, enteral feeding was started, and parenteral nutrition was diminished daily. The patient was extubated on the fourth day of life. He was stable without any respiratory or oxygen support.

On the sixth day of life, the infant developed cutis marmorata, hypothermia and acute abdominal distension. Serohemorrhagic fatty fluid-like parenteral nutrition was obtained from the UVC while trying to get a blood sample for the septic work-up. The complete blood cell count showed a white blood cell count of $15.7 \times 10^3 / \mu\text{L}$, with a differential of 40% neutrophils, 14% bands, 10% monocytes, and 52% lymphocytes; hemoglobin was 12.3 g/dl, hematocrit 38.1%, and platelet count $79 \times 10^3 / \mu\text{L}$. Alanine aminotransferase (ALT) level was 810 IU/ml and aspartate aminotransferase (AST) level was 1964 IU/ml. Serum electrolytes were normal. The patient's symptoms were attenuated after acute isotonic saline loading (20 ml/kg) and appropriate fluid replacement. Erythrocyte suspension of 20 ml/kg was transfused because of low hemoglobin level.

Emergency abdominal US was performed, which revealed hyperechoic, thick-walled, 50x30x35 mm collection with air-fluid level in the liver and a massive amount of intraabdominal free-fluid with internal echo patterns (Fig. 1). Antibiotic therapy was changed to vancomycin, meropenem, amikacin, and fluconazole. A drainage catheter

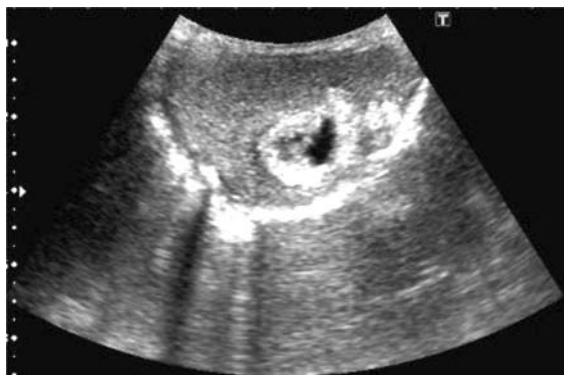


Fig. 2. Large hepatic abscess formation.



Fig. 1. Involved hepatic abscess formation one month after discharge.

was inserted to the hepatic abscess by the interventional radiologist. Paracentesis was also applied contemporaneously, and 130 ml of hemorrhagic and chylous fluid was drained. Follow-up abdominal US showed minimal fluid collection in the liver, but portal vein thrombosis remained (Fig. 2). The drainage catheter was removed. ALT and AST levels were normal on the 10th day of life with no additional intervention. No bacterial or fungal growth was determined in the hepatic abscess or paracentesis fluid.

The newborn was discharged on the 16th day of life after receiving 10 days of antibiotic therapy. He was fed with full enteral nutrition. Doppler US performed one month after discharge showed spontaneously resolved portal vein thrombosis. Neurologic evaluation was normal during his routine follow-up visits thereafter.

Discussion

The use of UVCs in infants requiring intensive care has led to an increased incidence of adverse effects. Hepatic fluid collection has been reported as one of the rare but serious complications of UVC in a small number of case reports⁹. Osmolality of the infused solution, tip of the catheter, and recently, the catheter type used have been suggested as the etiologic factors of liver fluid collection in infants with UVC⁹.

Hepatic abscess due to UVC malposition is seen in infants, with high mortality rates varying from 50%-75%^{3,10}. The UVC malposition rate at the first insertion attempt was reported as

nearly 51%¹¹. Different methods, such as X-ray, US, echocardiography, and electrocardiography, have been mentioned in the literature for verifying the correct positioning of the UVC. Abdominal radiography remains the most frequently used technique. The catheter must be placed in a high position, which is defined as its tip in the inferior vena cava, just before the entrance to the right atrium. In this position, the tip should be seen above the diaphragm in the abdominal radiograph, at the level of the eighth and ninth thoracic vertebrae^{4,12}. After entering the abdomen, the umbilical vein divides into different branches, and while one of them, the ductus venosus, directly joins and carries blood to the inferior vena cava, others join the portal vein and deliver blood to the liver. There is increased risk of malposition to the portal vein when the catheter tip is placed in the subdiaphragmatic region. Because of difficulties in deciding the correct position with X-ray, US evaluation seems to be a more valuable technique to diminish complications with high mortality.

We had no US confirmation of the second catheter position in our case, but it was positioned correctly in the X-ray evaluation. We believe the catheter tip was displaced to the portal vein during interventions like blood drawing and paracentesis, because the patient's condition had been improving until the sixth day, when it worsened suddenly. In our patient, increased intraabdominal pressure due to the fetal ascites might also have been a factor in the dislocation of the UVC. Thus, practitioners should be aware of UVC dislocation if there is a condition that elevates intraabdominal pressure, like ascites, tumors or bowel distension.

Improvements such as closer monitoring of infants, more rapid diagnosis with better imaging techniques, more effective antimicrobial therapy with extended spectrum, and non-invasive drainage methods with diminished complications have been observed in the management of hepatic abscess in recent years. Tan et al.¹⁰ reported mortality rates of 50% in their case series of six preterm infants with liver abscess; five of them were under 27 weeks of gestational age and had UVC. Extremely preterm infants (EPIs) need UVC more than term infants hospitalized for other reasons. As EPIs have comorbid diseases that

also increase mortality, neonatal practitioners should be more careful while following EPIs with UVC.

Even if true localization of the catheter tip is proven at the first application, caregivers should be cautious about any signs of UVC complications during follow-up in the NICU. The UVC should be removed as soon as possible when there is no need for catheter in order to prevent the related complications.

REFERENCES

1. Green C, Yohannan MD. Umbilical arterial and venous catheters: placement, use, and complications. *Neonatal Netw* 1998; 17: 23-28.
2. Al Nemri AM, Ignacio LC, Al Zamil FA, Al Jarallah AS. Rare but fatal complication of umbilical venous catheterization. *Congenit Heart Dis* 2006; 1: 180-183.
3. Lam HS, Li AM, Chu WC, Yeung CK, Fok TF, Ng PC. Mal-positioned umbilical venous catheter causing liver abscess in a preterm infant. *Biol Neonate* 2005; 88: 54-56.
4. Yigiter M, Arda IS, Hicsonmez A. Hepatic laceration because of malpositioning of the umbilical vein catheter: case report and literature review. *J Pediatr Surg* 2008; 43: E39-41.
5. Ancora G, Soffritti S, Faldella G. Diffuse and severe ischemic injury of the extremities: a complication of umbilical vein catheterization. *Am J Perinatol* 2006; 23: 341-344.
6. Monteiro AJ, Canale LS, Barbosa R, Meier M. Cardiac tamponade caused by central venous catheter in two newborns. *Rev Bras Cir Cardiovasc* 2008; 23: 422-424.
7. Nadroo AM, al-Sowailem AM. Extravasation of parenteral alimentation fluid into the renal pelvis--a complication of central venous catheter in a neonate. *J Perinatol* 2001; 21: 465-466.
8. Hermansen MC, Hermansen MG. Intravascular catheter complications in the neonatal intensive care unit. *Clin Perinatol* 2005; 32: 141-156, vii.
9. Mahajan V, Rahman A, Tarawneh A, Sant'anna GM. Liver fluid collection in neonates and its association with the use of a specific umbilical vein catheter: report of five cases. *Paediatr Child Health* 2011; 16: 13-15.
10. Tan NW, Sriram B, Tan-Kendrick AP, Rajadurai VS. Neonatal hepatic abscess in preterm infants: a rare entity? *Ann Acad Med Singapore* 2005; 34: 558-564.
11. Haase R, Hein M, Thale V, Vilser C, Merkel N. Umbilical venous catheters - analysis of malpositioning over a 10-year period. *Z Geburtshilfe Neonatol* 2011; 215: 18-22.
12. Dettaille T, Pirotte T, Veyckemans F. Vascular access in the neonate. *Best Pract Res Clin Anaesthesiol* 2010; 24: 403-418.