Hepatic laceration as a life-threatening complication of umbilical venous catheterization

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Umbilical venous catheterization is an intravenous infusion route for maintenance fluids, medications, blood products, and parenteral nutrition in preterm neonates. However, this procedure may be associated with several complications, such as infection, thrombosis, vessel perforation, and cardiac and hepatic injuries. Hepatic laceration is a rare but life-threatening complication of umbilical venous catheterization that is a result of direct injury through the liver parenchyma. Here, we present a preterm newborn with hepatic laceration as a rare and serious complication of umbilical venous catheterization.

Key words: hepatic laceration, neonate, umbilical vein catheter.

Umbilical venous catheterization has become a standard procedure in neonatal intensive care units after the significant increase in the number of preterm newborns[1]. The umbilical vein catheter (UVC) allows rapid and reliable vascular access for administration of fluids, parenteral nutrition, intravenous medications, and blood sampling. However, there are several mild to serious complications of umbilical venous catheterization, including infection, thrombosis, catheter misplacement, vascular injury, pericardial effusion, cardiac tamponade, and hepatic injury[1-7]. Complications related to the liver are rare but may be life-threatening, particularly in small preterm newborns[3,5-8]. Here, we present a preterm newborn with hepatic laceration and subsequent extravasation of total parenteral nutrition (TPN) secondary to malpositioned UVC through the liver parenchyma.

Case Report

A male infant was born at 28 weeks gestational age by vaginal delivery after the spontaneous onset of labor because of maternal chorioamnionitis. He was referred to our neonatal intensive care unit at 3 hours of age because of extreme prematurity and respiratory distress syndrome. He weighed 950 g. The infant was extubated to nasal continuous positive airway pressure after receiving porcine surfactant (Curosurf®). Prophylactic antibiotics were initiated because of maternal chorioamnionitis. Umbilical arterial and venous catheters were inserted on day 1 of life. The position of the UVC tip was determined to be subdiaphragmatic by a plain abdominal radiograph. On the 1st day of life, TPN was infused through a 5F UVC. Because of progressive respiratory distress, he was intubated on the 3rd day of life. On day 5, the patient suddenly deteriorated. Significant abdominal distension and hepatic enlargement were detected on physical examination. He developed profound apnea and bradycardia associated with marked hypotension and significant drops in hemoglobin level (12 g/dl to 6 g/dl). An abdominal radiography showed distended, nearly gasless abdomen, and the tip of the UVC was seen inside the liver shadow (Fig. 1). An urgent abdominal ultrasonography revealed massive echogenic free fluid within the peritoneal cavity and hyperechoic heterogeneous lesion measuring 2 x 1.2 x 1 cm involving segments IV and VIII of the liver, consistent with hematoma. The main portal vein demonstrated normal flow. Contrast study through the UVC showed extravasation of contrast within the liver around the UVC tip (Fig. 2). The UVC was removed. Non-

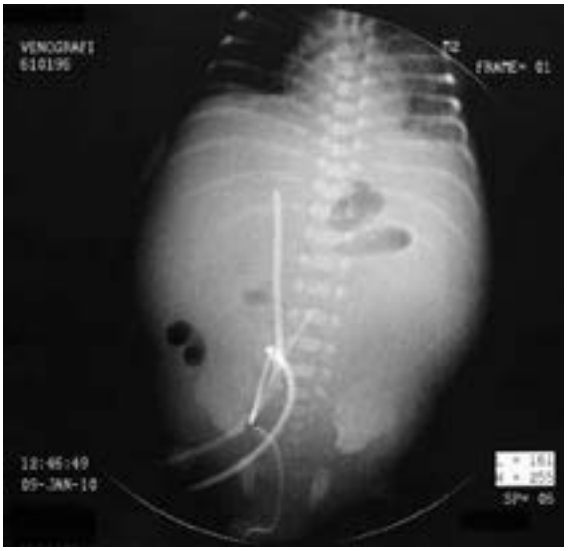

Fig. 1. Plain abdominal radiography shows a distended, nearly gasless abdomen on day 5 of life. The tip of the UVC is seen inside the liver shadow.

operative management was applied including aggressive fluid resuscitation and high-dose inotropic support for refractory hypotension, packed red blood cells, fresh frozen plasma, and platelet concentrates to correct severe disseminated intravascular coagulopathy (DIC) that developed secondary to massive bleeding. Unfortunately, the patient continued to deteriorate despite potent antibiotics and vigorous supportive treatment. He died on day 21 of life due to resistant metabolic acidosis, non-correctable coagulopathy, and overwhelming nosocomial sepsis.

Discussion

Catheterization of the umbilical vein was first described in 1947 for use in exchange transfusion as a treatment of severe indirect hyperbilirubinemia because of erythroblastosis fetalis[1]. Since that time, UVC placement has become the most common procedure in neonatal intensive care units during the first hours of life. The rate of catheterization of the umbilical vein varies from 9% in newborns with birth weight over 2000 g to over 50% in newborns with birth weight below 1000 g[9].

The ideal position of the UVC tip should be at the junction of the inferior vena cava (IVC) and the right atrium (outside the heart in IVC) or just above the diaphragm[1,2,5,6]. This correlates with the eighth and ninth thoracic vertebrae. An abdominal X-ray or ultrasound should be taken to confirm the catheter tip position following catheter placement. Optimal placement of the UVC may not always be possible, and suboptimal positioning is often clinically accepted if it is to be used only for intermittent access or for maintenance fluid administration[1]. Even a properly placed catheter may be accidentally withdrawn and the tip of the catheter may be dislocated into the liver shadow. In a review of umbilical vein catheterizations, Seguin et al.[9] reported a 92% initial success rate for UVC placement, but only 68% of catheters were placed to be above the diaphragm. Greenberg et al.[10] compared the UVC tip position with radiographs and ultrasound in 79 patients. They showed that two-thirds of catheters located at T10 on radiographs were found to be within the liver on ultrasound.

While umbilical venous catheterization offers considerable benefits for newborns, it also carries significant potential complications. Complications that are specific to UVCs are commonly the result of malposition of the catheter. Some of these complications are relatively minor, whereas complications related to the liver and heart are potentially life-threatening[2]. The catheter tip placed high in the right atrium can result in arrhythmias or perforation, with subsequent pericardial effusion

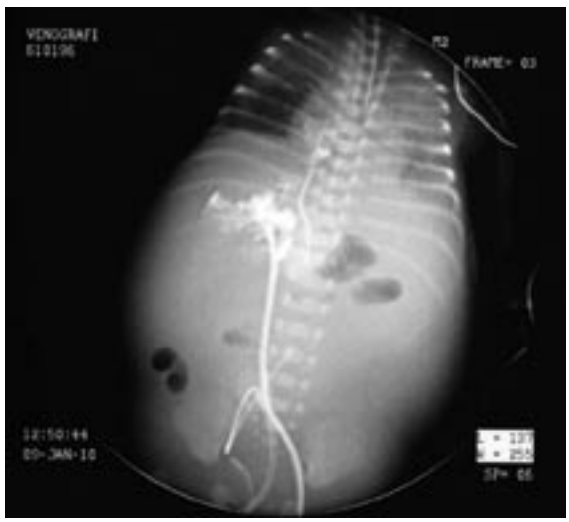

Fig. 2. Contrast study through the UVC shows intrahepatic extravasation of contrast around the tip of the UVC.

and tamponade[1-4]. The subdiaphragmatic positioning of the UVC tip appears to be an important factor contributing to the development of complications related to the liver[4,5-8,11,12]. Coley et al.[5] reported that catheter tips were at or below T10 at initial placement in seven of eight patients with liver erosion by UVCs. Lim-Dunham et al.[6] identified the characteristic sonographic findings of patients with hepatic erosion by UVCs. They showed that catheter tips in all patients were located below the hemidiaphragm and superimposed over the liver. In our patient, the UVC tip was between T10 and T11, projecting over the liver.

Hepatic injuries are more severe than the other complications, with a high risk for morbidity and mortality. Inadvertent placement of the UVC tip in the liver can result in portal vein thrombosis and subsequent portal hypertension, subcapsular hematoma, hepatic necrosis, hepatic laceration, hepatic abscess, and hepatic encystment formation[1-3,5-8,11].

The infusion of hypertonic solutions such as TPN or high-concentration dextrose solution through a suboptimally placed UVC may produce local hepatic parenchymal injury and necrosis[2,3,5,6]. If the damage is severe enough to disrupt the liver capsule, free intraperitoneal leak of TPN may occur. Osmotic injury over the myocardium, which results from pericardial effusion and cardiac tamponade in cases of administration of hypertonic TPN solution, was reported by Traen et al.[4] Lam et al.[8] described a case with UVC-related liver abscess due to hypertonic parenteral solution and catheter tip malposition. Our patient was also receiving hypertonic TPN through the UVC. The duration of UVC use may also contribute to the development of complications related to the liver. Seguin et al.[9] noted that the mean duration of UVC use in newborns was 4.4 days, with longer use up to 5.5 days in newborns weighing <1500 g. The mean durations of UVC use before clinical presentation were documented as 8.9 days by Coley et al.[5] and 6.75 days by Lim-Dunham et al.[6]. In our patient, the duration of UVC use was 5 days before detection of hepatic laceration. There were several potential factors leading to the development of hepatic parenchymal injury and subsequent extravasation of TPN in our patient. The most important risk factor was subdiaphragmatic positioning of the UVC. Hypertonic parenteral nutrition solution and the long duration of catheter use were the other significant contributing factors.

To the best of our knowledge, seven reports of hepatic injury by UVCs have been published in the English literature to date[4,5-9,11]. All neonates had abdominal distension, and in all of them, the tips of the UVCs were located below the diaphragm and superimposed over the liver on plain radiographs. Our patient also had significant abdominal distension and right upper quadrant fullness. Furthermore, our patient had hypotension, significant drops in hemoglobin concentration and extravasation of TPN. These findings showed evidence of hepatic parenchymal injury close to the UVC tip. Removal of the catheter resulted in resolution of the ascites.

In conclusion, hepatic laceration by UVC is a life-threatening but preventable complication of the subdiaphragmatic-positioned UVC. Initial and subsequent positions of the UVC must be monitored radiologically to prevent catheter-related complications. The UVC should also be used for as short a time period as possible if shown to be suboptimally placed. Hepatic parenchymal injury should be kept in mind in any patient with UVC who suddenly develops abdominal distension and shock-like symptoms. Early diagnosis and removal of the catheter may be life-saving in these patients.

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