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Umbilical venous catheterization gone wrong: Hepatic complications

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Abstract

Hepatic complications of malposition of umbilical venous catheter (UVC) are uncommon and occur due to extravasation of hypertonic fluids and the blood products in the liver tissue. Various hepatic complications include thrombosis of hepatic vessels, hepatic necrosis, hepatic fluid collections, and hematoma, with the intraparenchymal liver lesions seen along the course of ductus venosus. Radiologists must be aware of these complications and their imaging findings, as the timely recognition and immediate management can prevent the fatal outcome. Here, we present a rare case of intraparenchymal liver lesions associated with malposition of UVC in a preterm baby.

Key words: Extravasation; hepatic necrosis; total parenteral nutrition; umbilical venous catheterization

Introduction

Umbilical venous catheterization (UVC) is commonly done for gaining an access to the central line in neonates for administering antibiotics, total parenteral nutrition (TPN), and for transfusion of blood products. UVC obviates the pain and the complications associated with repeated venous punctures. However, the placement of UVC requires experience. The position of the tip should be monitored, as its malposition can cause various complications with the reported rates ranging from 20% to 37%.[1] Complications related to the liver have a high rate of morbidity and mortality, particularly in small premature newborns.[2] Although post UVC hepatic complications like hepatic abscess, laceration, and hematoma have been described previously, with only <100 cases reported worldwide,[3] our case is unique as multiple hepatic complications including hepatic necrosis, hematoma, and TPN fat accumulations were seen together, which has not described previously to the best of our knowledge.

Case Report

A late preterm male infant born at 36 weeks was referred to our hospital on day 15 of life. The infant had a history of respiratory distress syndrome soon after birth, for which surfactant was administered intratracheally. The infant was being treated for multiple conditions since birth, and these included neonatal respiratory distress, left-sided pneumothorax, culture-positive sepsis, thrombocytopenia, refractory anemia, hypokalemia with metabolic acidosis, deranged kidney functions, and necrotizing enterocolitis. The child had been intubated for management of respiratory distress; intravenous catheter and UVC were inserted for infusion of TPN, antibiotics, and transfusion

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of packed RBCs and platelets for management of anemia and thrombocytopenia. On day 13, the baby showed clinical deterioration, abdominal distension, worsening of thrombocytopenia, and raised levels of C-reactive protein. UVC had been removed and the child was referred to our hospital for further management. On examination, the baby showed abdominal distension with hepatomegaly. USG abdomen performed using high-frequency linear transducer (Envisor color Doppler ultrasound; Philips, Bothel, USA) revealed multiple heteroechoic lesions in the left lobe of liver in segment IVA adjacent to the left portal vein [Figure 1A]. Another large heteroechoic lesion with hyperechoic component in the center was seen in IVB segment and posterior segments of the right lobe of liver with a large exophytic component [Figure 1B]. The wall of the left portal vein also appeared echogenic and edematous; however, normal color flow was seen. On the basis of USG, a possibility of multiple hepatic abscesses/hematoma in different stages was considered. Subsequently, contrast-enhanced computed tomography (CECT) abdomen was done (16-slice Brilliance Scanner, Philips, Haifa, Israel) for the characterization of lesions, which revealed multiple non-enhancing mixed density lesions in the left lobe of liver with areas of blood and fat component at the periphery, with the largest lesion measuring 8.5 cm × 3.3 cm in IVB and the posterior segments of the right lobe having an exophytic component [Figure 2A and B]. Few of these lesions seemed to be communicating with each other. As there was history of umbilical vein catheterization, previous abdominal radiograph was reviewed which showed the abnormal position of the catheter at D10–D11 level in the right hypochondrium with the tip likely in hepatic parenchyma [Figure 3]. So, based on the imaging findings, these focal lesions appeared to have occurred as a result of delivery of TPN fluids and blood products into the hepatic parenchyma with consequent hepatic necrosis. USG guided fine needle aspiration done from the lesions confirmed the presence of blood and TPN fat. The child was treated with low-molecular-weight heparin for 4 weeks for the condition. Follow-up USG and limited CECT sections of the upper abdomen revealed significant reduction in the size of the lesions and resolution of hematoma. The lesions in the left lobe appeared hyperechoic with fine calcification present at places [Figure 4]. The child was clinically stable and was discharged in the 5th week of life, and follow-up USG was advised.

Discussion

UVC is a common procedure which is done in neonates in the first few days of life and is used as vascular access for administering fluids, medication, parenteral nutrition, and blood products.[4]

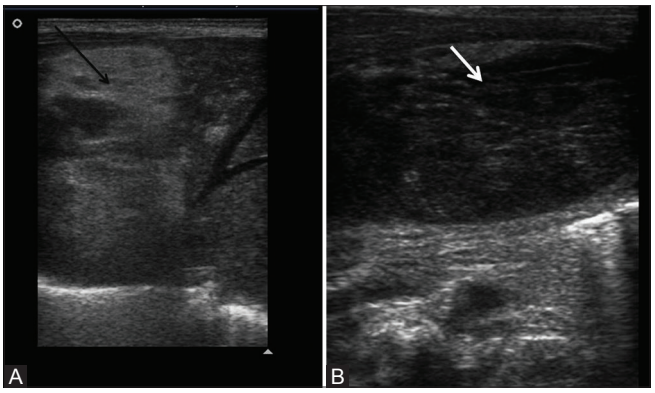

Figure 1 (A and B): (A and B) USG abdomen shows multiple heteroechoic predominantly hyperechoic lesions in superior segment (black arrow) and predominantly hypoechoic in inferior segment of left lobe and posterior segments of right lobe with large exophytic component (white arrow)

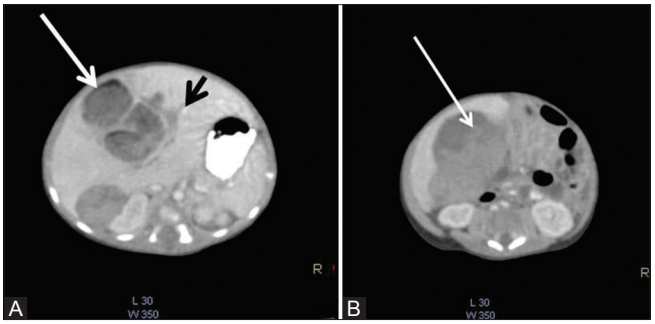

Figure 2 (A and B): (A and B) CECT abdomen at the level of portal vein bifurcation showing multiple mixed density intraparenchymal hepatic lesions, Lesions show mixed areas of hemorrhage and fat density at the periphery (white arrow in A). Note is made of oedema around left portal vein and its branches (small black arrow in A). Similar large heterodense lesion with exophytic component is seen in segment IVB and posterior segments of right lobe shows fluid collection with areas of hemorrhage (white arrow in B)

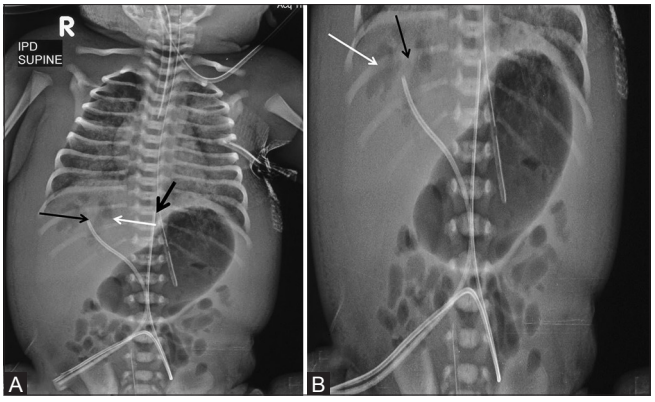

Figure 3 (A and B): (A) Chest and abdominal Radiograph showing the abnormal position of UVC with tip (black arrow) at D10-D11 level likely in hepatic parenchyma (white arrow). The gas medial to the catheter outlines the umbilical venous recess. Note is made of umbilical artery catheter with tip lying in midline at the level of D10 vertebra (small black arrow). Note is made of reticulonodular shadowing in bilateral lung. (B) Zoomed image shows the air in the branches of left portalvein (black arrow) and hepatic parenchyma (white arrow)

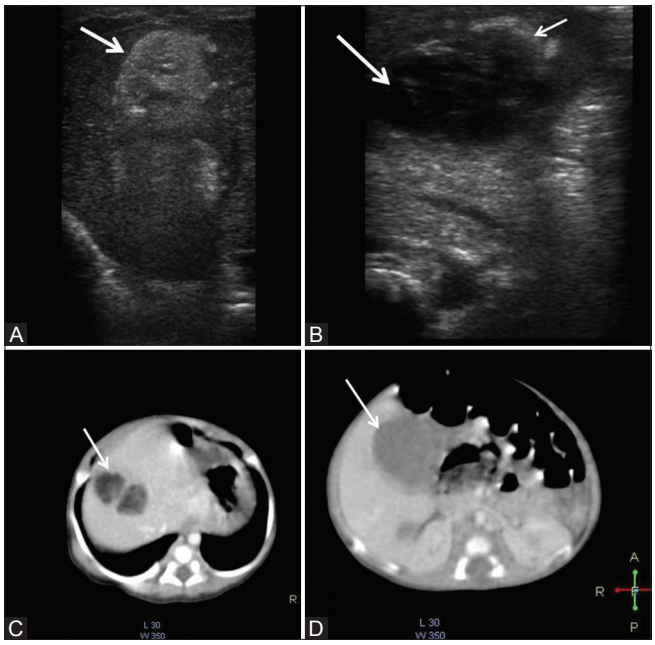

Figure 4 (A-D): Follow up USG and CECT after 3 weeks show marked regression in the size of the lesions. The lesions in left lobe in superior segment (arrow in A) are more echogenic. Large exophytic lesion with areas of hemorrhage (arrow in B) also shows significant reduction in the size and is more hypoechoic consistent with resolving hematoma. Peripheral foci of calcification was also seen (small arrow in B). Similar changes were appreciated on CT images with decrease in the size and the hematoma is resolving and appear hypodense (C and D)

The fetal umbilical vein communicates with the portal vein through its branches that continues as ductus venosus which further joins the middle or left hepatic vein. Ideally, the catheter should be placed in supradiaphragmatic inferior vena cava (IVC) just below the atriovenous junction which normally lies at D8-D9 level.[5,6] The catheter tip can be misplaced anywhere along the course through which it traverses and can be positioned in the umbilical vein with or without forming a loop, in the right or left portal vein, in the hepatic parenchyma, in the splenic vein, or can traverse superiorly into intracardiac location. Abdominal radiographs are used to look for the position of UVC; however, due to the concern for radiation and as the atriocaval location can vary in relation to bony landmarks, real-time USG is used to localize the tip. The chances of misplacement of the catheter are reduced if the catheter is placed under USG guidance. Small amount of saline can be injected to confirm the position. The factors affecting the catheter malposition and its resultant complication depend on the insertion technique, size and composition of the catheter, number of manipulations, and contents of parenteral infusion. During insertion, if the catheter moves toward liver through the portal system, it should be removed immediately as it can cause thromboembolism and portal hypertension. After placement, the catheter should be secured on the skin and the position and patency of the umbilical vein should be assessed by saline flushing or by drawing blood. Even if the catheter is placed in proper position, it can be withdrawn accidentally and can be misplaced into the liver parenchyma and cause hepatic injury.[2]

Complications associated with UVC include thromboembolism, vasospasm, vascular injury, hemorrhage, infection, hepatic, renal, gastrointestinal damage, ascites, hydrothorax, and various cardiac complications like pericardial tamponade and erosion of atrial or ventricular wall. UVC can rupture or break and there can be migration of fragments.[7,8]

Apart from portal vein thrombosis and portal hypertension, various other hepatic complications include encysted fluid collections, subcapsular hematoma, hepatic necrosis, abscess, laceration, and biliary venous fistula. Hepatic fluid collection, necrosis, and abscess are its rare complications. Fluids which are infused in TPN are hypertonic and alkaline in nature and can cause hepatic necrosis, collection, and abscess formation. Hepatic parenchymal injury occurs more commonly either due to chemical irritation or, to a lesser extent, by compression of liver parenchyma by extravasated fluid.[7] Intraparenchymal hematoma is either due to vascular injury or erosion or laceration of the hepatic parenchyma.[9] History of UVC insertion and oozing of parenteral fluids through the umbilical vein after the removal of UVC and appearance of hepatic lesions should raise the possibility of hepatic complications due to malposition of UVC insertion. In our case, there were multiple mixed density lesions with presence of fluid, fat, and blood, which was probably due to delivery of transfused blood and TPN fluid and fat into the hepatic parenchyma due to liver necrosis. Hepatic complications associated with UVC require an emergent treatment, making it imperative for the radiologists to be aware of the proper position of UV catheter and the imaging appearance of these hepatic lesions due to catheter malposition.

Imaging includes abdominal radiograph to look for the position of UVC, USG, and CT scan. Hepatic lesions lie in the course of umbilical vein and ductus venosus. These lesions show varied appearance and typically are well-defined hypoechoic lesions with cystic component and peripheral hyperechoic rim. Hyperechoic rim is due to the encystment of TPN fat periphery and hypoechoic center is due to aqueous material. On follow-up USG, these lesions appear hyperechoic and few may show calcification as was seen in our case. Hematomas appear hyperechoic in the earlier stage and hypoechoic in the later stage with or without septations. When there is disruption of liver capsule, there is extravasation and peritoneal spillage of parenteral nutrients. Ascites is simple in the earlier stage; septations and debris appear later. Intraperitoneal hemorrhage is also one of the complication of malposition of UVC due to erosion of hepatic parenchyma and rupture of liver capsule.

Differential diagnosis includes liver abscess due to generalized infection, hamartoma, and hepatoblastoma. Hepatoblastomas appear as sharply demarcated mass and are slightly hypoattenuating than the adjacent liver parenchyma on non-contrast CT images. Speckled or amorphous calcification is seen in more than 50% of the lesions. On dynamic images, these masses show arterial enhancement and significant washout in portal venous phase and significantly high alpha fetoprotein (AFP) level.[10] In newborns, there is high level of AFP, but it decreases with time.[11] On USG, mesenchymal hamartoma appears as a well-defined multiloculated cystic mass with presence of echogenic septae, which is described as Swiss cheese appearance.[12] On CT, the mass appears hypodense with presence of septae and the solid component which enhances on post-contrast scan.[13] Liver abscesses are rarely seen in newborns and are seen only when there is generalized infection. Fever, rapidly increasing markers of infection, and abdominal manifestations with peculiar imaging characteristics favour the diagnosis of abscess.

This case highlights the hepatic complications related to malposition of UVC. Therefore, the radiologists must be aware of the proper position of UVC and varied imaging characteristics of these hepatic lesions, so that catheter could be repositioned as early as possible.

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Conflicts of interest

There are no conflicts of interest.

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