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CASE REPORT »»

Massive Liver Mass and Parenteral Nutrition Extravasation Secondary to Umbilical Venous Catheter Complication

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ABSTRACT

Umbilical vein catheters (UVC) are widely used in neonatal medicine. Serious complications from UVC placement are uncommon but do exist, including infection, thrombosis, arrhythmias, and hemorrhage. Although rare, hepatic complications, in particular, have been associated with significant morbidity and mortality. Correct positioning of the catheter prior to starting infusion of hyperosmolar solutions and early recognition of UVC-related complication are crucial in minimizing iatrogenic injury. We report the case of a neonate who was found at 10 days of age to have large pleural and peritoneal effusions and a massive fluid collection in the liver due to malposition of a UVC.

Key words:
Hepatic hematoma, intravenous lipids, liver mass, Neonate, total parenteral nutrition, umbilical venous catheter

INTRODUCTION

Umbilical vein cannulation was first utilized in 1947 for early venous access in the sick neonate. Serious complications, including vascular, hepatic, and cardiac injuries are uncommon but do exist. TPN extravasation and hepatic injury from UVC malposition are rare but potentially life-threatening complications.

CASE REPORT

The patient was born at 36 weeks gestation via urgent cesarean section secondary to maternal status epilepticus. Because of perinatal asphyxia, the infant was intubated and transported to our Neonatal Intensive Care Unit within a few hours after birth. On admission, the infant was hemodynamically stable. No anomalies or hepatosplenomegaly were detected. A UVC and umbilical artery catheter (UAC) were placed shortly after admission. An initial radiograph of the chest and the abdomen showed the catheter tip at the level of T9 near the midline, just below the diaphragm [Figure 1a]. Total parental nutrition (TPN) and intravenous lipids were initiated via the UVC.

On day of life (DOL) 10, the infant required increased ventilatory support. Examination showed abdominal distension. A chest X-ray revealed a right pleural effusion [Figure 1b]. A limited abdominal ultrasound revealed ascites. The abdominal viscera were not completely shown on this study. Pleural and peritoneal drains were placed, both of which produced a large amount of milky white fluid. Analysis revealed high triglyceride (TG) levels, 375 mg/dL in the pleural fluid and 1446 mg/dL in the peritoneal fluid (serum TG level was 174 mg/dL). Neither fluid had evidence of blood contamination. The UVC line was promptly removed. An attempt to aspirate from the UVC prior to its removal did not yield blood or other fluid.

One day after the removal of the UVC, the white blood cell count increased to 33,000/mm^3, and the ratio of immature to total neutrophils increased to 0.57. The patient was empirically treated with antimicrobial antibiotics for 7 days. Blood, peritoneal, and pleural cultures remained negative. On DOL 17, a follow up abdominal ultrasound revealed a complex hypoechoic lesion in the liver measuring 75 × 64 × 57 mm. A Doppler study showed normal hepatic vessels, with absence of Doppler flow in lesion. A CT scan [Figure 2] revealed a large lesion (80 × 72 × 38 mm) of heterogeneous intensity involving multiple hepatic segments. Three days later, given concern to exclude pre-existing tumor (i.e. mesenchymal hamartoma or hemangioendothelioma) and to track interval changes

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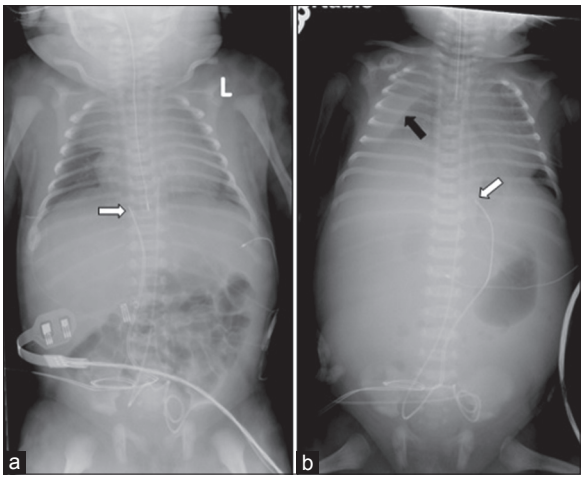

Figure 1: (a) Chest and abdominal AP radiographs at day 1 after UVC placement with tip at T9 near midline. (b) Radiographs with large right pleural effusion, ascites, and the UVC displaced to the left of the midline. White arrows point to UVC tip. Black arrow points to pleural effusion

with better soft tissue imaging, an MRI with contrast was obtained. This revealed a large, lobulated cystic lesion without significant post-contrast enhancement, consistent with a benign fluid collection most likely related to prior UVC, without features suggestive of tumor.

Sequential laboratory values are summarized in Table 1. Notably, the hematocrit dropped from 46% on DOL 2 to 30% on DOL 6. At the time, the drop was attributed to phlebotomy losses, but in retrospect, a contribution from possible internal bleeding into the liver parenchyma could not be ruled out. At 10 days, all liver function tests were elevated suggesting the presence of hepatocellular injury and cholestasis.

After a multidisciplinary evaluation, a conservative approach was adopted. The patient was monitored with hepatic function studies and serial liver ultrasounds. The peritoneal drain was discontinued at DOL 24. The patient was discharged home at 48 days, receiving full enteral feeds. Ultrasound prior to discharge estimated the liver lesion to be 40 × 61 × 46 mm. A follow up ultrasound at 8 months of age showed the lesion had decreased in size to about 40 × 30 × 16 mm with a patent portal vein.

DISCUSSION

The umbilical vein gives off branches into the liver and becomes the ductus venosus, which feeds into the supradiaphragmatic inferior vena cava (IVC). When optimally placed, the UVC tip will be at the junction of the IVC and right atrium, above the diaphragm, usually at T8 to T9.[1]

In this case, malpositioning of the UVC within the liver and subsequent infusion of hypertonic TPN likely led to vessel

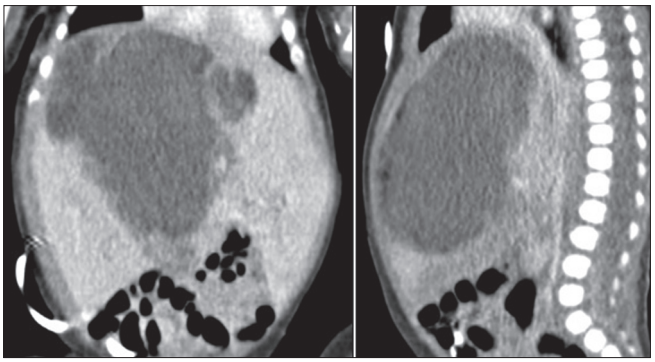

Figure 2: Abdominal CT scan of abdomen with intravenous contrast at 17 days of age revealing a large cystic liver lesion with irregular borders and small hypoechoic regions representing air. The lesion measures 72 × 80 × 38 mm

Table 1: Laboratory blood test trends

Age Days	Alb g/dL	AST U/L	ALT U/L	Alk Phos U/L	TB mg/dL	DB mg/dL	GGT U/L	AFP ng/ml	INR	Hct
2	2.4	119	36	119	2.4	0.5	-	-	1.6	46
6	2.4	99	50	-	1.4	0.5	-	-	-	30
13	3.0	253	241	108	2.7	1.9	-	-	-	34
19	2.7	43	47	113	2.9	2.1	192	5,260	1.2	37
35	-	98	117	362	7.9	5.5	645	24,400	-	36
48	3.8	69	59	407	2.3	1.1	512	17,900	-	-
118	4.1	41	50	282	0.3	-	69	261	-	35

Alb – Albumin; AFP – Alpha fetoprotein; Alk Phos – Alkaline phosphatase; ALT – Alanine aminotransferase; AST – Aanspiptidase; INR – International normalized ratio; Hct – Hematocrit; TB – Total bilirubin

erosion. Subsequently, the intravenous fluids administered via the UVC then collected and created an enlarging cystic space within the liver, and eventually escaped into the peritoneum and then entered the pleural space from the abdomen. Since the catheter tip was not above the diaphragm [Figure 1a] on the initial film, a cross-table lateral film would have shown whether the tip of the catheter was in the IVC below the diaphragm or within the liver, but this was not done. The cystic lesion was likely the result of a multifactorial process including vessel damage and bleeding as well as exposure of hepatocytes to hypertonic TPN within the liver leading to hepatocellular necrosis. Interestingly, the alpha fetoprotein (AFP) peaked at the time of decreasing mass size, possibly indicating hepatocellular regeneration. Despite the reassuring recovery, residual long term concerns for our infant include future portal hypertension from portal vein thrombosis or stenosis.

A previous case report describes a similar case in which the sterile, serous fluid collection was percutaneously drained; however, the long term follow up is not well described.[2] Other case reports describe fatal complications of hepatic parenchymal laceration, in which patients developed significant abdominal distension and had rapid deterioration of vital signs.[3,4]

A separate case series describes 11 cases of hepatic erosion.[5] Ten of these patients had spontaneous clinical improvement after removal of the UVC. One patient required percutaneous drainage of the liver lesion. In all of the neonates in this series, the catheter tip was between T8 and T12, all projecting over the liver.

The length of time a catheter remains in use may be important factor. Mean UVC duration in neonates has been reported to be about 4.4 days.[6] In a separate case series of 8 patients, mean duration of UVC use before detection of TPN extravasation was 8.9 days.[7] In our patient, the UVC was in place for a total of 10 days.

The differential diagnosis for a liver lesion in a neonate must include UVC complication. Neonatologists, consulting gastroenterologists, radiologists, and surgeons should be aware of this potential complication to allow for prevention or early recognition of this iatrogenic injury. We suggest that if the radiograph does not show the tip of the UVC above the diaphragm on an anterior-posterior view, a cross-table lateral film should be obtained to determine if the catheter tip is above the diaphragm. If the tip is within the liver, the catheter should be repositioned or removed. Vascular and/or hepatic complications should be considered in a neonate who develops abdominal distension after UVC placement. Long term follow up of such patients is recommended to track resolution of the hepatic lesion and to monitor for signs of portal hypertension.

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