CASE REPORT

Neonatal Total Parenteral Nutrition Ascites Secondary to Umbilical Venous Catheterisation

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ABSTRACT

Two cases of hepatic perforation with total parenteral nutrition ascites and hepatic calcification are reported. Hepatic perforation is a rare complication of umbilical venous catheterisation, resulting in intraperitoneal extravasation of the total parenteral nutrition fluid.

Key Words: Ascites, Hepatic calcifications, Total parenteral nutrition, Umbilical venous catheter

INTRODUCTION

Umbilical venous catheter (UVC) use has risen in recent years due to the increasing aggressiveness in the resuscitation of very low birth weight infants with difficult vascular access. UVC can be used for the administration of fluids and medications, exchange transfusion, and central venous pressure monitoring. Many complications of umbilical venous catheterisation can occur, however, including vascular injury, infection, thrombosis, portal hypertension, and fistula tract formation. Furthermore, there is the danger of introducing fluid through an abnormally positioned UVC.

This report describes 2 cases of hepatic perforation with total parenteral nutrition (TPN) ascites and hepatic calcification, a rare complication of UVC associated with intraperitoneal extravasation of TPN fluid.

CASE REPORTS

Case 1

A preterm baby girl was delivered at 30 weeks gestation with a birth weight of 1220 g and an Apgar score of 10 at one minute after birth. A size 5F umbilical venous catheter was inserted on day one, with the tip of the catheter at the level of the diaphragm, superimposed on the liver. Total parenteral nutrition was started on day 2. On day 6 of life, the neonate developed abdominal distension, respiratory distress and hypotension. An abdominal radiograph showed the tip of the umbilical catheter projecting at the L3/4 disc level, 1.5 cm to the right of midline (Figure 1). Ultrasonographic examination of the abdomen showed a circular hyperechoic lesion in the left hepatic lobe measuring 11 mm in diameter, and a large amount of ascites (Figure 2). A clinico-radiological diagnosis of TPN ascites was made. The UVC was removed and a peritoneal drain with low-pressure suction was introduced under local anaesthesia. Fifty mL of milky, blood-stained fluid, with a biochemical composition...
similar to the TPN fluid was drained. The baby’s condition improved rapidly and the drain was removed on day 7 of life. A follow-up ultrasound indicated a decrease in size of the hyperechoic lesion in the left lobe of the liver, and calcification of the lesion (Figure 3). The ascites had completely resolved.

Case 2
A 2660 g baby boy was born at home at 37 weeks of gestation to a mother with a psychiatric illness. A UVC was introduced on day 1 of life for resuscitation, due to hypothermia and TPN commenced on day 3. The neonate developed hypotension, with abdominal distension on day 7. Ultrasonographic examination of the abdomen showed the tip of the UVC to be in the left lobe of the liver. A lobulated echogenic mass, 13 mm in diameter, was noted surrounding the catheter tip, with a moderate amount of ascites present. The UVC was removed and a glove drain was inserted. Approximately 30 mL of serosanguinous fluid, rich in glucose, triglyceride, and cholesterol was drained. The baby’s abdominal distension resolved and the drain was removed the following day. Shrinkage of the echogenic lesion in the liver with posterior acoustic shadowing was seen on follow-up ultrasound examination.

DISCUSSION
The 2 cases described illustrate the typical clinical presentation of TPN ascites. As all TPN formulations are hypertonic with an alkaline pH, local tissue necrosis will result if the catheter tip is directly in contact with the hepatic parenchyma. In severe cases, disruption of the liver capsule gives rise to free intraperitoneal spillage of the TPN fluid. In keeping with previously reported cases, both patients in this report had TPN fluid in the peritoneal cavity. The echogenic lesion found in the liver around the UVC tip probably represented local hepatic parenchymal damage. In comparison to the case reported by Coley et al., the lesions noted were more homogeneous in appearance, perhaps reflecting a relatively smaller amount of hepatic parenchymal damage in the current patients. Of note, the presence of a large amount of ascites causes splinting of the diaphragm, resulting in sudden cardiorespiratory decompensation and hypotension.

The diagnosis of TPN ascites can be confirmed by a variety of methods. In babies utilising a UVC who demonstrate sudden clinical deterioration, immediate clinical and radiographic examination to confirm the position of the catheter is warranted, as complications can be life threatening. Abdominal and chest radiographs can be used but are more time-consuming. In addition, multiple studies are often required, due to the variability of right atrium and inferior vena cava positions in relation to bony landmarks and the procedure involves the use of ionising radiation. A contrast study is sometimes helpful but is usually not required for diagnosis. Ultrasonographic examination by comparison, is readily available to confirm the location of the UVC tip and offers immediate diagnosis of TPN ascites.

Treatment involves rapid discontinuation of the TPN infusion and removal of the catheter. Although TPN formulations are hypertonic, limited peritoneal irritation usually occurs and the ascites does not reaccumulate. Ascitic fluid can be removed by simple paracentesis, and rapid clinical improvement can be expected. Introduction of a drainage tube can probably be avoided.
with prompt identification of the condition, as shown in both of the cases outlined. This is in keeping with 8 cases of TPN ascites reported in the literature, in which drainage was not required. Follow-up studies in the current patients showed progressive shrinkage of the parenchymal injury, with peripheral calcifications.

The optimal position of a UVC tip is above the level of the diaphragm, at the inferior vena cava and right atrial junction. Studies have shown that all UVCs located from T7 to T9 were shown radiographically to be positioned correctly within the inferior vena cava or right atrium. However, in some cases placement of UVC at the ideal position may be difficult, placing the patient at risk, especially if the UVC is to deliver potentially injurious materials such as TPN. The radiologist has an important role in helping to identify malpositioning of a UVC and in alerting paediatric colleagues to the potential complications of UVC use.

REFERENCES