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Original article

Parenteral nutrition extravasation associated liver injury – A case series with varying clinical-radiological presentations

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Abstract

Umbilical vein catheterisation is a very common procedure in Neonatal Intensive Care Units. The tip of the umbilical vein catheter (UVC) should be located at the junction of inferior vena cava and right atrium, which is confirmed by an abdominal radiography or ultrasonography. The tip of the UVC can migrate to a different position because of a myriad of reasons. If hypertonic solutions are infused through an UVC which is malpositioned in the liver, it can result in serious complications. This is a case series of three neonates with malpositioned UVC and the resultant clinical presentations along with the characteristic ultrasonographic findings.

Keywords

Umbilical vein catheterisation, total parenteral nutrition, liver injury, extravasation.

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Background

The insertion of umbilical venous catheter (UVC) is a routine intervention in Neonatal Intensive Care Units. As per the current practice guidelines, most of the preterm neonates are instituted total parenteral nutrition (TPN) from the first day of life [1]. Most of the TPN infusates used in the initial days of life are hypertonic. A case series of three neonates with malpositioned UVCs and the resultant clinical complications along with the characteristic hepatic lesions accompanying it are presented here. The clinical features of the three cases are presented in **Tab. 1**.

Case 1. Sudden onset of abdominal distension and shock in a neonate treated for NEC II: perforation?

A 31 weeks, dichorionic diamniotic twin, male, with a birth weight of 1,830 grams, was delivered by Caesarean section. The neonate had cried immediately after birth and did not require any resuscitation. A diagnosis of necrotising enterocolitis stage II (NEC II) as per modified Bell's classification was kept on day 6 of life. A 5 Fr UVC was inserted and TPN was started on day 6 of life. On day 11 of life, the baby had sudden onset of abdominal distension and shock. Ultrasonography revealed ascitic fluid with debris. There was a 4.1 cm x 4 cm x 3.8 cm hypoechoic lesion with a hyperechoic rim in the liver. Ascitic tap was done, which showed a milky yellowish fluid resembling the TPN fluid that was instituted. Abdominal radiograph revealed malpositioned UVC, most likely in the superior mesenteric vein (**Fig. 1**). The UVC was removed and a peripherally inserted central catheter (PICC) line was inserted. The ascitic fluid biochemical analysis revealed high lipid, sugar and protein levels with no cells confirming TPN extravasation. Over the course of the next 12 hours, there was symptomatic improvement in the form of resolution of abdominal distension. The hepatic lesion resolved by 2 months of age.

Case 2. Progressive abdominal distension, hepatomegaly and anaemia in a preterm baby with RDS: liver hematoma?

A 28 weeks monochorionic diamniotic twin with a birth weight of 1,100 grams and of male sex was delivered by Caesarean section. The neonate had cried immediately after birth and did not require any resuscitation. The baby had severe respiratory distress syndrome (RDS) requiring surfactant replacement followed by high-frequency oscillation ventilation. A 5 Fr UVC was inserted on the first day and TPN was initiated. Abdominal radiograph revealed the position of the UVC to be in the inferior vena cava (IVC). On day 7 of life, the baby had abdominal distension and anaemia requiring packed cell transfusion. Abdominal ultrasonography revealed a hyperechoic lesion with a hypoechoic rim with a measurement of 3.8 cm x 3.6 cm x 3.2 cm, which resolved by 33 weeks postmenstrual age (**Fig. 2**).

Case 3. Yellowish fluid output from a peritoneal drain in a post-operative baby with intestinal stricture: anastomotic leak?

A 36 weeks singleton male neonate with a birth weight of 2,100 grams was delivered

Table 1. Clinical features of the three cases with total parenteral nutrition (TPN)-induced hepatic injury.

Case	GA (weeks)	PNA at presentation of TPN induced liver injury (days)	Clinical presentation of TPN induced liver injury	Established diagnosis at presentation	Differential diagnosis	Outcome
1	31	11	Sudden onset of abdominal distension and shock	NEC II – Diagnosed on day 6	Worsening NEC with perforation	Improved after UVC removal; discharged on day 24
2	28	7	Sudden onset of abdominal distension and pallor	RDS	Liver hematoma/ hemangioma/ hepatoblastoma	Lesion regressed spontaneously by 35 days; discharged by 42 days of life
3	36	5	Yellowish output from the peritoneal drain inserted intraoperatively	Operated case of jejunal stricture with end-to-end anastomosis (operated on day 4)	Anastomotic leak	Drain output decreased after UVC removal and the hepatic lesion resolved by 3 months of age

GA: gestational age; PNA: post-neonatal age; TPN: total parenteral nutrition; NEC: necrotising enterocolitis stage; UVC: umbilical venous catheter; RDS: respiratory distress syndrome.

vaginally and shifted for suspected surgical abdomen. The neonate had cried immediately after birth and did not require any resuscitation. A 6 Fr UVC was inserted, TPN instituted and UVC positioned confirmed by abdominal radiograph. He underwent laparotomy at 98 hours of life and primary anastomosis was done for jejunal stricture following which a peritoneal drain was inserted. On the first post-operative day, there was a copious output of yellowish milky fluid, which resembled the TPN that was instituted. Abdominal radiograph, which was repeated, revealed that UVC had migrated and was malpositioned in the right portal vein. The UVC was removed and a PICC line was inserted. The output from the peritoneal drain decreased and diagnosis of TPN extravasation was made. Ultrasonogram of the liver revealed a dense hyperechoic lesion of size 3 cm x 2.2 cm x 2 cm, which resolved by 3 months of age (**Fig. 3**).

Discussion

TPN extravasation induced liver injury is a well-known complication of malpositioned UVCs [2-5]. However, the characteristic sonographic features of the hepatic injury caused by TPN extravasation have been increasingly documented recently [6-8]. The clinical presentation can vary from asymptomatic abdominal distension due to hepatomegaly to cardiovascular compromise due to abdominal compartment syndrome or hypovolemia.

Nour et al. [5] had described infants being treated for NEC having sudden abdominal distension with respiratory distress and ascitic tap revealing TPN like fluid [9]. While two of those neonates were conservatively managed, one required a laparotomy. The first case in this case series had a very similar presentation to these newborns. This neonate was also being managed as NEC. Sudden abdominal distension with yellowish ascitic fluid as seen in Nour et al.'s and our case might mislead the clinician to make a diagnosis of worsening NEC and the neonate might have ended up with an unwarranted laparotomy.

In a case series of four neonates, Lim-Dunham et al. described the ultrasonographic features of TPN extravasation induced hepatic injury [6]. Whereas we had described the lesions as uniformly hyperechoic as in case 3 or hypoechoic with a hyperechoic centre as in case 2, it was hypoechoic with a hyperechoic rim in Lim-Dunham's cases. The case 1 had a hypoechoic lesion with a hyperechoic rim as described by Lim-Dunham. Coley et al. and

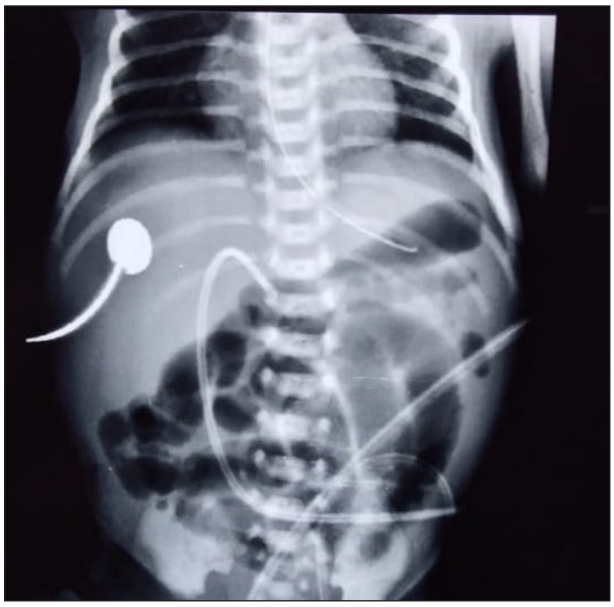

Figure 1. Abdominal radiograph revealing malpositioned umbilical venous catheter (UVC) in the superior mesenteric vein.

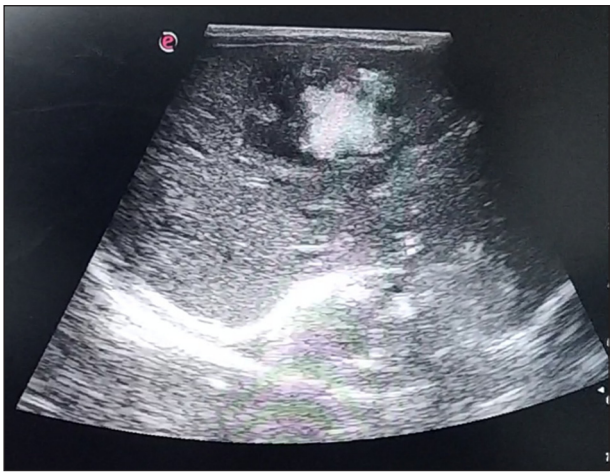

Figure 2. Abdominal ultrasonography revealing a hyperechoic lesion with a hypoechoic rim with a measurement of 3.8 cm x 3.6 cm x 3.2 cm.

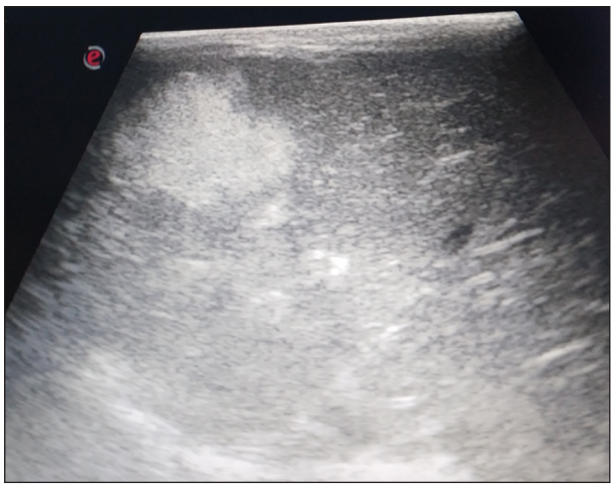

Figure 3. Ultrasonogram of the liver showing a dense hyperechoic lesion of size 3 cm x 2.2 cm x 2 cm.

Hui et al. have described diffusely hyperechoic lesions in TPN extravasation induced hepatic injury [7, 9]. The most recent case report by Hartley et al. describes these lesions as "TPNoma" [10]. Like the outcomes in our cases, the follow up of the cases reported by Lim-Dunham et al. had also shown the lesions to either disappear completely or leave a small residual calcification. Overall, "TPNomas" can vary significantly in their appearances on initial evaluation and usually disappear on their own with no long term complications.

Hagerott et al. had described in a case series of five neonates the clinical-radiological features of TPN induced hepatic necrosis [8]. Similar to the second case described here, two of Hagerott's case had anaemia requiring transfusion. It was postulated that the anaemia was caused due to hematoma that developed at the site of the liver necrosis. The mechanism postulated for the appearance of the lesion as being hyperechoic and hypoechoic was that the TPN which is made of proteins, lipids and dextrose would separate into an aqueous hypoechoic part and a lipid-rich hyperechoic region.

Ideally, the UVC passes through the umbilical vein into the medial part of the left portal vein and then onto the ductus venosus through which it enters the IVC [11]. The ideal position is at the junction of IVC and the right atrium. The review of literature on TPN induced hepatic injury shows one common factor in all the cases, which is the malposition of UVC overlying the hepatic shadow in the abdominal radiograph. This case series further emphasizes the importance of checking the position of UVC immediately after insertion and either repositioning it if required or obtaining an alternate central venous access. Not doing so can result in severe complications such as hepatic injury and ascites which can be life-threatening also.

Declaration of interest

The Authors declare that there is no conflict of interest.

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