Pictorial essay

Med Ultrason 2021, Vol. 23, no. 1, 89-93
DOI: 10.11152/mu-2615

Imaging appearance of post umbilical venous catheter displacement: liver collections in children

Ayşe Keven[1], Adnan Kabaalioğlu[2], Emel Durmaz[1], Kağan Çeken[1]

[1]Akdeniz University, Medical Faculty, Department of Radiology, Antalya, [2]Koç University, Medical Faculty, Department of Radiology. Istanbul, Turkey

Abstract

Umbilical venous catheters (UVCs) have become a part of routine perinatal care. In the case of its misplacement, extravasation into liver parenchyma might be observed and unusual findings might be detected and a suspicion of tumoral lesions emerges during the ultrasound examination. To avoid the unnecessary liver biopsies and catastrophic complications of UVC misplacement in the pediatric population, clinicians and radiologists must be familiar with the radiological findings. We aimed to present sonographic and computed tomographic images of liver collections resulting from UVC malposition.

Keywords: liver mass; pseudotumor; umbilical venous catheter

Introduction

The umbilical venous catheter (UVC) is a commonly used method to provide fluids, total parenteral nutrition (TPN) and drug to infants followed in neonatal intensive care unit [1].

The umbilical vein starts from the umbilicus and runs along the midline of the anterior wall of the abdomen, advancing to the liver, puring in the left portal vena, the flow being then directed to the ductus venosus and into the inferior vena cava (IVC) [2,3] (fig 1).

Umbilical venous catheters are inserted by pediatricians without the need for imaging methods. The depth of the insertion of the UVC can be calculated by several formulas and graphs using various body measurements [1]. On a frontal radiograph, the tip of the umbilical venous catheter should be above the diaphragm, in the suprahepatic segment of the IVC [2]. After catheterization, the position of the cannula should be assessed with radiological examinations of the abdomen and chest (fig 2). Yeh et al 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 ob-

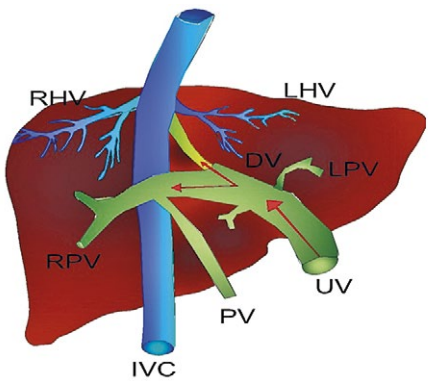


Fig 1. Diagram of the neonatal umbilical and portal venous system. The umbilical vein joins the left portal vein in the liver, which runs to the hepatic sinusoid, and direct communication is established between the umbilical vein and the ductus venosus, that bypasses the liver and joins the inferior vena cava. DV, ductus venosus; IVC, inferior vena cava; LHV, left hepatic vein; LPV, left branch of the intrahepatic portal vein; PV, portal vein; RHV, right hepatic vein; RPV, right branch of the intrahepatic portal vein; UV, umbilical vein.

Received 09.05.2020 Accepted 04.07.2020
Med Ultrason
2021, Vol. 23, No 1, 89-93
Corresponding author: Ayşe Keven, MD, Research Associate
Akdeniz University, Medical Faculty,
Department of Radiology. Antalya, Turkey
Dumlupınar bulvarı, Akdeniz Üniversitesi
Hastanesi, 07059 Arapsuyu, Antalya, Turkey
E-mail: aysekeven@gmail.com
Phone: 90 533 523 8027
ORCID ID: 0000-0002-1662-8586

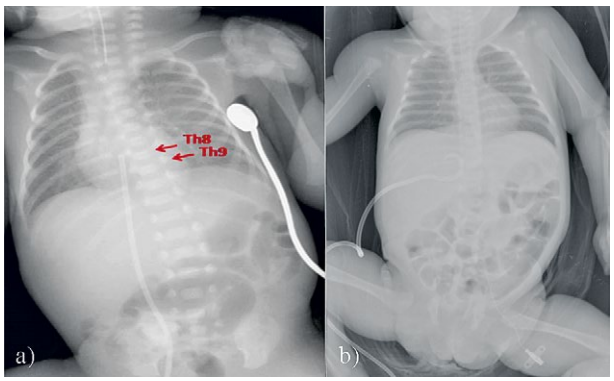


Fig 2. a) Normal radiographic appearance of the umbilical venous catheter. The umbilical venous catheter enters the abdomen at the umbilicus; it then travels in the cephalad direction, and ends at the level of the thoracic 8-9 vertebra; b) On the abdomen radiograph showing the abnormal position of UVC, the tip of the catheter is under the diaphragm, likely in hepatic parenchyma.

tained to determine the position of the catheter [4]. Prior studies have concluded that the US is the more precise techniques in determining the appropriate position of the UVC tip, showing that the technique is reliable and may replace a plain radiograph for the identification of the UVC position, preventing complications from malposition and reducing exposure to ionizing radiation [5,6].

However, despite the fact that UVC has many benefits, it carries many risks such as infection, thrombosis and extravasation. Mutlu et al [7] found that the complication rate after UVC was 20.3% the most common being malposition. Derinkuyu et al [8] reported liver complications after UVC as 33.6% (20.1% air in the portal venous system, 6.1% portal system thrombosis and 7.4% liver parenchymal lesions). When it is misplaced, UVC can lead to extravasation into the liver parenchyma and various unusual findings; sometimes tumor-like lesions might be suspected when examined by ultrasound.

In this essay, we present sonographic and computed tomographic images of the liver lesions caused by the leaks from the UVC in light of the relevant medical literature. The presence of asymptomatic cases indicates that liver complications may be much more common than previously believed. Therefore, clinicians and radiologists must be familiar with the radiological findings to avoid unnecessary liver biopsies and catastrophic complications of UVC misplacement in the pediatric population.

Liver complications and imaging features

Hepatic complications sometimes can be life-threatening in newborns. They are categorized as early and late complications as follows:

Early complications

Hepatic fluid collection is a serious complication of UVC misplacement. To minimize iatrogenic injury, it is crucial to position the catheter correctly before starting the infusion of hyperosmolar solutions and recognize UVC-related complications early.

The problems related to the displacement of the catheter may not be significant if the leak is small. The result may be a small accumulation that may resolve spontaneously, may require aspiration or become infected. If the displacement is noticed early, the catheter is usually removed rather than trying to correct its position, so that the damage remains minimal and reversible. However, if the misplacement is not noticed early on, then the damage may lead to catastrophic events such as hepatic necrosis, collection and abscess formation. Subsequently, accumulation of the intravenous fluids administered via the UVC creates an enlarging cystic space within the liver, escapes into the peritoneal cavity eventually and enters the pleural space from the abdomen. The development of this kind of cystic lesion results likely from a multifactorial process including vessel damage and bleeding as well as the exposure of hepatocytes to hypertonic TPN that leads to hepatocellular necrosis [4]. The intraparenchymal hematoma can be due to vascular injury or erosion or laceration of the hepatic parenchyma (fig 3) [9,10].

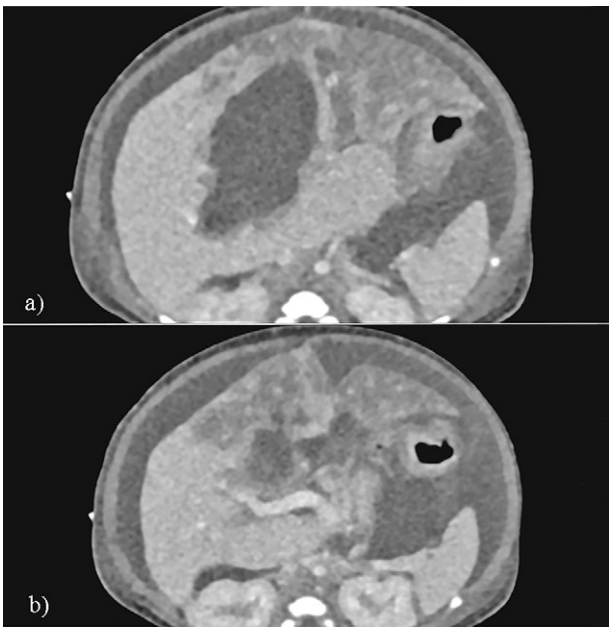


Fig 3. A 5-day-old girl had suddenly deteriorated - clinical features with abdominal distention. a) On the CT images, a large fluid accumulation that compressed the left portal vein and impairment of perfusion in the left lobe was observed at segment 2, 3 and segment 4; b) Rupture of the liver capsule led to the accumulation of extensive free fluid within the abdomen.

Late complications

Late hepatic complications due to UVC are important causes of morbidity and mortality. The most important of these are *thromboembolic complications* that cause *portal hypertension*. The catheter tip may damage the endothelial wall, and thus, thrombus formations, stenosis and occlusions may be seen in the portal system. The hepatic arterial and venous system may also be affected, usually indirectly or through compression [11]. Endothelial damage-causing portal vein thrombosis is related to catheter type, duration, and location of the catheter and composition of the infusate. Low birth weight, low flow state, hypercoagulation disorders and hypoxia also cause thrombosis [12,13]. Kim et al [13] report that the most significant risk factor is the duration of the UVC and whether most of the small thrombi were attached to the catheter rather than the wall of the portal vein so that a catheter in the lumen is closely related to the initiation of thrombosis. Portal vein thrombosis may lead to cirrhosis in the long term (fig 4).

Complications due to the misplacement of UVC may be sometimes asymptomatic or overlooked. Another less known major diagnostic challenge is that the residual lesions due to the leaks from previous UVC might later mimic a *liver mass* and maybe interpreted as neoplasms. Residual lesions usually resolve slowly over months.

On ultrasound (US) examinations, the hepatic fluid collection is typically seen along the course of ductus venosus; the left lobe of the liver in segment IVA, segment IVB and anterior segments of the right lobe (fig 5). Sometimes, a malpositioned catheter pushed too far peripherally into the portal vein can produce perforation and extravasation in the distal segments of the right or left lobes. The important feature of these lesions is the hyperechogenicity, due to the fat content. Lim-Dunham et al [14] described a change in the sonographic appearances of the liver collections over time. Initially, they are well-defined with a hyperechoic rim and a hypoechoic center

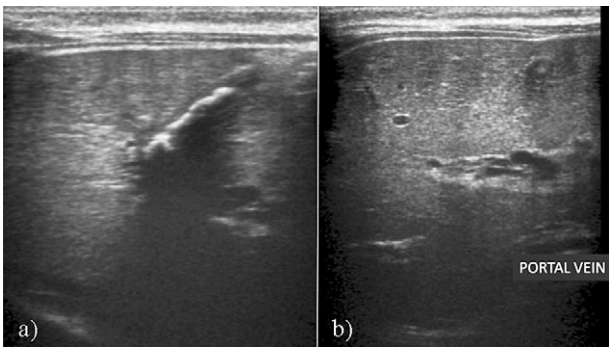


Fig 4. Ultrasonographic examination of a 3-month-old male shows calcification on the course of ductus venosus (a) and portal venous thrombosis with cavernous transformation (b).

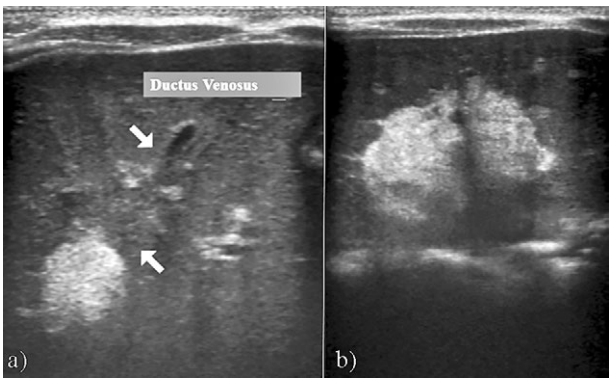


Fig 5. In a 5-month-old male a) and b) transverse sections of the left hepatic lobe show a well-marginated echoic lesion with a lobule contour along the course of ductus venosus. (arrows show ductus venosus trace)

with cystic areas (fig 6). Cystic areas are usually seen in the early period and reflect the fresh extravasated fluid or sometimes the fresh blood due to the erosion of the liver by the catheter tip. With progression, the collections become more echogenic, presumably from the initial absorption of the liquid components (fig 7a). A striking feature that frequently goes unnoticed is the high attenuation

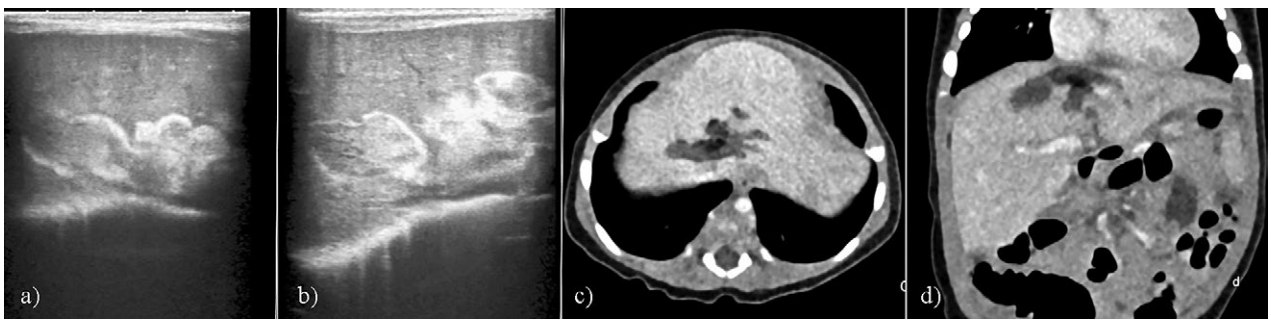


Fig 6. A 17-day-old girl with typical sonographic findings: a) and b) showing a hyperechogenic heterogeneous lesion with an echogenic rim in segment IV A of the left lobe and segment 8 of the right lobe; c and d) Axial and coronal portal phase CT images showing mixed-density intraparenchymal hepatic lesions with fat density. There is no contrast enhancement.

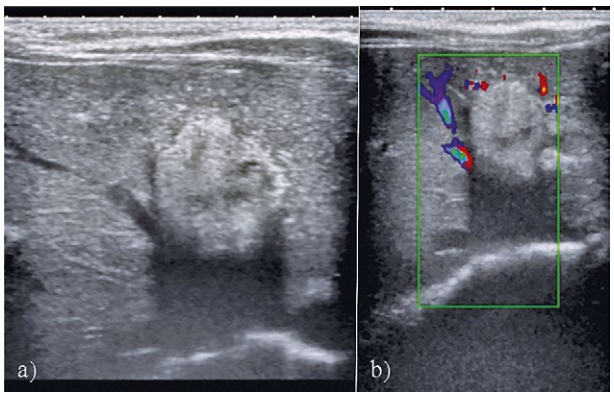


Fig 7. Transverse sonogram of a 4-month-old male: a) showing a well-marginated, heterogeneously hyperechoic parenchymal lesion with a hyperechoic rim; b) Color Doppler image with no vascularity.

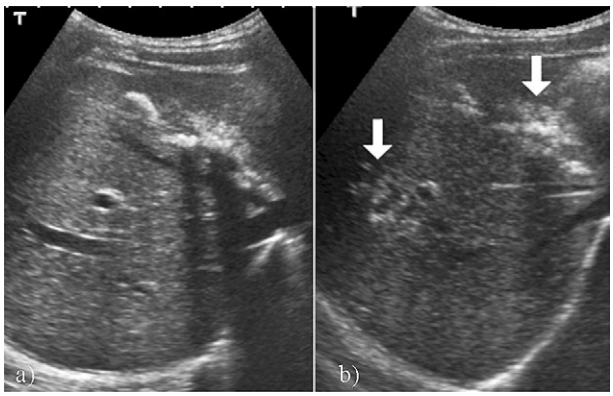


Fig 8. Transverse sonogram of a 4-month-old male infant through the left lobe of the liver during the follow-up: a) and b) the lesion decreased in size, dissolved and shows the development of calcification with posterior acoustic shadowing.

of the lesions, characterized by prominent distal acoustic shadowing. This may at times be interpreted as the shadowing of the "calcified echogenic lesion". Some cystic areas may be seen in the late phase reflecting the residual liquid content. With further healing, a calcified rim develops. These lesions do not show vascularity on the Doppler imaging (fig 7b). The shapes of the lesions are also unique and reflect their non-neoplastic nature: geographic, usually irregular and sometimes perivascular, band-like or tubular. During follow-up, these lesions tend to decrease in size and/or develop calcifications (fig 8).

On contrast-enhanced CT, residual lesions can appear non-enhancing mixed density lesions along to the umbilical vein course and it would always raise a suspicion of a residue of UVC. It has fluid, blood and especially fat, which is probably due to the delivery of transfused blood, TPN fluid and fat into the hepatic parenchyma as well as due to liver necrosis. When there is a disruption of the liver capsule, there is extravasation and peritoneal spillage of parenteral nutrients. If the entity is known with its sonographic features and if there is a history of UVC in the previous months, follow-up can be suggested instead of aggressive approaches such as biopsy or excision of the lesions with a suspicion of neoplasm. Follow-up will certainly reveal the benign non-neoplastic nature of these lesions. If a biopsy or aspiration is done, the aspirate might be the content of the fluid given by UVC. In the late phase, a biopsy will not usually be diagnostic but it can only be helpful to eliminate malignancy suspicion.

Differential diagnosis

The differential diagnosis for hepatic lesions with heterogeneously hypoechoic centers and hyperechoic rims in neonates are abscesses and hematomas. Clinical history correlation can exclude the possibility of a hematoma from a lack of birth trauma and an abscess from a lack of history of necrotizing enterocolitis and prolonged sepsis [15]. Lack of peripheral hypervascularity on sonography can exclude the possibility of an abscess. Abscess or hematoma may develop due to UVC extravasation. Aspiration, in this case, will help the diagnosis.

The echogenic appearance of UVC extravasation in the resorption stage may confuse the differential diagnosis of common liver masses. The most common liver lesions in the neonatal and infantile period are hemangioma, mesenchymal hamartoma and hepatoblastoma [15]. Intensive care hospitalization and catheterization history will be the greatest help in this distinction. However, when a differential diagnosis cannot be made by the US, MRI should be the selected method for the diagnosis.

In **conclusion**, the differential diagnosis of a liver lesion in neonates and infants must include UVC complications. Radiologists should be aware of the presence of asymptomatic cases. Also, the typical sonographic appearance and location are of diagnostic significance without the need for any advanced imaging methods. Long term follow-up of such patients is recommended to review the resolution of the hepatic lesion and to monitor the signs of portal hypertension.

Conflict of interest: none

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