Hepatic Complications of Umbilical Venous Catheters in the Neonatal Period

The Ultrasound Spectrum

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Objectives—Umbilical venous catheterization is commonly used in the neonatal period; however, it has some complications. In this study, we evaluated neonates who underwent umbilical venous catheterization and developed hepatic complications. Furthermore, we aimed to define all of the possible lesions and to clarify the imaging findings of umbilical venous catheter–induced hepatic injury.

Methods—Two hundred forty-four neonates who underwent umbilical venous catheterization between March 2013 and September 2015 in a single tertiary care referral center were included in this study. To determine whether they had any hepatic complications, all patients underwent abdominal grayscale and Doppler ultrasound examinations, and their clinical data were recorded.

Results—The frequency of liver-related complications from umbilical venous catheterization was 33.6% (82 of 244). Air in the portal venous system was the most frequent complication (20.1% [49 of 244]). Left portal venous thrombosis was noted in 6.1% (15 of 244). Parenchymal lesions in the liver related to umbilical venous catheterization were seen in 7.4% of patients (18 of 244) as follows: single nodular echogenic lesions (4.1% [10 of 244]), branching small nodular echogenic lesions (2.1% [5 of 244]), and large irregular heterogeneous lesions with laceration and perihepatic fluid (1.2% [3 of 244]). There was no statistical significance for any type of complication according to the gestational age ($P > .05$).

Conclusions—Hepatic complications due to umbilical venous catheters are not uncommon in the neonatal period. Ultrasound is the best imaging modality for confirming the diagnosis and for follow-up.

Key Words—laceration; liver; neonate; pediatrics; ultrasound; umbilical venous catheter

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Abbreviations

CT, computed tomographic; NICU, neonatal intensive care unit; TPN, total parenteral nutrition; US, ultrasound

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Umbilical venous catheterization is a commonly used procedure in the neonatal period. It allows vascular access for drug administration, exchange transfusion, or total parenteral feeding, especially in premature and low-birth-weight neonates.[1–3] As with every other interventional procedure, umbilical venous catheterization has some complications, including commonly related infections, thrombosis, and malpositioning (to the portal venous system or heart).[3–8] Since umbilical venous catheterization carries the risk of these complications, its necessity must first be accurately assessed, and then the catheter should be properly placed and removed soon. Therefore, neonates who undergo umbilical venous catheter placement should be monitored radiologically to be able to detect any complications as early as possible.

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An umbilical venous catheter generally passes directly superiorly and remains relatively anterior in the abdomen. It passes anterosuperiorly through the umbilicus, umbilical vein, left portal vein, ductus venosus, and middle or left hepatic vein and terminates into the suprahepatic inferior vena cava. The tip of the catheter is the most important factor in the development of hepatic injury. The tip should lie at the junction of the inferior vena cava with the right atrium, not in the right atrium or inside the liver parenchyma.[2,3,6] The catheter is blindly inserted by the pediatrician with respect to a predetermined length. Given the small size of neonates (especially those requiring umbilical catheters), a little difference in the catheter length can cause substantial malpositioning, which may result in structural injury, such as hepatic hematoma from perforation of an intrahepatic vascular wall.[4,5,7–11] Some complications, such as formation of a thrombus along the catheter, can also occur in malpositioned or well-positioned catheter as well.[12,13] Also, a properly placed catheter may be withdrawn or may migrate, and the tip of the catheter may be displaced into the liver.[14–16] For these reasons, neonates with umbilical venous catheters should be closely monitored, and great care must be taken to prevent possible complications. In this study, we evaluated neonates who underwent umbilical venous catheterization and developed hepatic complications. Furthermore, we aimed to define all of the possible lesions and to clarify the imaging findings of umbilical venous catheter–induced hepatic injury.

Materials and Methods

Patients

This study was approved by our Institutional Research Ethics Board. All studies performed for clinical indications were in line with the local ethical guidelines and standard departmental imaging protocols. Informed consent forms were signed by the parents of all participants included in the study. We prospectively evaluated all of the neonates who underwent umbilical vein catheterization between March 2013 and September 2015 in a neonatal intensive care unit (NICU) of a single tertiary care referral center. Of 251 patients, 7 were excluded. The exclusion criteria were having parenchymal liver lesions (4 patients) and ascites (3 patients) on ultrasound (US) imaging before catheter placement. Consequently, 244 patients were included in the study.

Technique

The umbilical venous catheters were inserted by a pediatric neonatologist without imaging guidance. After the procedure, in every neonate, the catheter tip was controlled with an anteroposterior thoracoabdominal radiograph to reposition the catheter tip if necessary. The correct position of the catheter tip was at the level of the right hemidiaphragm or between T8 and T10. Double-lumen 3F to 5F umbilical venous catheters were used to allow simultaneous administration of fluids, drugs, and total parenteral nutrition (TPN) as needed.

Abdominal grayscale and Doppler US examinations were performed before catheter placement, after insertion of the catheter at 3- to 7-day intervals in the NICU, and after removal of the catheter at 3- to 7-day intervals until hospital discharge. Furthermore, follow-up US examinations were performed after discharge at 1- to 3-month intervals if considered necessary. The abdominal US examinations were performed by a pediatric radiologist at the bedside in the NICU or in a US room at the Department of Radiology after hospital discharge. Repeated abdominal US examinations and abdominal computed tomographic (CT) examinations were performed if clinically required. The pediatric radiologist evaluated the liver parenchyma and noted the presence of liver parenchymal lesions, which were classified into 3 different groups according to the US appearance: (1) single nodular echogenic lesions, (2) small branching echogenic lesions, and (3) large heterogeneous lesions with laceration and effusion. On the Doppler evaluation of the hepatic vascular system, the patency was assessed, and thrombosis or air in the portal venous system was noted. Eventually, abdominal ascites, splenomegaly, and any collateral vascularity compatible with portal hypertension were evaluated. All of the umbilical venous catheter–related complications associated with the liver were noted and followed.

All statistical analyses were done with SPSS version 16.0 software (IBM Corporation, Armonk, NY). $P < 0.05$ was taken to represent statistical significance.

Results

Among the 244 neonates who underwent umbilical venous catheterization in the study, 54.9% (134 of 244) were female, and 45.1% (110 of 244) were male. Of the included infants, 88.5% (216 of 244) were preterm, and 11.5% (28 of 244) were term (according to the

gestational age). All of the umbilical venous catheters were placed within 48 hours after birth. All 244 patients had normal liver parenchyma and a patent portal vein on the grayscale and Doppler examinations before catheter placement.

After catheter placement, 20.1% (49 of 244) of the neonates had air in the portal venous system (via liver US) without any evidence of necrotizing enterocolitis (clinically or radiologically) (Table 1). The air was located peripherally (pushed along by the blood) in the liver parenchyma with a small-caliber branching configuration (Figure 1). On US examinations, air in the portal venous system was seen as small particles of high echogenicity flowing in the portal vein, with prominent shadowing or a reverberation artifact. Of the 49 neonates with air in the portal venous system, 69.4% (34 of 49) had air in the anterior segments of only the left portal vein branches, whereas 30.6% (15 of 49) had air in the anterior segments of both the right and left portal vein branches. The air was detected by US 1 to 3 days after catheter placement. Ultrasound revealed that all of the air was gone 5 to 7 days after placement.

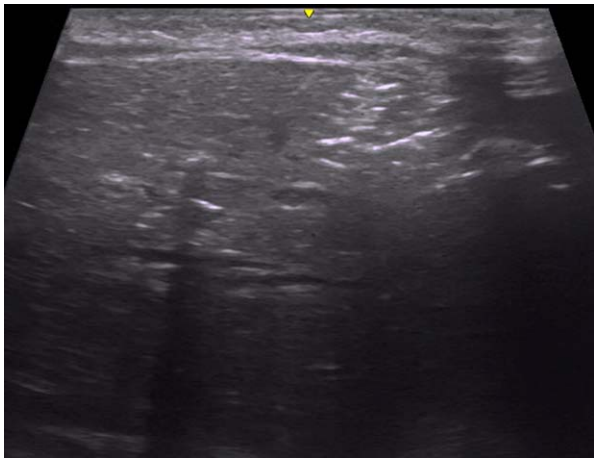
Portal venous thrombosis was detected in 6.1% (15 of 244) of the neonates. In all of these cases, the thrombus was seen in the left portal vein, which is located between the umbilical vein and the ductus venosus (Figure 2). Left portal venous thrombosis in 9 (60.0%) of the 15 patients was diagnosed before catheter removal. In 7 (77.8%) of these 9 patients, color Doppler US revealed that a small thrombus was attached to the catheter, or the catheter was partially filled with a thrombus and allowed blood flow from the remaining patent lumen. In the remaining 2 patients (22.2%), a complete thrombus filled the entire lumen, and blood flow was not seen (Figure 3). The left portal venous thrombosis of the remaining 6 patients (40.0%) was diagnosed after removal of the catheter. In these patients, the thrombus did not extend into the right or main portal vein, although it did spread into the more peripheral branches of the left portal vein in 5 (33.3%) of the 15 cases. The liver and the spleen were not enlarged, and neither ascites nor extrahepatic collateral vessels were noted. There were no specific clinical findings suggesting acute portal occlusion in any of the 15 cases with portal venous thrombosis. The affected neonates were treated with low-molecular-weight heparin. After detection of the thrombus, the patients were with grayscale and Doppler US. Thrombosis was monitored to complete resolution in 8 neonates (53.3%). Resolution of thrombosis after catheter removal varied from 7 to 45 days (mean, 15 days). In the remaining 7 cases (46.7%), thrombosis was persistent, and either the thrombus contracted to become an echogenic linear structure, or the lumen of the portal vein became obliterated without any flow signal on the color Doppler US examination. In addition, 5 cases (71.4%) that had persistent thrombosis of the left portal vein appeared to have a smaller left lobe of the liver compared with that seen in previous scans. In all of these cases, the right and main portal veins were intact, and splenic venous flow was preserved. We did not encounter any case that had a cavernous transformation of the portal vein, and none of these patients developed portal hypertension.

After umbilical venous catheter placement, we detected different types of liver parenchymal lesions via liver

Table 1. Hepatic Complications of Umbilical Venous Catheter in 244 Neonates

	%
Air in the portal vein	20.1% (n=49/244)
Left portal vein thrombosis	6.1% (n=15/244)
Liver parenchymal lesions	7.4% (n=18/244)
Single echogenic lesion	4.1% (n=10/244)
Multiple small branching echogenic lesions	2.1% (n=5/244)
Large heterogeneous lesion with laceration and effusion	1.2% (n=3/244)

Figure 1. A 2450-g male neonate was born at 37 weeks' gestation. The mother had oligohydramnios and placental abruption. The neonate had an umbilical venous catheter replacement. On the day 1 US examination, there were small air particles in the left portal venous system peripherally.



US. Parenchymal lesions in the liver related to the catheter were seen in 7.4% (18 of 244) of patients as follows: 4.1% (10 of 244) had a single nodular echogenic lesion; 2.1% (5 of 244) had branching small nodular echogenic lesions; and 1.2% (3 of 244) had large irregular heterogeneous lesions with laceration and perihepatic fluid.

Of the 18 patients with liver parenchymal lesions, 55.6% (10 of 18) had single nodular echogenic lesions (Figures 4 and 5). Of these, 7 lesions (70.0%) were located at segment 4 of the left liver lobe; 2 (20.0%) were located at segment 2 of the left liver lobe; and 1 (10.0%) was located at segment 8 of the right liver lobe. Of the 10 single nodular lesions, 6 (60.0%) were homogeneously echogenic, whereas 4 (40.0%) showed some heterogeneity and contained a small hypoechoic area inside, which likely represented necrosis. The parenchymal echogenic lesions had a well-circumscribed contour in 8 lesions (80.0%) and a lobulated contour in 2 (20.0%). The dimensions of these single echogenic lesions were between 8 and 20 mm (mean, 12 mm).

Of the 18 patients with liver parenchymal lesions, 28.7% (5 of 18) had small branching nodular echogenic lesions. All 5 of these patients had echogenic lesions located at segments 2 and 3 of the left liver lobe, and all of them had left portal venous thrombosis that spread into the more peripheral branches as well (Figure 6).

Figure 2. A 2800-g female neonate was born at 33 weeks' gestation. On the fifth day after the umbilical venous catheter placement, there was a partial thrombus around the umbilical venous catheter (**A**, black arrows) in the left portal vein (white arrows), and the centrally located catheter lumen was patent (**B**). Note the echogenic thrombus (white arrows) around the catheter in the short axis.

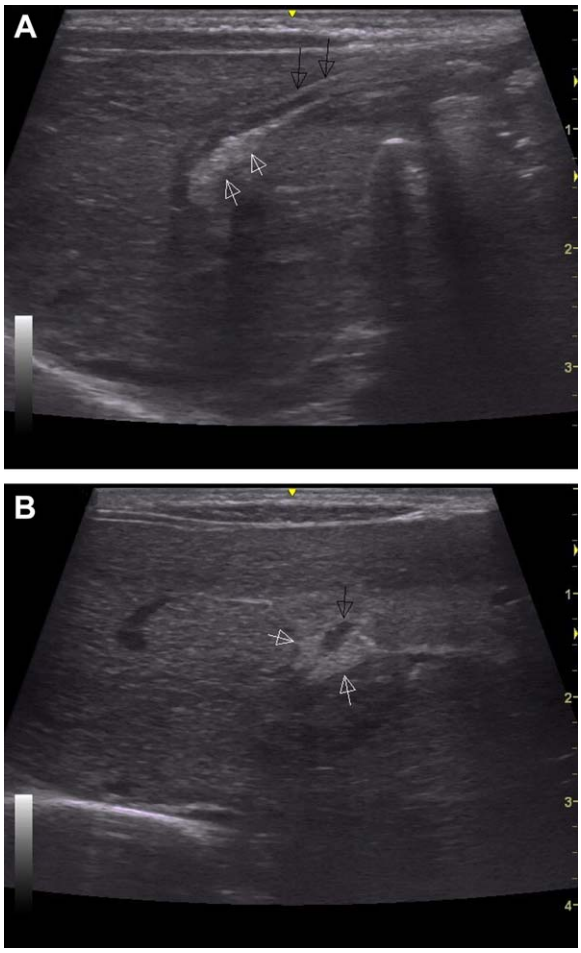
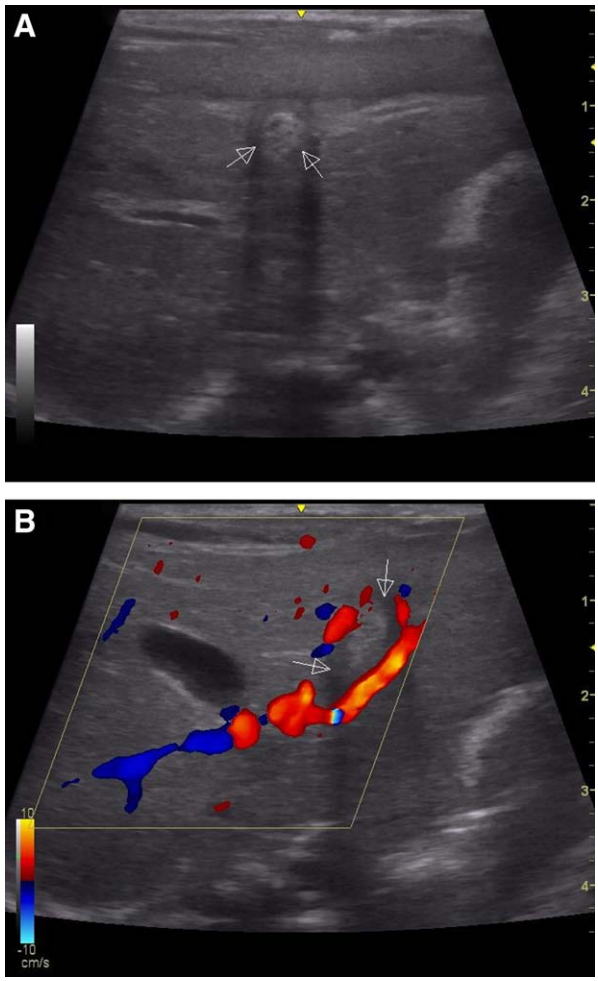


Figure 3. A 3150-g male neonate was born at 38 weeks' gestation. The neonate had asphyxia. The umbilical venous catheter was removed after 4 days, and a US examination was performed on day 5. The left portal vein (**A**) was totally thrombosed (white arrows), and on color Doppler imaging (**B**), the vein was not filling.



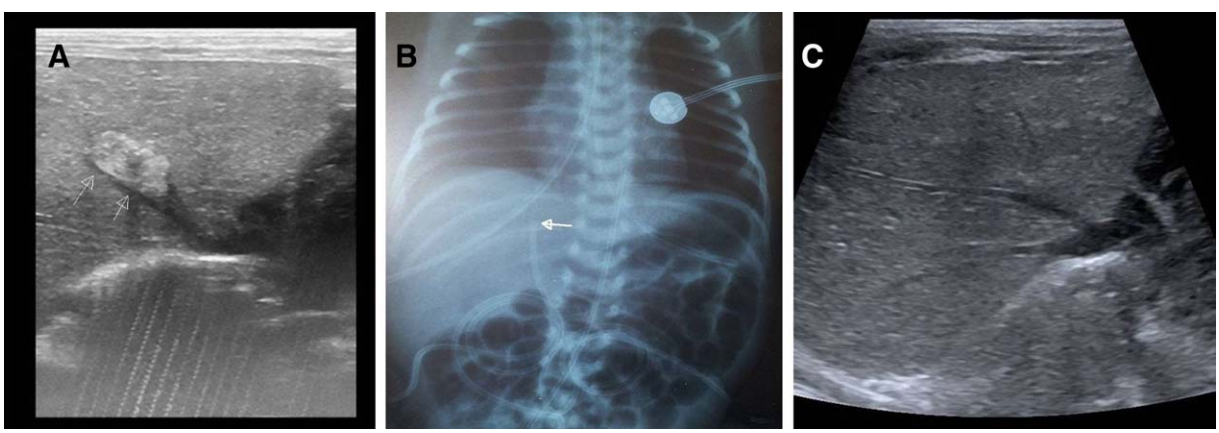
The small lesions were seen in a ramified manner around the left portal vein branches. The dimensions of these small echogenic lesions were between 3 and 8 mm (mean, 5 mm) with a well-circumscribed contour.

Of the 18 patients with liver parenchymal lesions, 16.7% (3 of 18) had large, irregular heterogeneous lesions; all of these had liver parenchymal laceration and perihepatic effusion (Figure 7). The dimensions of these parenchymal lesions were between 20 and 35 mm (mean, 28 mm) with a lobulated and irregular contour. All 3 of the patients with large parenchymal lesions had a heterogeneous echo pattern with a necrotic area, calcifications, and posterior shadowing. In addition to the large parenchymal lesions, all 3 of these patients had an echogenic line reaching to the liver capsule, which was compatible with laceration. All 3 patients had perihepatic fluid, abdominal distension, and elevated liver enzyme levels. All 3 patients received TPN, with varying concentrations of dextrose, amino acids, and lipids. Two of these patients (66.6%) had chylothorax as well. One of the 3 (33.3%) had left portal venous thrombosis on a Doppler US examination. Two of the 3 (66.6%) underwent abdominal CT examinations to determine the extensiveness of the damage. Abdominal CT revealed fat density inside the parenchymal lesions in the liver (Figure 7). All 3 of these cases underwent US-guided aspiration from the perihepatic effusion, which confirmed the TPN ascites by a biochemical analysis.

The follow-up US examinations of these parenchymal lesions revealed complete resolution or a substantial reduction in size between 30 and 180 days of follow-up. Among the 18 parenchymal lesions seen in this study, 7 (38.9%) developed calcifications on follow-up US examinations. Among the 10 single parenchymal echogenic lesions, 9 resolved spontaneously without any clinical importance, whereas 1 patient died of other serious medical conditions. The small branching echogenic lesions disappeared, with atrophy of the lateral segment of the left liver lobe. All of these branching lesions were accompanied by left portal venous thrombosis. Among the 3 neonates who had large heterogeneous lesions with laceration and ascites, 1 died. The remaining 2 lesions had a substantial reduction in size, and both of them developed calcification.

Two of these patients died of other related serious medical conditions. One had intrauterine growth restriction, respiratory distress syndrome, and sepsis. There was severe clinical deterioration on day 8, and US revealed a large lobulated parenchymal collection at segments 8 and 4 with perihepatic and intra-abdominal ascites (Figure 8). Aspiration of the perihepatic fluid showed TPN material in a biochemical analysis. The umbilical venous catheter was removed, but the neonate died secondary to sepsis, intracranial hemorrhage, and multiorgan insufficiency on day 15. The other neonate, who died on day 9, had multiple major cardiac abnormalities. This neonate had a single echogenic

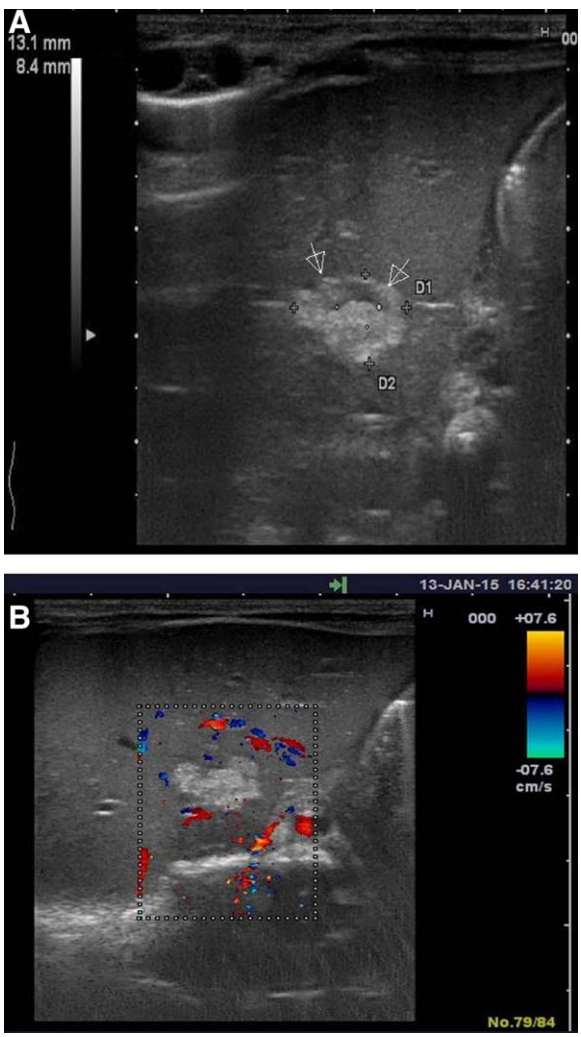
Figure 4. A 1650-g twin male neonate was born at 30 weeks' gestation due to premature membrane rupture. On day 1 of life, an umbilical venous catheter was inserted appropriately. Parenteral nutrition was started through the catheter. On day 6, a US examination (**A**) revealed a well-marginated, slightly heterogeneous hyperechoic parenchymal lesion at segment 4 of the left liver lobe adjacent to the middle hepatic vein (dimensions, 14 × 11 mm; white arrows). A radiograph (**B**) revealed that the tip of the catheter (white arrow) was at the level of the T11 vertebra, below the level of the diaphragm. Also, its position was quite more lateral than the expected. The catheter was removed. A follow-up US examination (**c**) revealed total resolution of the lesion within 3 months.



parenchymal liver lesion on a US examination on day 3 after catheter placement.

Among the 18 neonates with liver parenchymal lesions, the frequency of lesions was 7.4% (16 of 216) in preterm neonates, whereas it was 7.1% (2 of 28) in term neonates ($P > .1$). Among the 15 neonates with thrombosis in the left portal vein, the frequency of thrombosis was 6.5% (14 of 216) in preterm neonates, whereas it was 3.6% (1 of 28) in term neonates ($P > .1$). Among 49 neonates with air in the portal vein, the frequency of air was 20.4% (44 of 216) in preterm neonates, whereas it was 17.9 % (5 of 28) in term neonates ($P > .1$). There was no statistical significance in either group according to the gestational age.

Figure 5. A 2050-g female neonate was born at 32 weeks' gestation. The infant had multiple congenital cardiac abnormalities. Three days after umbilical venous catheter placement, a US examination (**A**) revealed a lobulated echogenic single parenchymal lesion (white arrows) with a hypoechoic area inside segment 4 of the left liver lobe. On color Doppler imaging (**B**), the echogenic lesion did not show any vascularity inside, even at low flow settings.



Discussion

The most frequent complication in our study was air in the portal venous system (20.1%). The air was seen anteriorly and peripherally in the nondependent portions of the liver. This peripheral gas distribution is related to the direction of blood flow into the liver. It is important to differentiate this complication from pneumobilia, which is centrally located because of the biliary anatomy and the direction of bile flow. Previous studies have shown that air in the portal venous system is associated with a high mortality rate (56%–90%).[17,18] However, the increased use of US and CT has allowed for the detection of an increased number of non–life-threatening causes of air in the portal venous system, especially those related to interventional procedures.[17–19] Actually, air in the portal venous system is very important in the neonatal period because it is a late sign of necrotizing enterocolitis, which is why this finding should be differentiated. In our study, 49 patients had air in the portal venous system without any evidence of necrotizing enterocolitis clinically or radiologically; follow-up US revealed that this problem spontaneously resolved in all 49 cases. Moreover, according to the gestational age, there was no statistical significance in the frequency of air in the portal venous system between preterm and term neonates.

In our study, portal venous thrombosis was noted in 6.1% of patients as another common liver-related complication. The thrombus resolved in 8 cases but was persistent in the remaining 7 cases; in these 7 cases, the thrombus either became an echogenic linear structure, or the lumen of the portal vein became obliterated without any flow signal on color Doppler US scans. Also, in these 7 cases that had persistent thrombosis in the left portal vein, the left lobe became smaller at follow-up compared with the previous scans. None of these patients developed portal hypertension. According to our experience, nonvisualization of the left portal vein as a normal variant in the later-childhood period may be a late sequela of unresolved left portal venous thrombosis related to umbilical venous catheterization. The results of this study show that left portal venous thrombosis related to umbilical venous catheterization may develop

during the neonatal period, but most of these thromboses resolve spontaneously or do not cause portal hypertension, which is similar to the results of previously published studies.[12,13] Moreover, according to the gestational age, there was no statistical significance in the frequency of thrombosis in the portal venous system between preterm and term infants.

Liver parenchymal lesions related to umbilical venous catheterization were seen in 7.4% of the patients in our study. However, according to the gestational age, there was no statistical significance in the frequency of liver parenchymal lesions between preterm and term infants. In this study, the most common type of parenchymal lesions were single nodular echogenic lesions, and the second most common were branching nodular echogenic lesions, followed by large irregular heterogeneous lesions with laceration and perihepatic fluid. First of all, single echogenic lesions may present as small hematomas. The umbilical venous catheter tip may cause a structural injury, including hepatic hematoma, due to perforation of an intrahepatic vascular wall.[7–9] Second, these single echogenic lesions may be an early manifestation of TPN-induced liver injury. The hypertonicity and high pH of the TPN infused through the catheter produces endothelial damage and leakage to the adjacent liver parenchyma. Over time, local liver parenchymal injury may turn into necrosis and laceration.[10,11,16] The US appearance of TPN-induced parenchymal injury may differ according to the stage, which can be assessed by the duration and length of TPN administration. Endothelial damage and any adjacent parenchymal injury may be the first stage, which may be seen as relatively homogeneous hyperechogenic lesions. Parenchymal necrosis may be the second stage, and it may be seen as a heterogeneous collection with relatively hypoechoic centers and hyperechoic rims.[4] Parenchymal laceration and perihepatic effusion may be the last stage. In our study, the most important but least frequent complication of umbilical venous catheterization was large heterogeneous parenchymal lesions with laceration and perihepatic fluid. All of these neonates had clinical deterioration, abdominal distension, and elevated liver enzyme levels, which is why early diagnosis and early treatment are crucial in this group.[4,5,8–10]

On the basis of the results of our study, we believe that branching small echogenic lesions are important because of their association with portal venous thrombosis. These small echogenic lesions may be small hematomas or may be formed secondary to a perfusion defect, likely representing small infarcted areas as a result of left portal venous thrombosis. We would like to point out that both of the aforementioned may be true. In addition, these small echogenic lesions may also be the result of a hemorrhagic transformation secondary to a perfusion defect. It is generally believed that a hemorrhagic transformation occurs as a result of preserved collateral perfusion from adjacent vessels or from reperfusion of infarcted tissues, which have weakened vessels, especially in stroke evaluations.[20,21]

Figure 6. A 1400-g female neonate was born at 31 weeks' gestation. The neonate had respiratory distress and surfactant deficiency disease. Two days after umbilical venous catheter placement, a US examination (**A**) revealed left portal venous thrombosis (black arrows) spreading through the distal branches. Note the multiple small echogenic lesions (**B**, white arrows) in a ramified manner around the thrombosed left portal vein branches.

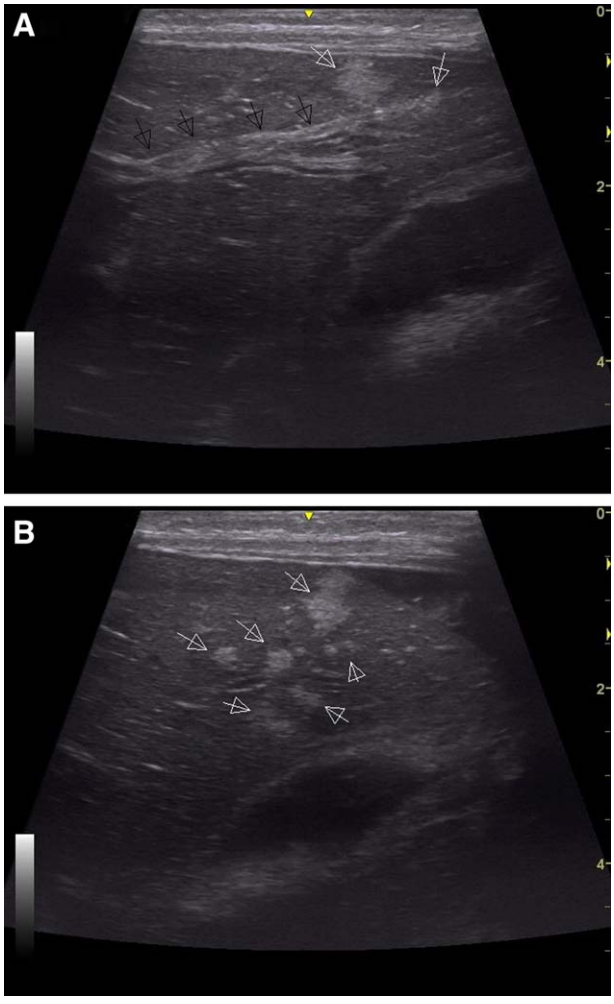
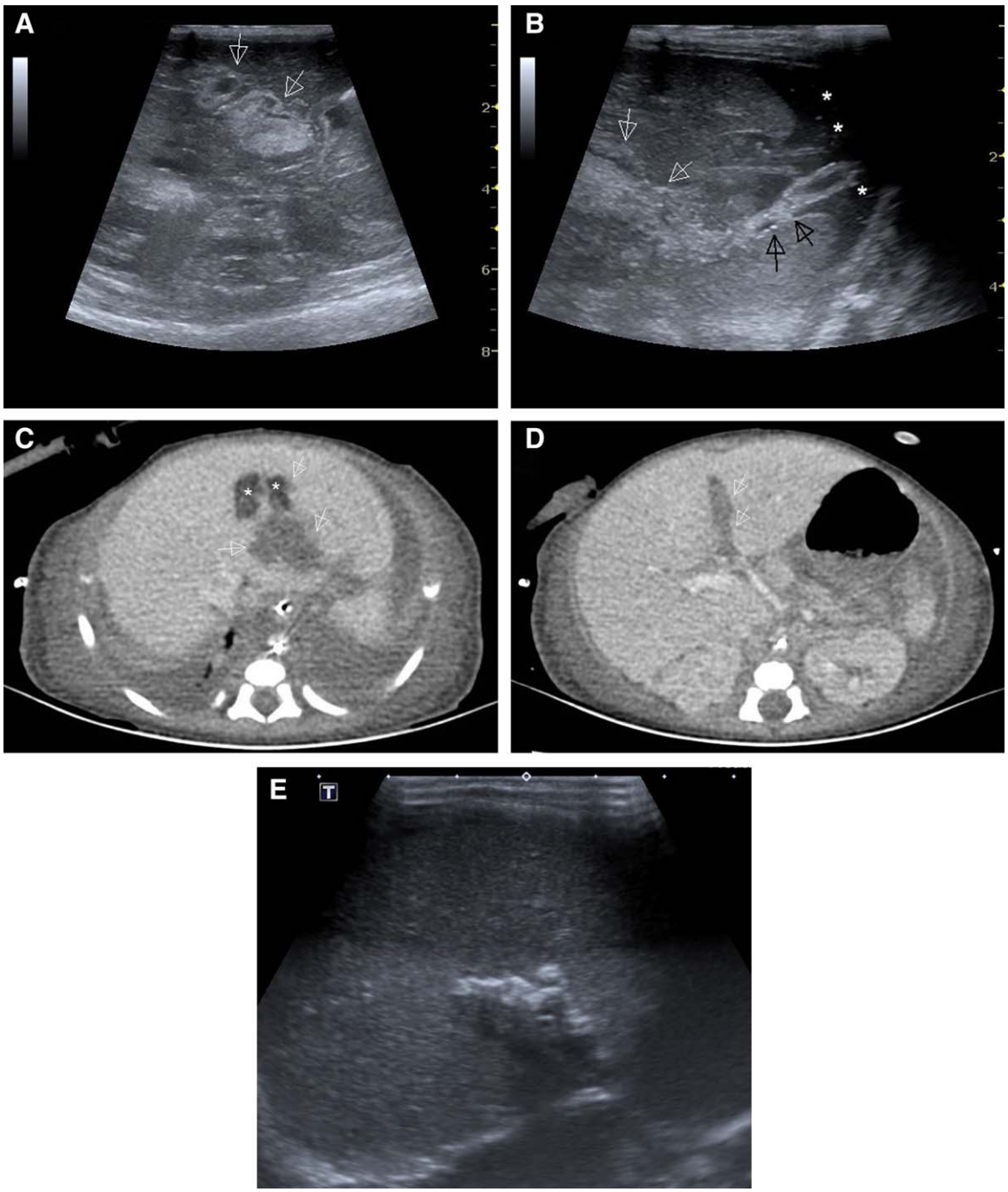


Figure 7. A 2300-g male neonate was born at 31 weeks' gestation due to hydrothorax. The mother had gestational diabetes. After birth, a chest tube was placed bilaterally for pleural effusion. The neonate had surfactant deficiency disease with persistent pulmonary hypertension and respiratory insufficiency; therefore, he was intubated. On day 1 of life, an umbilical venous catheter was inserted, and parenteral nutrition was started. On day 7, the neonate had abdominal distension; laboratory findings revealed elevated liver enzymes. A US examination (**A** and **B**) revealed areas of parenchymal necrosis (white arrows) and laceration (black arrows) along with perihepatic effusion (white asterisks) on the left lobe of the liver. Abdominal CT (**C**) also showed an area of liver necrosis (white arrows) in the left lobe. There was also fat density (white asterisks) inside the lesion, as well as pleural and perihepatic effusions. In addition, the left portal vein was thrombosed without any enhancement (**D**, white arrows). The neonate underwent US-guided aspiration from the perihepatic effusion, the TPN ascites were confirmed by a biochemical analysis. The umbilical venous catheter was removed, and a follow-up US examination revealed resolution of the parenchymal necrotic area with macrocalcifications (**E**).

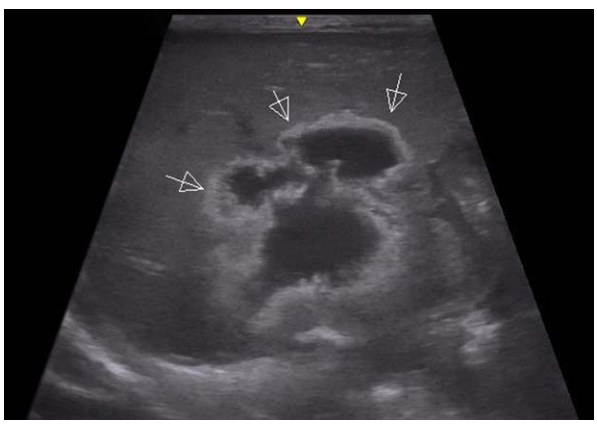


Ultrasound is an inexpensive, noninvasive, widely available, safe, fast, relatively painless, portable, and bedside-capable modality without ionizing radiation, which makes it ideal for premature neonates. It does not usually require sedation or injection of a contrast medium. However, the quality and interpretation of the images are highly dependent on the skill of the person doing the scan. A high-frequency linear array transducer is very helpful for assessing the liver parenchymal echo texture and evaluating the flow on color Doppler imaging in neonatal abdominal US examinations, which are substantially practical and valuable for screening and follow-up. In the NICU, proper positioning of an umbilical venous catheter is generally confirmed by radiography. In addition, US and echocardiography are other useful tools for determining the position of the catheter tip.[16,22] Furthermore, US can be used to detect and monitor any complications of umbilical venous catheterization in the follow up period. Hence, US-guided aspiration might be needed in diagnosis and emergent treatment of neonates with laceration and large effusion.

This study had some limitations. First, we did not evaluate the catheter tip position with US, and we did not correlate the hepatic complications according to the catheter tip position, which may be an interesting and supplementary subject for another article that could be done in the future. We included all patients who underwent routine umbilical venous catheter placement in our NICU. Finally, no histopathologic specimen of the liver tissue was obtained.

In conclusion, to our knowledge, our study is the first that determined the variety of hepatic complications caused by umbilical vein catheterization in the neonatal period. According to our study, air in the portal venous system was the most frequent complication, which was followed by left portal venous thrombosis and parenchymal lesions. The parenchymal lesions had 3 different patterns: single nodular echogenic lesions, branching small nodular echogenic lesions, and large irregular heterogeneous lesions with laceration and perihepatic fluid. Umbilical venous catheter–related liver lesions should be considered to have a high index of suspicion in the differential diagnosis of liver lesions in neonates with these catheters. Liver US is the best imaging modality for confirming the suspicion and following the resolution of parenchymal lesions. Therefore, radiologists must be familiar with the varied imaging characteristics of these hepatic complications. Awareness of the imaging findings of umbilical venous catheter–induced hepatic injury may lead to a straightforward diagnosis with US.

Figure 8. A 665-g male neonate was born at 28 weeks' gestation. The neonate had intrauterine growth restriction, respiratory distress syndrome, and sepsis. An umbilical venous catheter was inserted on day 1 of life. The tip of the catheter was placed at the level of the diaphragm. The child received parenteral nutrition and antibiotics. There was severe clinical deterioration on day 8, and a US examination revealed a large lobulated parenchymal collection at segments 8 and 4 with an echogenic border (white arrows). In addition, the infant had perihepatic and intra-abdominal ascites. Aspiration of the perihepatic fluid showed TPN material. The umbilical venous catheter was removed, and the infant died secondary to sepsis, intracranial hemorrhage, and multiorgan insufficiency on day 15.



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