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Case report

Liver laceration and hemoperitoneum in a premature neonate: a rare case of perinatal trauma

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Abstract

We present a case of a premature very low birth weight neonate who developed abdominal distension and acute anemia on his 2nd day of life, in which a liver laceration was diagnosed.

Neonatal hepatic injury is rare but frequently fatal. It usually happens due to perinatal trauma in complicated labor or delivery or when resuscitation maneuvers take place. It can result in a subcapsular hematoma of the liver but can also translate as a simple laceration. When the hepatic capsule ruptures, a hemoperitoneum usually develops. Umbilical vein catheterization, hepatic congestion and an increased bleeding tendency have also been associated with liver injury and hemorrhaging in the neonate. Clinical presentation can vary from abdominal distension to acute anemia or even hemorrhagic shock. Unspecific signs such as lethargy, tachypnea, feeding intolerance, jaundice and laboratory findings of thrombocytopenia, coagulation disturbances and hyperbilirubinemia can occur. Diagnosis may be difficult, and imaging methods are essential. A conservative approach to treatment is usually preferred, even in the case of a hemoperitoneum.

In the case described, there was a history of a difficult extraction and need for resuscitation maneuvers at birth. The abdominal radiograph showed a large liver, and ultrasound revealed a hepatic laceration and a hemoperitoneum. Treatment was supportive, consisting of blood components, expectant observation and strict avoidance of abdominal manipulation. Follow-up of the lesion through serial ultrasounds was performed, and the infant survived, recovering well. This case is one of the few in recent literature describing a good outcome in neonatal liver injury. Awareness of the possibility of its occurrence is important, especially in premature neonates, who are at particular risk.

Keywords

Liver injury, laceration, hemoperitoneum, perinatal trauma.

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Introduction

Liver injury in the neonate has been scarcely described in the literature. There is a considerable time gap between the first studies reporting autopsy findings and more recent case reports and small case series [1-3], mentioning newborn's survival. Incidence is not well known. According to post-mortem studies, it is estimated to be approximately 9.6% [4], with an incidence of subcapsular hematoma of the liver (SHL) between 1.2% and 15% [5, 6]. Other studies have also reported that one-quarter of SHL cases are preterm babies between 24 and 27 weeks of gestation, and that hemoperitoneum is present in 10% of those [6]. We found no data in the literature regarding the incidence of hepatic laceration alone.

Clinical presentation can be similar to other more common problems in the neonate (e.g., necrotizing enterocolitis, septic shock, surgical causes of abdominal distension), making it a challenging diagnosis.

Case report

A healthy pregnant woman, with negative serologies and an uneventful pregnancy thus far, was transferred from her local hospital at 24 weeks + 2 days of gestation due to preterm premature rupture of membranes. Antibiotics were started, and fetal monitoring was initiated. Pulmonary maturation was completed with 4 doses of dexamethasone. At 29 weeks of gestational age, a clinical picture of chorioamnionitis (maternal fever, leucocytosis with neutrophilia, and fetal tachycardia) became evident. The newborn was then delivered through an emergent C-section.

Due to breech presentation, extraction was remarkably difficult. The Apgar scores were 2, 5, 6 at 1, 5, and 10 minutes of life and birth weight was 1,345 g (very low birth weight – VLBW). Positive pressure ventilation (PPV) was initiated, and the baby was intubated within the 1st minute of life. Additional resuscitation maneuvers were needed, including chest compressions for 2 minutes and an epinephrine bolus, after which there was a positive response (HR > 100 bpm). The newborn was then mechanically ventilated. On physical examination, there were bruises on his inferior limbs.

First blood analysis revealed mixed acidosis (pH 6.96; pCO_2 69.2 mmHg; HCO_3 11.9 mEq/L; BE -14.5 mmol/L; lactate 9.6 mmol/L), a CK of 867 U/L, troponin T of 387 U/L and liver enzymes (AST and ALT) of 151 and 10 U/L. The radiograph findings were compatible with respiratory distress syndrome. A large liver (**Fig. 1**) was also apparent. The umbilical venous

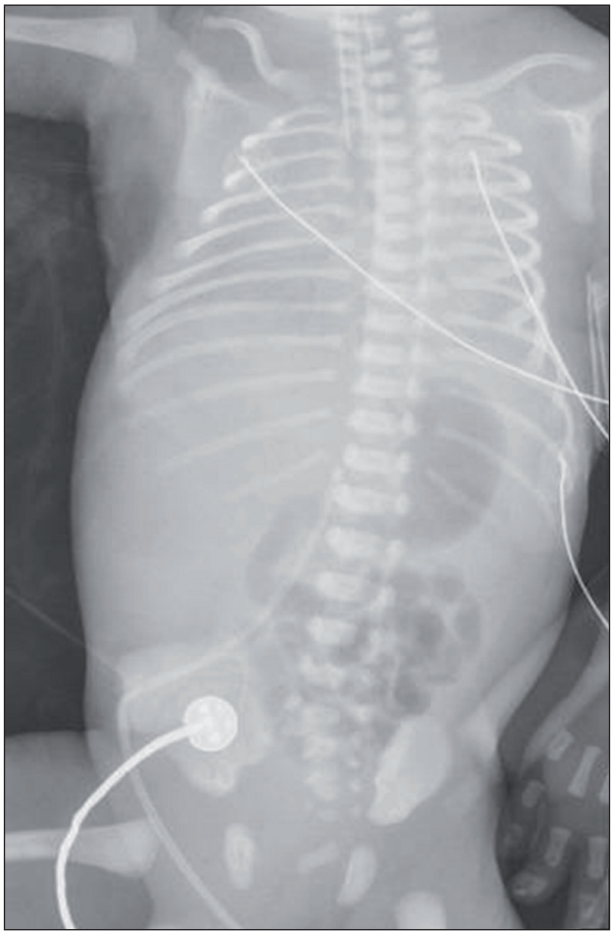

Figure 1. Abdominal radiograph on the 1st day of life.

catheter (UVC) tip was seen located at T11-12 level.

Surfactant was administered twice (total of 200 mg/kg). Antibiotics (ampicillin and gentamycin) were started. A single dose of vitamin K was given IM.

On his 2nd day of life, the newborn developed abdominal distension, despite successful meconium elimination and tolerance of trophic enteric feedings. A substantial decrease in hemoglobin (14.9 g/dL on day 1 to 11.6 g/dL on day 2), a fall in platelet counts (178,000/μL to 129,000/μL) and an elevation in CRP (from 1.1 mg/dL to 6.19 mg/dL) were observed. Blood cultures collected on the 1st day were negative.

The abdominal ultrasound revealed a fusiform hypoechogenic lesion in the right hepatic lobe (**Fig. 2**), suggesting a laceration. There was also evidence of hepatic capsule rupture, as well as free fluid in the peritoneal cavity.

Despite transfusion of 10 mL/kg of packed red blood cells (RBC) on his 2nd day of life, hemoglobin level (minimum of 9 g/dL on day 4) and platelet counts (minimum of 106,000/μL) continued to decrease. Coagulation tests were within the normal range. The abdominal radiograph (**Fig. 3**) and ultrasound performed on the 4th day showed increased peritoneal free fluid, compatible with a hemoperitoneum. Transfontanellar ultrasound showed absence of intraventricular hemorrhage.

Management was conservative, consisting of expectant observation and transfusional therapy with packed RBC (10 mL/kg) and fresh frozen plasma (10 mL/kg), both given on the 4th day. Furthermore, following the surgical team's recommendations, the baby was consistently kept in supine position from then on, and strict avoidance of abdominal manipulation was put into practice. The baby remained hemodynamically stable and with no evidence of new blood loss throughout the rest of his stay in the Neonatal Intensive Care Unit (NICU).

Upon follow-up on the 35th day, the abdominal ultrasound showed complete absence of fluid, and a calcified lesion in the same location of the previous laceration (**Fig. 4**). At 86 days, a scar of the liver parenchyma was apparent. (**Fig. 5**)

The baby was discharged from hospital care on his 89th day of life. To this date, he remains well, albeit the sequelae of his prematurity, such as chronic lung disease and a mild global developmental delay.

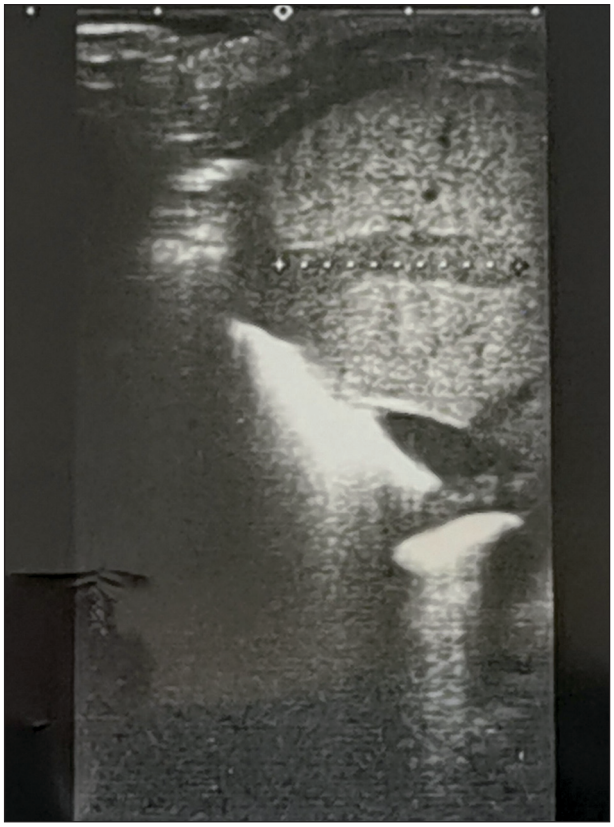

Figure 2. Abdominal ultrasound on the 2nd day of life. A focal fusiform hypoechogenic lesion (laceration), as well as the presence of perihepatic free fluid, are evident.

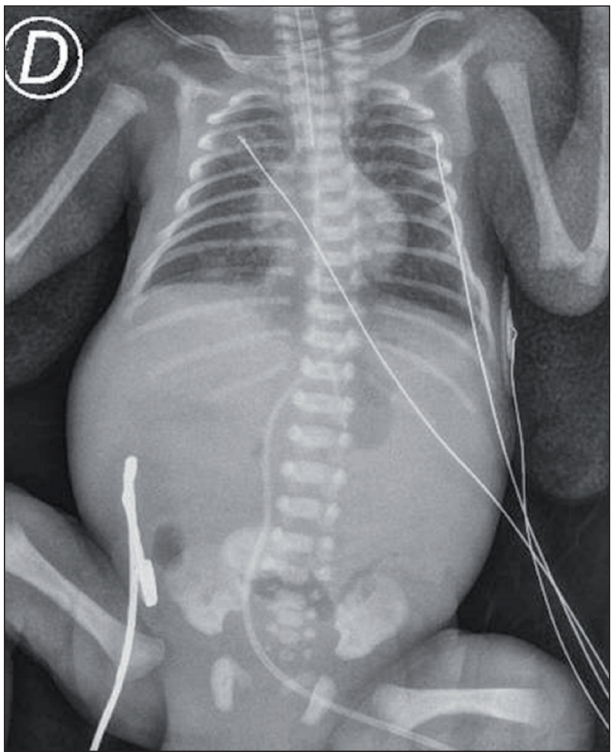

Figure 3. Abdominal radiograph on the 4th day of life. Hardly any bowel gas was visualized, suggesting the presence of abdominal free fluid.

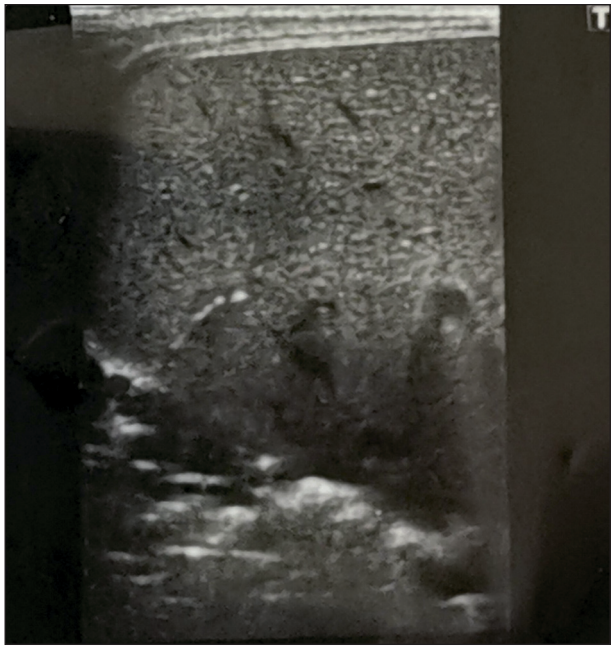

Figure 4. Abdominal ultrasound at 35 days. In the same topography of the previous laceration, there is a hyperechogenic horizontal line, suggestive of incipient calcification.

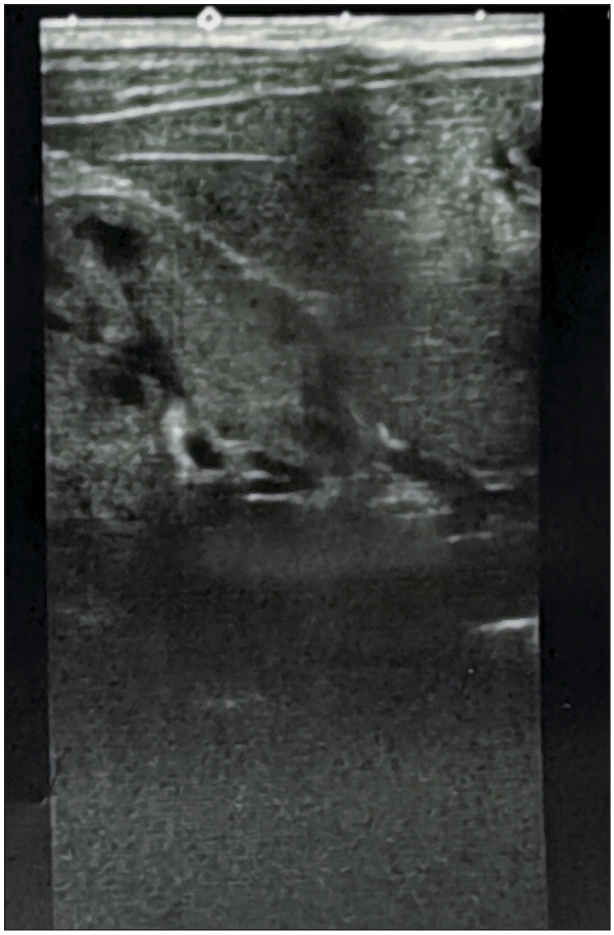

Figure 5. Abdominal ultrasound at 86 days. A hyperechogenic line is still visible in the same location, suggesting scarring of the liver parenchyma at that site.

Discussion

Intra-abdominal injuries at birth are thought to be rare and involve rupturing and/or hemorrhaging of the liver, spleen or adrenal glands. Of these, the most common is liver injury [7], with the frequent involvement of the right hepatic lobe due to its anatomic situation and larger volume.

This is more likely to happen in complicated labor or delivery, in premature neonates, breech presentation, and when there is a need for resuscitation maneuvers [4, 7]. Moreover, hepatic enlargement due to liver congestion secondary to assisted ventilation [5], trauma associated with umbilical vein catheterization [1], and hemorrhage in consequence of an increased bleeding tendency [3, 5] have also been implicated in the pathogenesis.

As clinical findings, abdominal distension [1, 3-5] is almost always present, rarely with a palpable mass [3]. Also, a sudden fall in hematocrit [1, 3-5], and in more severe cases, a picture of hemorrhagic shock [5, 7] are frequent, with the tendency to manifest within the first 3 to 5 days of life [5]. An SHL can present up to 7 days after birth, since it is prone to first increase in size before rupture [4]. Non-specific signs, such as jaundice, lethargy or irritability, feeding intolerance and tachypnea [3], might also be found.

Apart from acute anemia, thrombocytopenia [1, 2, 5], elevation of liver enzymes and bilirubin levels [2, 3], and coagulation disturbances can sometimes be observed as laboratory findings [3].

Serial hematocrit determinations [7] and careful screening of shock signs may help to first raise clinical suspicion of hepatic hemorrhaging. Image methods play a crucial role in accurate diagnosis. Abdominal radiography may show enlargement of the liver or an abnormal course of a UVC [4]. Ultrasonography can directly demonstrate hepatic hemorrhage, as well as the presence of intraperitoneal fluid, namely a hemoperitoneum [4]. It can also rule out bleeding from other abdominal organs [5].

Management of neonatal liver injury tends to be conservative [2, 8], consisting of blood transfusions, correction of coagulopathies and avoiding excessive handling of the baby [3]. Surgical intervention is usually reserved for cases of hemoperitoneum, but only performed under particular circumstances such as clinical worsening, hemodynamic instability or evidence

of new bleeding after an initial well-conducted resuscitation.

There were several risk factors for hepatic injury in the neonate. Rapid diagnosis was only possible due to high clinical suspicion and the precision of our ultrasonographers. Ultrasound was also useful for monitoring the lesion's evolution. The supportive management approach proved to be effective, even in the presence of hemoperitoneum. Maintaining the baby in supine position to minimize handling might have compromised adequate neurodevelopmental stimulation for some time, but it was probably essential in preventing the injury's aggravation.

This case highlights the importance of rapid recognition and adequate management of neonatal hepatic injury. Although potentially fatal, a better outcome is possible if there is awareness of the possibility of its existence in a neonate presenting with abdominal distension and acute anemia with or without early signs of shock.

Declaration of interest

The Authors declare that there is no conflict of interest. The Authors declare that they did not receive any funding, neither from private or public entities, in the making of the present work.

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