SWISS SOCIETY OF NEONATOLOGY

Acute abdomen in a preterm infant: appearances can be deceptive



January 2016

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Title figure: Source: Thomas M. Berger This baby of a 36-year-old healthy mother was born by Caesarian section at 28 weeks of gestation for suspected chorioamnionitis. Her birth weight was 1160 g (P37) and Apgar scores were 6, 9, and 9 at 1, 5, and 10 minutes, respectively. Maternal antibiotics had been given after premature rupture of membranes nine days prior to delivery. At birth, she presented with respiratory distress and was intubated 20 minutes after delivery due to an oxygen requirement of up to 60%. Umbilical venous and arterial catheters (UVC, UAC) were inserted 30 minutes after birth and 10% intravenous glucose solution was commenced along with the first dose of amoxicillin and amikacin.

A chest X-ray following endotracheal intubation showed diffuse, fine granular global opacities consistent with respiratory distress syndrome of the newborn (Fig. 1). The endotracheal tube was positioned slightly high, the arterial line was deemed to be in a correct high position (i.e., between thoracic vertebrae 6 – 9) and the venous line was thought to be in an indifferent low position, apparently just below the liver. Response to intra-tracheal surfactant administration was excellent with an FiO₂ quickly coming down to 0.21 and she was extubated to nasal continuous positive airway pressure (nCPAP) at the end of day one of life. Physical examination at that time was entirely normal.

CASE REPORT



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Fig. 1

Babygram one hour after birth following intubation and placement of umbilical catheters: poorly aerated lung consistent with hyaline membrane disease (UVC, umbilical venous catheter; UAC, umbilical artery catheter). Whereas the UAC was removed after extubation, the UVC was kept in place. The first three days of life were otherwise uneventful and the baby tolerated introduction of enteral feeds very well.

On day four of life, the girl developed signifiabdominal distension with red discoloracant tion and induration. She did not show any other gastrointestinal abnormalities, specifically, she did not have any gastric residuals, there was no vomiting, and she had normal bowel movements and normal bowel sounds. Her general appearance was remarkably well and her vital signs remained stable. Although enteral feedings were stopped and complete parenteral nutrition was administered, abdominal distension increased and oxygen requirements on CPAP increased up to 60%. Abdominal X-ray studies revealed a distended abdomen with central intestinal gas distribution as an indirect sign of ascites, which was verified by ultrasound imaging of the abdomen. No findings suggestive of necrotizing enterocolitis or focal intestinal perforation were noticed (Fig. 2 - 4).

Whole blood count and C-reactive protein concentration were within normal limits, and the blood culture remained sterile. Nevertheless, on day five of life, due to gross abdominal distension, members of pediatric surgery and neonatology decided to proceed with explorative laparotomy. When the abdominal cavity was opened, 150 ml of red, milky fluid was discovered and evacuated (Fig. 5). The liver showed a small, nonbleeding laceration from the UVC (Fig. 6). The UVC was subsequently removed. No further abnormalities were identified. Small and large intestines were intact and well perfused.

The baby recovered very well after surgery and reintroduction of enteral feedings was well tolerated. Repeat ultrasound of the abdomen one day after surgery revealed a liver hematoma of about 5 cm³ in the region where the tip of the UVC had been placed. She did not have clinical signs of anemia or liver dysfunction and her hemoglobin level after surgery was 149 g/l. The liver hematoma resolved spontaneously without further complications.



Fig. 2

Abdominal X-ray on day of life 4 (90 hours after birth): midline position of intestines, suggestive of ascites (UVC, umbilical venous catheter).



Fig. 3

Abdominal X-ray on day of life 4 in left lateral decubitus position (96 hours after birth): no evidence of pneumoperitoneum (UVC, umbilical venous catheter).





Abdominal X-ray on day of life 4 (96 hours after birth): midline position of intestines, suggestive of ascites (UVC, umbilical venous catheter). Fig. 4



On day of life 5, an explorative laparotomy was performed: when the abdominal cavity was opened, 150 ml of red, milky fluid was discovered and evacuated.



On day of life 5, an explorative laparotomy was performed: a small non-bleeding hepatic laceration most likely related to the UVC was found. FIG. 6

DISCUSSION

The insertion of a UVC is a common and potentially lifesaving procedure in the neonatal intensive care unit, but obviously not without risks. Intraperitoneal extravasation of parenteral nutrition has been documented as one of several complications associated with malpositioned UVCs (1 - 4). Most children reported in the literature showed abdominal distension and the tip of the UVC was always located below the diaphragm and superimposed over the liver on a.p. radiographs. Other complications of UVCs include infection, thrombosis, vascular injury, pericardial effusion, cardiac tamponade and hepatic injury (4 - 7).

In our case, a 3.5 F double-lumen polyurethane catheter had been inserted into the umbilical vein without any difficulty. The tip of the UVC was noted to be positioned subdiaphragmatically at the level of the tenth thoracic vertebra on a.p. radiography. The ideal position of the UVC tip is recommended to be in the inferior vena cava just below the level of the diaphragm corresponding to the eighth or ninth thoracic vertebral body (8). The literature shows that two-thirds of UVCs located at the tenth thoracic vertebral body on radiography are positioned within the liver on ultrasound (9). The absence of hemodynamic instability and the delayed time course of onset of symptoms in our case suggest that hepatic necrosis rather than primary perforation of the blood vessel might have led to disruption of the liver capsule. Risk factors of hepatic necrosis identified in prior reports include the administration of hypertonic fluids and a long dwell time of the catheter (2). In our case, 100 ml of the parenteral nutrition solution contained 10.5% glucose, 3 mmol sodium acetate, 1.5 mmol potassium chloride, and 2 g of amino acids. Dwell time on presentation of the first clinical symptoms was four days, which is not considered exceptionally long for such a catheter. An alternative explanation for the origin of the injury relates to the UVC being used for blood withdrawal. The line was used three times for this purpose in the first three days of life. Although this is a common procedure, it is conceivable that the negative pressure exhibited through this measure had caused or aggravated the laceration.

Ultrasonography holds promise as a more accurate method to confirm catheter tip position compared to radiography (8, 9). Ultrasonography has been successfully used to quickly confirm suspected diagnosis of hepatic erosion by UVC (10). Intraparenchymal liver lesions (hypoechoic to hyperechoic) in the expected course of the umbilical vein and ductus venosus are typical in this context (10). Ascites caused by intraperitoneal leakage of parenteral nutrition solution can be simple but more complex ascites including debris or septations may develop over time. In our case, the hepatic lesion was only identified retrospectively and the ascites was simple probably due to the relatively short dwell time of the UVC. Treatment of the liver laceration and ascites caused by UVC primarily consists of removal of the UVC, which usually results in rapid resolution of the ascites. Therapeutic paracentesis can be performed in cases of severe cardiorespiratory problems.

In conclusion, it is of utmost importance to ensure proper positioning of the UVC after insertion and to keep in mind that with each day of its use, the risk of complications increases. The advantages of a central vascular line need to be carefully weighed against the risks associated with such a catheter.

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