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A case of delayed hemorrhage of a subcapsular liver hematoma in a neonate

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A subcapsular liver hematoma (SLH) is a relatively common lesion in fetuses and neonates. Although an SLH ruptures rarely, it may be life threatening. We report on a term neonate with a delayed rupture of an SLH that occurred on day 7 of life. The infant had been resuscitated with intubation, positive pressure ventilation, and chest compression at birth because of meconium-associated perinatal depression. The SLH was diagnosed by abdominal ultrasonography and paracentesis, and the ruptured SLH was treated operatively. After intensive medical and surgical management, the infant was discharged healthy on day 27 of life. A newborn infant presenting with the sudden onset of extreme shock and pallor associated with abdominal distension should undergo differential diagnosis for SLH and a clinical evaluation concurrent with fluid resuscitation and timely surgery. **(Korean J Pediatr 2008;51:89-92)**

Key Words : Subcapsular liver hematoma (SLH), Neonate

Introduction

An SLH is the accumulation of blood in the region of the hepatic capsule. A variety of etiologies for SLH in neonates has been implicated[17]. Most cases of SLH involve a small amount of hemorrhage so that the diagnosis of SLH is made at autopsy rather than by clinical or laboratory evaluation. Rupture of an SLH results in extensive intra-abdominal bleeding, which is associated with a high mortality rate, and early recognition and stabilization are critical.

In this report, we describe a term neonate who had meconium-associated perinatal depression and who was resuscitated with intubation, positive pressure ventilation, and cardiac massage at birth. The infant developed a ruptured SLH with associated hemoperitoneum, which was treated successfully.

Case Report

The patient was born weighing 3,600 g by emergency cesarean section after failure of vaginal delivery at the local hospital in week 41 of gestation. Immediately after birth, she was depressed and received endotracheal intubation and suction because the amniotic fluid was stained with thick meconium. After suctioning of the meconium-stained fluid, she was extubated. Her Apgar scores at 1 and 5 minutes were 3 and 8, respectively. However, 30 minutes later, the patient suddenly presented with bradycardia and desaturation, and she received neonatal resuscitation including intubation, positive pressure ventilation, and cardiac massage. The patient was transported to our hospital for further intensive care.

On admission, the infant exhibited total pallor, weak self-respiration, and moderate substernal retraction. Her body temperature was 36.5℃, heart rate was 140 beats/min, blood pressure was 60/29 mmHg, and percutaneous oxygen saturation was 66%. Arterial blood gas analysis revealed severe metabolic acidosis (pH 7.0; pCO_2, 28 mmHg; HCO_3, 7.5 mmol/L; base excess, 23.1 mmol/L). She was treated with mechanical ventilation, continuous infusion of vasopressors (dopamine, dobutamine), antibiotics (ampicillin, cefotaxime), and sedation (fentanyl). She was catheterized through the umbilical artery and vein. Her condition improved steadily. On day 5 of life, she initiated feeding of milk, and extubation was performed on day 6. On day 7 of life, abrupt-onset tachycardia, tachypnea, fever (37.9℃), chest retraction,

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abdominal distension, low urine output, and bile-colored drainage through the orogastric tube were noted. Despite NPO, gastric decompression through the orogastric tube, and intravenous antibiotics, she developed progressive abdominal distension and an ill-looking appearance (Fig. 1). Abdominal ultrasonography revealed a large amount of ascites in the anterior aspect of the left lobe of the liver, which appeared as hyperlucent with multiseptal formation (Fig. 2). After aspiration of 70 cc of bloody and turbid fluid by diagnostic abdominal paracentesis, the infant had an emergency laparotomy. Surgery revealed a hematoma and an old blood clot anterior to the lateral segment of the left lobe of the liver. A large amount of blood was found in the peritoneum, and the peritoneum was irrigated and packed to control the bleeding (primary anticoagulant therapy). We treated her postoperatively with conservative management including a 10-day course of antibiotics comprising vancomycin, amikin, and metronidazole. She started to feed on postoperative day 4 and completed fully on postoperative day 13 (day 26 of life). On day 27 of life, she was discharged healthy. She was followed up until 36 months of age, and she now displays normal growth and development.

Discussion

SLH is a relatively common lesion in neonates. Although rare beyond the perinatal period[1], SLH may be life threatening. First reported in a neonate in 1870 by Hodge[2], the incidence of SLH in the newborn has been reported as 5% to 9.6%[3]. French and Waldstein found a 15% incidence of SLH in 783 autopsies; 16% had a large SLH, and 54% had a small hemorrhage[3]. However, the true incidence is difficult to determine because most cases are diagnosed at postmortem rather than clinically[4-6].

A variety of etiologies for SLH has been implicated, and trauma is considered as the main etiology of SLH[1, 2, 5-10]. The liver is the abdominal organ injured most frequently during the birth process, and the autopsy incidence of liver injury varies from 0.9% to 9.6%[8]. Infants with macrosomia, hydrops fetalis, or breech delivery, and infants of diabetic mothers are at risk[8]. Trauma to the liver results more often in subcapsular hematoma than actual laceration of the liver, and a capsular rupture with hemoperitoneum can follow[3, 7, 8, 11]. Additional predisposing factors of SLH may include hypoxia, asphyxia, low birth weight, coagulopathies, maternal

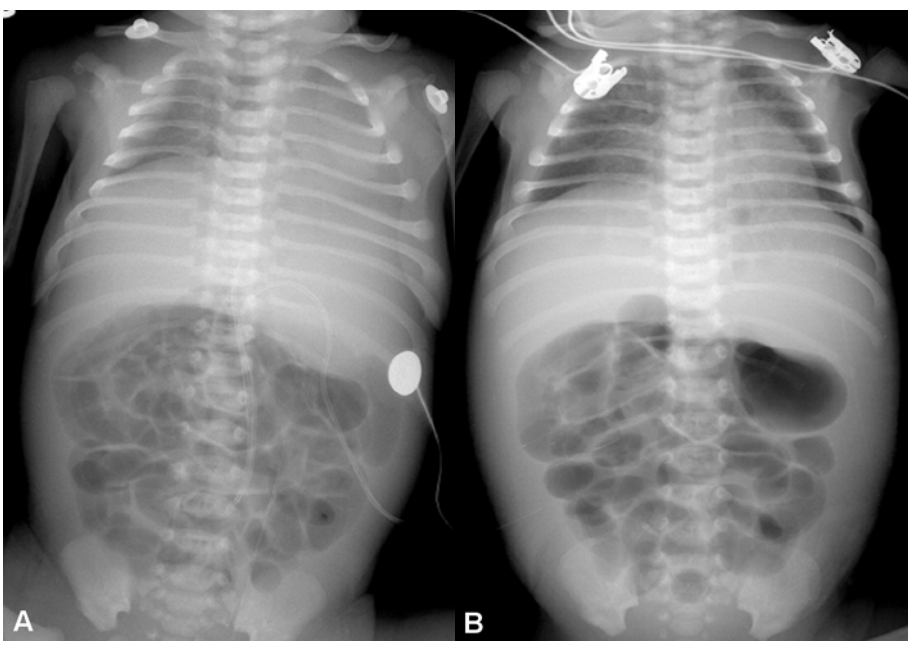

Fig. 1. (A) Plain film of the abdomen on day 7 of life, 30 min after hypovolemic symptoms appeared. The film shows moderate ileus of the small intestine and hepatomegaly. An echocardiographic monitoring lead and umbilical venous catheter are visible. (B) Plain film of the abdomen 18 hours after the appearance of hypovolemic symptoms demonstrates the remaining ileus of the small intestine and aggravating ascites. The umbilical venous catheter was removed.

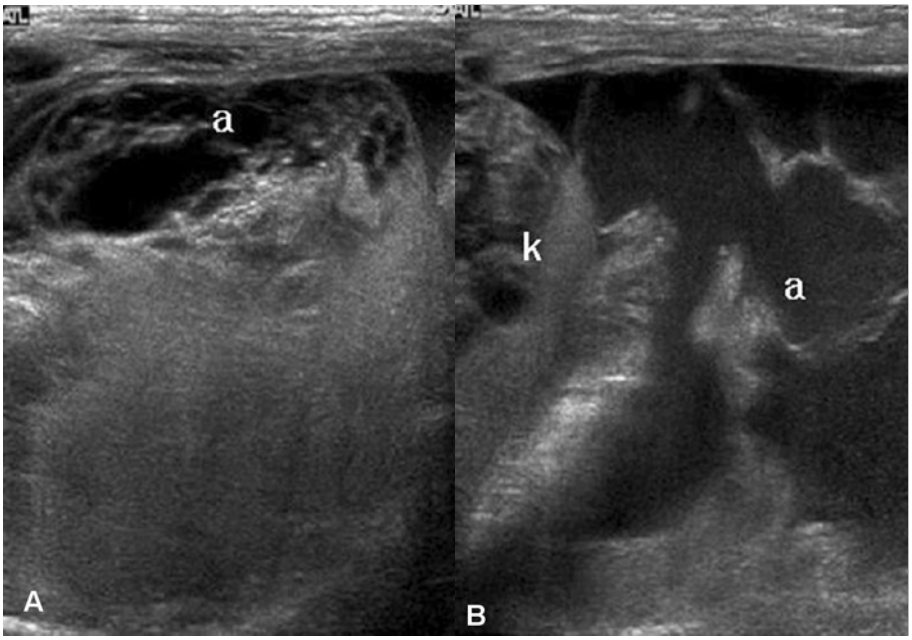

Fig. 2. (A) Ultrasonography of the liver on day 7 of life demonstrates a hypoechogenic, large amount of ascites (a) anterior to the left lobe with a multiseptal formation and (B) complicated ascites (a) inferior to the right kidney (k).

disease, placental lesions, and physical manipulation such as external cardiac massage, intubation, and umbilical venous catheter placement[1, 2, 5-10]. Side effects of drug taken by the mother (e.g., salicylic acid, anticonvulsants) may contribute in a minority of cases1). Singer et al. examined 755 perinatal autopsies and found SLH in 52 (6.9%), including 31 stillborn fetuses and 32 liveborn infants[1]. Sepsis was associated with 62% of these cases and Group B streptococcus infection was the most common cause of sepsis. Interestingly, cerebral germinal matrix hemorrhages were present in 35% of these cases with hepatic hematoma[1]. French and Waldstein reported that neonates with SLH tend to be premature male infants with low Apgar scores, a maternal gestational history complicated by chronic problems, and some difficulties during labor and delivery, especially placenta abruptio[5]. Furthermore, physical manipulations including umbilical venous catheter placement, tracheal intubation, and chest tube placement are typically seen in the affected neonates[5]. The infant in our report was a term infant and had a history of tracheal intubation and cardiac massage on the first day of life because of meconium aspiration syndrome. She underwent mechanical ventilation and umbilical vein catheterization. We suspect that resuscitation or umbilical vein catheterization contributed to SLH in our patient.

Infants with an SLH usually appear asymptomatic for the first 13 days of life[8, 9]. A ruptured SLH might result in blood loss of 50 mL to 500 mL[3]. The incidence of hemoperitoneum is 10% among the infants with SLH[5]. Nonspecific signs related to loss of blood into the hematoma may appear early; these include poor feeding, listlessness, pallor, jaundice, tachypnea and tachycardia, and occasionally a palpable right upper quadrant abdominal mass[8, 9]. Systemic nonspecific symptoms are followed by sudden circulatory collapse when the hematoma ruptures through the capsule and blood enters the peritoneal cavity[8]. The abdomen may become distended, rigid, and dull to percussion, occasionally with a bluish discoloration of the overlying skin, which may extend over the scrotum in male infants[8, 9].

The useful diagnostic tools for SLH include plain films, ultrasonography, and CT of the abdomen. Abdominal plain films may suggest liver enlargement, and the abnormal course of the nasogastric tube or umbilical venous catheter, or uniform opacity of the peritoneal cavity may indicate free peritoneal fluid[8]. Although paracentesis can confirm whether free blood is in the peritoneal cavity, ultrasonography offers a noninvasive method of diagnosis[8, 11, 12]. Ultrasonography demonstrates a hypoechoic mass that may displace and occupy most of the liver[9-12] and discloses the SLH as an echolucent band between the hepatic parenchyma and the diaphragm[11]. Our patient exhibited, in addition to the

signs of SLH, a large amount of ascites anterior to the left lobe, which appeared hyperlucent with multiseptal formation, possibly occurring secondary to the ruptured SLH. Serial ultrasonography should be employed to confirm the diagnosis, identify the etiology of the SLH, confirm the resolution of the SLH, help exclude the possibility of malignant tumors, and decrease the number of unnecessary investiations[12]. A CT scan may assist in establishing the diagnosis of SLH[8].

Immediate medical management comprising blood transfusion to correct the anemia and treatment of shock and the coagulation disorders are essential[4, 8, 9, 11]. The application of other resuscitative, pharmacological, and ventilatory management procedures depends on the patient's condition. Unnecessary external manipulations should be avoided in these patients. Timely surgical intervention for infants who fail to respond to conservative management may improve the prognosis of the ruptured SLH[4, 6, 8, 10]. Once coagulopathies have been corrected, the patient should be treated with laparotomy to suture the liver laceration and maintain hepatic hemostasis with a collagen hemostat or fibrin glue[13].

In summary, SLH should be suspected in neonates with hypovolemia or anemia or with progressive hepatomegaly, and abdominal ultrasonography should be the study method of choice[12]. We present a newborn patient with a ruptured SLH, which was thought to result from tracheal intubation, cardiac massage, or umbilical venous catheterization.

한 글 요 약

신생아에서 발생한 대량 피막하 간 혈종의 지연성 파열 1례

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신생아의 피막하 간 혈종은 다양한 원인에 의해서 유발되는데 대부분은 출혈의 양이 적어 임상적으로 발견되기 보다는 부검시 발견되는 경우가 많다. 그러나 출혈의 양이 많아 혈종을 싸고 있는 피막이 파열되고 잇따른 출혈이 발생할 경우에는 창백, 혈관 허탈, 빈혈, 복부팽만, 저산소증, 산혈증 등의 증상을 보이므로 조기에 의심하여 정확한 진단을 내리고 적절한 수술적 치료를 실시하지 않으면 사망률이 높다. 저자들은 태변 흡입후 신생아 가사 소견을 보여 심폐소생술을 실시한 신생아에서 출생후 7일에 발현된 대량 피막하 간 혈종 파열 1례를 경험하였기에 보고하는 바이다.

References

1) Singer DB, Neave C, Oyer CE, Pinar H. Hepatic subcapsular hematomas in fetuses and neonatal infants. Pediatr Dev Pathol 1999;2:215-20.
2) Cywes S. Hemoperitoneum in the newborn. S Afr Med J 1967;41:1063-73.
3) Parker LA. Part 2: Birth trauma: injuries to the intraabdominal organs, peripheral nerves and skeletal system. Advances in Neonatal care 2006;6:7-14.
4) Ryan CA, Finer NN. Subcapsular hematoma of the liver in infants of very low birth weight. Can Med Assoc J 1987;136:1265-9.
5) French CE, Waldstein G. Subcapsular hemorrhage of the liver in the newborn. Pediatrics 1982;69:204-8.
6) Shankaran S, Elias E, Ilagan N. Subcapsular hemorrhage of the liver in the very low birth weight neonate. Acta Pediatr Scand 1991;80:616-9.
7) Emma F, Smith J, Moerman PH, Devlieger H, Vanhole C, Zegher F, et al. Subcapsular hemorrhage of the liver and hemoperitoneum in premature infants: report of 4 cases. Eur J Obstet Gynecol Reprod Biol 1992;44:161-4.
8) Fanaroff AA, Martin RJ. Neonatal perinatal medicine Disease of the fetus and infant. 6th ed. St. Louis: Mosby 1997:445-6.
9) Foss K. A case report of a low-birth-weight infant with a subcapsular liver hematoma and spontaneous bowel perforation. Adv Neonatal Care 2004;4:67-78.
10) Mouzard A, Cohen JY, Huault G. Ultrasonography in subcapsular hematomas of the liver in the newborn. Pediatrics 1982;70:1016.
11) Zorzi C, Perale R, Benini F, Angonese I. Diagnostic value of ultrasonography in neonatal liver rupture. Pediatr Radiol 1986;16:425-6.
12) Mouratidis B, Antonio G. Sonographic diagnosis of subcapsular liver hematoma mimicking tumor in a neonate. J Clin Ultrasound 2000;28:53-7.
13) Blocker SH, Ternberg JL. Traumatic liver laceration in the newborn: repair with fibrin glue. J Pediatr Surg 1986;21:369-71 •