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Citation	Hong Kong Medical Journal, 2006, v. 12 n. 1, p. 68-70
Issued Date	2006
URL	http://hdl.handle.net/10722/45493
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Splenic rupture in a premature neonate

早產嬰兒脾臟破裂

Splenic injuries are very rare in neonates. We report a case of splenic injury in a premature neonate, highlighting the importance of a high-index suspicion in early recognition of this rare but potentially fatal intra-abdominal injury. We also review the literature on possible aetiologies and mechanism of splenic injury, as well as its management. This is the first reported case of a very low-birth-weight neonate with splenic rupture who survived with intact neurology.

新生嬰兒出現脾臟破裂極為罕見。我們報告一宗早產嬰兒脾臟破裂病例，強調提高對這種罕見但致命的內腹部受傷的警覺性和及早診斷的重要性。我們亦翻查文獻以了解可能的病源、脾臟受傷的機理和治療方法。這是第一宗記載出生時體重過輕及脾臟破裂，但最後可以有完整神經系統的嬰兒病例。

Introduction

Splenic injuries are very rare among newborns, particularly low-birth-weight premature neonates. Acute haemorrhage from a traumatised spleen can be life-threatening. The initial signs and symptoms can be relatively vague and non-specific. Unfortunately, late recognition often results in fatality.¹ With advances in intensive care and surgical technique, more cases of neonatal survival following splenic injuries, including successful salvage of a ruptured spleen, have been reported. We present a case of splenic injury in a premature neonate of very low birth weight, along with a review of the literature.

Case report

The neonate presented in April 2004 was born at 27 weeks' gestation and had a birth weight of 845 g. His mother had an obstetric history of lower segment caesarean section (LSCS) at term for cephalopelvic disproportion 4 years previously and a preterm delivery at 26 weeks' gestation with neonatal death 2 years previously. During this pregnancy, cervical cerclage was performed at 16 weeks' gestation following transvaginal ultrasound examination findings of cervical incompetence. The cervix became incompetent again at 22 weeks' gestation, with gradual funneling of the cervical canal down to the external cervical os despite the in-situ cervical cerclage. Premature rupture of membranes occurred at 26 weeks' gestation. The mother was given prenatal steroids (two doses of intramuscular betamethasone 12 mg every 12 hours) and antibiotic prophylaxis (oral erythromycin 250 mg 4 times daily). Spontaneous onset of preterm labour occurred at 27 weeks' gestation. The foetus was in footling breech presentation, with both legs prolapsed out through the cervical os into the vagina. An emergency LSCS was performed. The baby was delivered without difficulty and Apgar scores noted were 6 (at 1 minute), 6 (at 5 minutes), and 8 (at 10 minutes). Cord blood pH was 7.36 (arterial) and 7.43 (venous). No external bruises were seen.

The baby developed respiratory distress syndrome soon after birth and was treated with surfactant replacement. He was also treated with antibiotics for perinatal settings of clinical sepsis. He was placed on continuous blood pressure monitoring from the time of birth.

The baby remained relatively stable until 20 hours of life, when apnoea and sudden hypotension was noted. Physical examination showed pallor, poor

Key words:

Hemoperitoneum;
Infant, premature;
Infant, very low birth weight;
Splenic rupture

關鍵詞：

腹腔積血；
 嬰兒，早產的；
 嬰兒，體重過輕；
 脾臟破裂

Hong Kong Med J 2006;12:68-70

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perfusion, and apnoea. Haemoglobin level decreased from 175 g/L to 64 g/L. Blood gas analysis showed a metabolic acidosis. He was resuscitated with a transfusion of packed cells. An urgent bedside ultrasound of the brain indicated bilateral grade II intraventricular haemorrhage, which did not explain the severity of his anaemia. Mild abdominal distension was evident and a concealed intra-abdominal haemorrhage was suspected. The neonate was considered too ill to safely transfer to the computed tomography (CT) suite. A paediatric surgeon was urgently consulted. On the basis of the clinical and laboratory findings, a diagnosis of intra-abdominal bleeding was made. Further imaging studies, such as a bedside abdominal ultrasound or abdominal paracentesis, were not performed to avoid delaying necessary treatment. Emergency laparotomy revealed a 50 mL blood clot in the peritoneal cavity and two lacerations measuring approximately 2 mm on the medial aspect of the spleen. Bleeding was stopped with argon beam and tissue glue. Splenectomy was considered not necessary, with control of bleeding achieved. The baby was in a critical condition after the operation. The postoperative course was complicated by acute renal failure and disseminated intravascular coagulation requiring massive blood product infusion. A further complication was the development of a subcapsular haematoma of the liver (maximum size: approximately 5.5 cm). The hepatic haematoma was managed conservatively and serial ultrasound monitoring showed eventual resolution at the age of 2 months.

The baby's condition gradually stabilised. After management of other typical problems associated with prematurity—including clinical sepsis, glucose intolerance, neonatal jaundice, and parenteral nutrition-related cholestasis—he was discharged at a corrected age of 42 weeks' gestation and with a birth weight of 2350 g. Neurological examination at this time was normal. Last follow-up at the age of 11 months showed that development was appropriate for corrected age and there was no neurological deficit. This is the first reported case of survival with intact neurology and normal development following splenic injury in a very low-birth-weight neonate.

Discussion

Aetiology and pathogenesis

Splenic rupture is a rare occurrence in the newborn, probably because of the high and well-protected location of the spleen beneath the diaphragm. Diagnosis during life and successful repair is very rare. More autopsy diagnoses are reported than are diagnoses of surviving cases.¹ There are few reports of splenic rupture in very-low-birth-weight neonates in the literature.^{2,3}

A pathologically enlarged or friable spleen, usually due to erythroblastosis, or in the past syphilis, is known to be more liable to rupture.^{4,5} In erythroblastosis, the congested spleen may more easily rupture due to mechanical distension

of the capsule. Moreover, babies with erythroblastosis have more frequent hypoxia. This could contribute to damage of endothelial and vessel wall integrity, and in turn, splenic rupture.⁴

Even in the histologically normal spleen, difficult delivery^{6,7} or in-utero trauma⁸ can result in intra-abdominal organ rupture. Interestingly, there have been reports of splenic injury in cases with an uneventful perinatal history and where the spleen was otherwise normal.^{9,10} Neither extrinsic aetiologies nor intrinsic pathology of the organ could be identified. The pathogenesis of splenic injury after uneventful delivery in the normal spleen remains obscure. In our case, before the baby was delivered by uneventful LSCS, the mother was in labour with regular uterine contractions and the foetus was in footling breech presentation, with both legs prolapsed out through the cervical os into the vagina. Increased intrathoracic pressure in the foetus during uterine contractions can force the liver and spleen out of the diaphragmatic hollow, causing severe tension in their supporting ligaments.^{9,11} Chrissy and Aaron⁹ reported a case of successful treatment of splenic rupture in an otherwise 'normal' spleen in 1980. In that case, a splenic tear at the site of implantation of the splenorenal ligament was found intra-operatively. Our case appears similar, with the absence of external bruises and the presence of lacerations on the medial aspect of the spleen near the supporting ligament.

Clinical presentation and diagnosis

Generally speaking, splenic rupture is believed to occur in two stages.⁶ Initially, there is formation of a subcapsular haematoma. Later, the capsule gives way and the baby develops acute symptoms of decompensation due to massive intra-abdominal bleeding. This explains why neonates with perinatal splenic rupture can present either within the first few hours of life or as late as the second week of life, following rupture of the splenic capsule. The classical presentations of shock, anaemia, and abdominal distension as seen in our case have been well described in the literature.^{2,6} Other possible signs include a discoloured abdomen, abdominal mass,¹⁰ or bluish scrotum.¹² Catastrophic intraventricular haemorrhage, severe necrotising enterocolitis, fulminant sepsis, and gut malrotation with volvulus can present similarly in the neonate and these differential diagnoses should also be kept in mind.

Specific roentgenogram features of haemoperitoneum have been well-described in the literature—free abdominal fluid, a large clear zone over the left flank with absence of the splenic outline on high-dose excretory urography, and floating of intestinal loops in the mid-abdomen.¹⁰ Diagnostic peritoneal lavage or exploratory laparotomy is undertaken to confirm the presence of bleeding. Nowadays, non-invasive modalities such as ultrasound and CT scanning are readily available to aid diagnosis.^{2,13} Moreover, they are useful in monitoring the progress of haematoma during conservative treatment.² The main drawback is

that transferring a critically ill neonate for CT scanning may further compromise the labile haemodynamic status of the baby.

In our case, the diagnosis of intra-abdominal bleeding was made on the basis of clinical and laboratory findings. Imaging studies, including ultrasound or CT scanning of the abdomen, could confirm the diagnosis. This, however, would have been at the expense of delaying life-saving treatment.

Treatment

There have been reports of successful survival of babies with splenic rupture with supportive treatment alone, owing to spontaneous cessation of the haemorrhage.^{14,15} This should be regarded as an exception, rather than the rule. Laparotomy and splenectomy have been the treatment of choice and this approach is well-described in early case reports of survival.^{5,6} Splenectomy, however, can result in serious immunologic consequences. Merikanto et al¹⁶ reported a deficiency of opsonic activity towards pneumococcus in the sera of children who underwent splenectomy as neonates. Overwhelming sepsis is dangerous and potentially fatal. Efforts to preserve the spleen are therefore indicated. An early report showed that in selected cases with splenic lacerations, bleeding could be stopped completely by application of oxidised cellulose and pressure.⁴ Suturing and absorbable mesh were also found to be useful in salvage of the spleen.^{17,18} In our case, the spleen was salvaged by achieving haemostasis using argon beam and tissue glue. This approach may have the potential complication of ongoing oozing due to subsequent coagulopathy. Nevertheless, the bleeding may be managed conservatively by supportive management, including top-up transfusion, correction of coagulopathy, and serial ultrasound monitoring of the residual haematoma. In Germany and Japan, reimplantation of splenic tissues in the subphrenic spaces¹⁹ and abdominal wall⁷ have been reported. Immune function was reported to have normalised at 3 to 5 months after surgery. When splenectomy becomes unavoidable, prophylaxis against encapsulated bacteria using antibiotics and vaccination is still widely practised to decrease the incidence of post-splenectomy sepsis.

Conclusions

Splenic rupture in a neonate is a rare surgical emergency associated with high mortality. Our case illustrates the classical triad of signs including hypovolaemic shock, anaemia, and abdominal distension. The diagnosis of acute intra-abdominal bleeding and splenic injury should be

considered, especially in neonates with congested organs or a history of difficult delivery. A high index of suspicion, early recognition, and prompt treatment are critical to the survival of these neonates.

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