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CASE REPORT

Splenic rupture in a neonate – a rare complication

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Birth trauma is well described in the neonatal literature, but intra-abdominal injuries occur infrequently and are often forgotten in the differential diagnosis of a hypovolaemic shocked infant with an abdominal mass. The symptoms of splenic rupture are nonspecific, creating a diagnostic dilemma for the clinician. As splenic rupture denotes a surgical emergency, increased awareness of this complication may decrease the delay in diagnosis, therefore increasing the infant's chances of survival. This applies not only to paediatricians but to every midwife, intern or medical officer who attends to deliveries and is involved with the care of the newborn.

Serious injury to the abdominal organs is a rare complication of birth trauma, and is seldom reported in the literature. The incidence of intra-abdominal injury related to birth has decreased during the past 50 years, and it is often forgotten in the differential diagnosis of a hypovolaemic shocked newborn. This leads to a delay in diagnosis, which increases the resultant morbidity and mortality. Hepatic injury occurs most commonly, followed by adrenal haemorrhage.

Case report

A 26-year-old woman (para 1, gravida 2) delivered a 3 640 g baby at home. The baby cried immediately, and was attended to by paramedics shortly after birth. She was taken to a district hospital, and was assessed by the doctor on duty at 12 hours of age. The baby was found to be lethargic and unwilling to breastfeed.

On physical examination the child had abdominal distension and no signs of respiratory distress. She also had a pansystolic murmur, audible over the left lower sternal border. She was referred to Steve Biko Academic Hospital (SBAH) with a differential diagnosis of a congenital cardiac lesion and possible septicaemia.

At SBAH the patient had an oxygen saturation of 90% in room air, with a pulse rate of 165/min and a blood pressure of 58/38 (mean 49) mmHg. She was active, but had subconjunctival haemorrhages, facial bruising and pallor. The findings on neurological and respiratory examination were normal. Cardiac examination confirmed the pansystolic murmur; there was no cyanosis, and all peripheral pulses were present. The abdomen was distended with normal bowel sounds and hepatosplenomegaly.

Initial laboratory results included a haemoglobin concentration of 9.5 g/dl, a haematocrit of 31%, a white cell count of 14.03×10^9/l and a platelet count of 351×10^9/l. The results of renal function tests were normal. Arterial blood gas values were pH 7.4, arterial oxygen tension (pO_2) 86.9 mmHg, arterial carbon dioxide tension (pCO_2) 20 mmHg, base excess -10.5 mmol/l, bicarbonate 17.5 mmol/l and oxygen saturation 97%. The abdominal radiograph was consistent with free fluid in the abdomen, and the chest radiograph revealed cardiomegaly (Fig. 1, a).

The patient was transfused with 15 ml/kg of packed red cells, but the post-transfusion haemoglobin was 7.5 g/dl and the haematocrit 23%. She deteriorated clinically and required intubation. The abdominal distension was worse, with a clearly palpable mass in the left upper quadrant (Fig. 1, b). A second transfusion of 15 ml/kg packed red cells was commenced.

An abdominal ultrasound scan revealed hepatomegaly with normal parenchyma. The spleen was difficult to demonstrate but measured 3 cm. There was also a clear mass noted above the left kidney, with an onion-skin appearance (Fig. 1, c). A large amount of free echogenic fluid was demonstrated (Fig. 1, d).

The cardiac ultrasound scan confirmed an acyanotic cardiac lesion with a complete atrioventricular canal defect, a common atrium, an interrupted inferior vena cava, a small patent ductus arteriosus and a moderate ventricular septal defect. The cranial ultrasound scan was normal.

The differential diagnosis at this stage was adrenal haemorrhage or congenital neuroblastoma, which has a known association with congenital cardiac disease.[1] Haemorrhagic disease of the newborn, congenital coagulopathies and sepsis with disseminated intravascular coagulation were also considered, but the clotting profile was normal and infectious markers were not raised.

The paediatric surgeons were consulted, and an abdominal needle paracentesis confirmed that the free fluid in the abdomen was blood. The patient was taken to theatre for an explorative laparotomy. A left transverse incision was made. A large amount of blood and blood clots were removed. Upon closer inspection a shattered spleen was found (Fig. 2, a). Owing to active bleeding the spleen had to be removed completely, leaving only a small splenicule *in situ* (Fig. 2, b).

A diagnosis of splenic rupture due to possible birth trauma was made. The mother admitted a month later that she had been assaulted two weeks before delivery, and this was considered to be a possible contributing factor.

Discussion

In the 1950s the incidence of intra-abdominal injury related to birth was as high as 3.5%, with the liver, adrenal gland and spleen most commonly affected.[2] However, splenic injury occurs much less often than rupture of the liver. This is partly because of the protected position of the spleen beneath the diaphragm in the left upper quadrant of the abdomen.[3,4] Injuries can range from small capsular lacerations to disintegration or shattering of the spleen.[4] Splenic rupture is a surgical emergency, but treatment may be delayed when

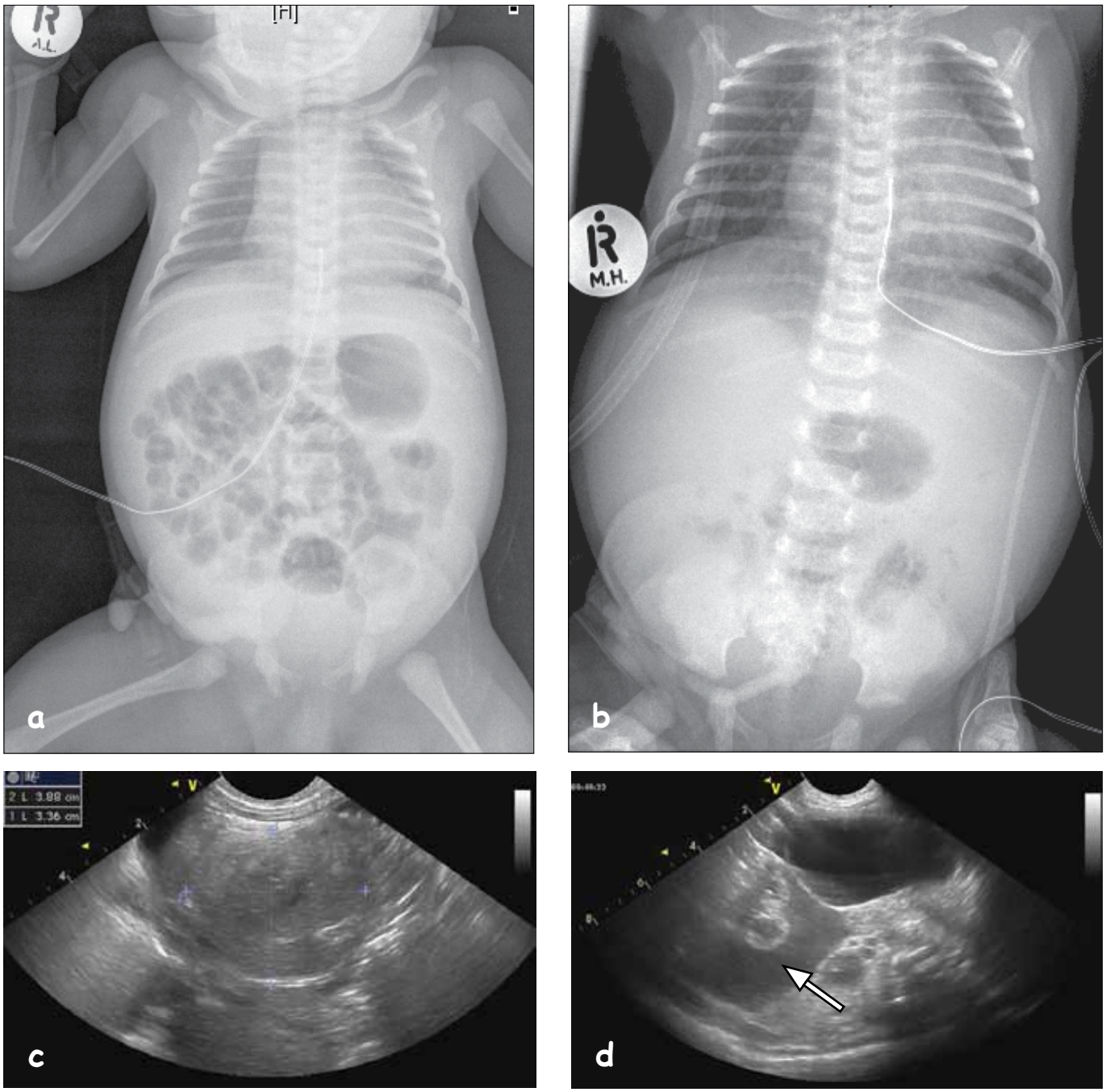

Fig. 1. Imaging studies. (a) Abdominal radiograph showing free fluid in abdomen and cardiomegaly. (b) Repeat abdominal radiograph after worsening of the patient's clinical condition. (c) Ultrasonogram illustrating a suprarenal mass measuring 3.88×3.36 cm. (d) Free echogenic fluid (arrow).

the diagnosis is not obvious due to absence of a history or a high index of suspicion.[5]

Aetiology

Birth-related intra-abdominal injuries are more likely to occur with breech or complicated vertex delivery as a result of excessive compression of the abdominal cavity.[3,5] They also occur in infants with conditions leading to intra-uterine pathology of the splenic parenchyma, e.g. erythroblastosis fetalis and congenital syphilis, as an enlarged spleen is more fragile than a normal organ.[3-5] Underlying clotting defects has been described. Rupture of the spleen can also occur in normal-sized infants with uneventful deliveries and no underlying disease.[5,6] An apparently normal labour and delivery do not exclude the diagnosis of intra-abdominal trauma. The incidence of splenic rupture is not known, and there are more autopsy diagnoses reported than surviving cases.[5]

Pathogenesis

The pathogenesis is not understood clearly. The mechanism of injury may be tension on the supporting ligaments of the liver and spleen that occurs when increased intrathoracic pressure forces these organs out of their normal positions.[5] Splenic rupture occurs in two stages. Initial subcapsular haematoma formation may have only mild symptoms of unexplained anaemia and a left upper quadrant mass. The second stage may occur after hours or days, and is usually characterised by the rapid development of shock, as the spleen and mesentery tend to bleed rapidly and copiously.[2-5] This 'delayed rupture' is described mostly with primary hilar and/or parenchymal injury. Rupture occurs within 14 - 21 days in 96% of cases.[2]

Clinical manifestations

The classic presentation is a triad of bleeding, abdominal distension and haemoperitoneum.[5] Blood loss and haemoperitoneum can lead to nonspecific signs such as poor feeding, listlessness, pallor,

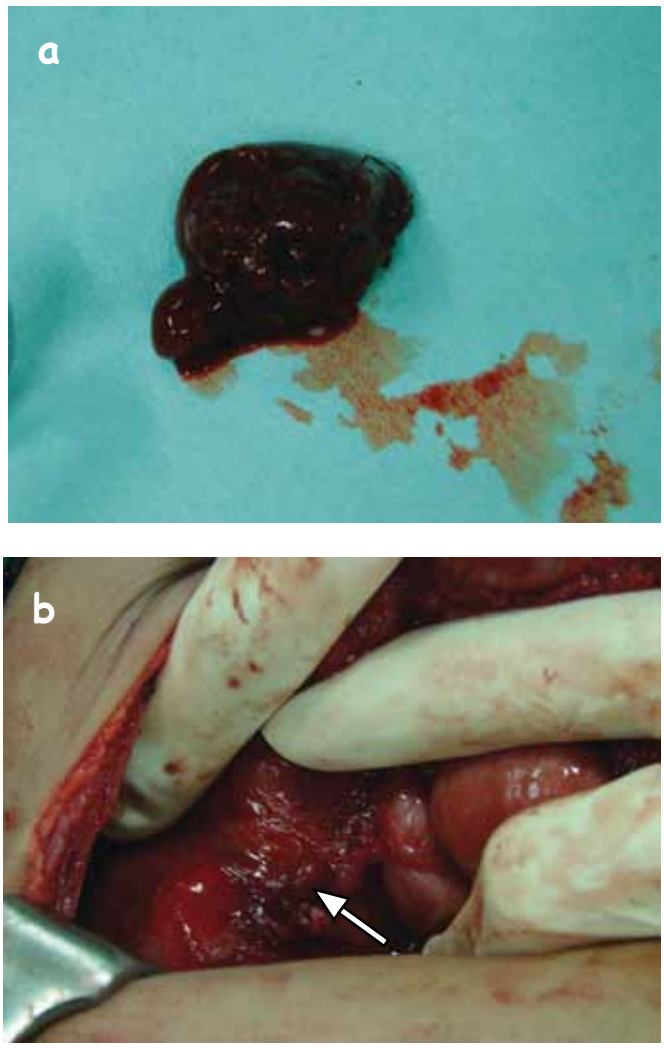

Fig. 2. (a) Ruptured spleen after removal. (b) Small splenicule left* in situ *(as indicated by arrow).

jaundice, tachypnoea and tachycardia. Circulatory failure and shock occur commonly.[5] A left upper quadrant mass may also be palpable. Haemoglobin and haematocrit values decrease, and abdominal paracentesis may reveal free blood. Patients do not present immediately after birth.[5]

Special investigations

Ultrasonography and computed tomography (CT) are the investigations of choice for suspected intra-abdominal haemorrhage.[4] Ultrasonography is considered the most appropriate screening method in the non-trauma situation, as it may illustrate anatomy better than CT in some instances.[4] Ultrasonography is also more readily available, as transport of a critically ill patient to a centre with a CT scanner is not always advisable.[5]

A mass effect displacing the stomach and intestine medially may be apparent on an abdominal radiograph (see Fig. 1, a). Intra-abdominal fluid may be visible on both ultrasound and CT. Diagnostic paracentesis may confirm the presence of haemoperitoneum.[5]

Differential diagnosis

Injury to the other abdominal organs may present similarly, e.g. liver rupture or adrenal gland haemorrhage.

Treatment

Fluid resuscitation with packed red cells should be initiated immediately, combined with the correction of clotting defects if present. Explorative laparotomy should follow as soon as possible.[6] Splenectomy was the standard treatment 20 years ago, but currently attempts should be made to repair and preserve as much splenic tissue as possible to prevent the increased risk of infection following splenectomy.[5,6] When haemostasis cannot be achieved, splenectomy may be performed.[5]

If the bleeding is small, it may be managed conservatively with continuous monitoring and fluid resuscitation, including the replacement of red blood cells. Surgery is advised when the patient becomes unstable, or if the bleeding is uncontrolled.[6]

Prognosis

With early recognition and surgery, the survival rate should approach 100%.[6]

Post-splenectomy prophylaxis[7]

Penicillin prophylaxis is advised lifelong, but at least for the first 2 years of life as compliance is reported to be poor. Immunisation against encapsulated bacteria (*Streptococcus pneumoniae*, *Haemophilus influenzae* and *Neisseria meningitidis*) is important. The patient/parents should also be advised regarding the possibility of overwhelming sepsis.

Conclusion

In our case splenic rupture was initially not included in the differential diagnosis, as it is a rare complication in modern medicine.

We identified two contributing factors: maternal trauma 2 weeks before delivery, and possible birth trauma during an unattended delivery. We could not find any literature clarifying a time frame during which maternal trauma could be linked to splenic rupture of the newborn. However, the onion-skin appearance of the haematoma on ultrasound suggests a more chronic onset. The history obtained from the mother did not suggest any difficulty during the delivery, but the presence of subconjunctival haemorrhages and facial bruising could indicate some trauma. We therefore cannot attribute the splenic injury to either of these factors with certainty.

Splenic rupture is a rare complication in a neonate, but should always be kept in mind when dealing with a hypovolaemic shocked newborn with an abdominal mass.

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