SHORT REPORT

Duodenal perforation: a diagnostic pitfall in non-accidental injury

M P Champion, C A Richards, S A Boddy, H C Ward

Arch Dis Child 2002;87:432-433

Duodenal perforation is a rare, life threatening injury associated with non-accidental blunt abdominal trauma. Diagnostic delay is common, as the true history is concealed and signs may be minimal. Double contrast computed tomography is the most sensitive investigation to confirm clinical suspicion. We report three cases, all with other features typical of non-accidental injury.

In the UK each year, 1 in 1000 children are severely physically abused, and at least 1 in 10 000 die from their injuries.¹ Blunt abdominal trauma may inflict severe injury that is not immediately apparent. We report three cases of duodenal perforation following blunt non-accidental trauma to highlight the difficulties in diagnosis.

CASE 1

A 9 month old girl presented with a week's history of diarrhoea and vomiting. She was pale and dehydrated, with facial and lumbar bruising. The parents were unable to account for her injuries, blaming an older sibling; their full extent only became apparent subsequently. An hour after arrival, she developed abdominal distension. *x* Ray examination revealed free intraperitoneal gas, a recent fracture of the 11th rib (without callus), and an older fracture of the 10th (with abundant callus). Cranial computed tomography (CT) scan was normal; enhanced CT of the abdomen confirmed free peritoneal gas and fluid, anterior displacement of the superior mesenteric vessels, and a posterior perfusion defect of the spleen (fig 1). Laparotomy revealed a complete rupture of the third part of the duodenum, requiring excision of necrotic tissue and end to end anastomosis. The histological features were in keeping with an injury at least 48 hours old, indicating that the perpetrator had access to the child a day or more before presentation, thus opening up the field of enquiry.

CASE 2

A 20 month old boy presented with a three day history of vomiting and abdominal pain. He was emaciated, tachycardic, and hypotensive. There was bruising over the face and trunk, and the abdomen was distended with absent bowel sounds. Chest *x* ray examination showed an old fracture of the left clavicle; abdominal *x* ray examination showed distended bowel loops. An ultrasound scan showed a small volume of free fluid and a possible subcapsular hepatic haematoma. Cranial CT was unremarkable. When questioned, the father attributed the injuries to falling out of bed onto a carpeted floor. The boy had previously been investigated for failure to thrive; the family were known to social services. He was admitted and initially improved with conservative management. However, by 48 hours, he had developed a pyrexia and abdominal guarding. Laparotomy revealed peritonitis with a 5 mm perforation of the third part of the duodenum, which

Figure 1 Enhanced CT of the abdomen of case 1, showing free intraperitoneal fluid and gas and a posterior perfusion defect of the spleen. Other cuts showed anterior displacement of the superior mesenteric vessels caused by retroperitoneal swelling.

was repaired. The skeletal survey showed an undisplaced spiral fracture through the left femur.

CASE 3

A 26 month old boy presented with a history of a generalised convulsion, lethargy, and vomiting. There was no history of trauma. On arrival, he appeared irritable and post-ictal, and had a swollen left knee. By the following morning, he was pyrexial with bile stained vomiting and a distended, tender, silent abdomen. Abdominal *x* ray examination showed patchy free gas, and ultrasound extensive loculated peritoneal fluid. Laparotomy revealed an 80% transection of the third part of the duodenum, which was repaired. Subsequent CT brain was normal, and it was determined that he had been investigated elsewhere two months previously for a metaphyseal fracture of the left femur.

DISCUSSION

Blunt abdominal trauma is the second commonest cause of death in abused children. The mobility of the small intestine is protective, but the fixed retroperitoneal course of the duodenum renders it vulnerable; the commonest visceral injury is intramural haematoma of the duodenum or proximal jejunum² following direct compression against the vertebral column. Perforation or disruption may occur at the time of injury, or later secondary to tissue necrosis. The posterior position of the duodenum within the abdomen implies that

significant force is required to disrupt it, increasing the likelihood of injury to neighbouring organs. Case 1 had additional injury to the spleen and a retroperitoneal haematoma; case 2 had a small mesenteric haematoma; none had evidence of injury to the pancreas. The forces required to cause intestinal injury are great,³ and a clear history of a dramatic event should be forthcoming from the carers to account for it, for example, a road traffic accident. Doubt should be cast on the validity of any story where only a minor or moderate mechanism for the injury is given.

Diagnosing duodenal perforation is notoriously difficult. In non-accidental injury the history is misleading and the true mechanism concealed, as shown by these three cases. The clinical signs of retroperitoneal injury may be limited, and overshadowed by other injuries. Clinical findings such as abdominal pain or tenderness may be subtle or delayed, and a high index of suspicion is required. When examining children with abdominal symptoms, with unusual or contradictory histories, particularly when there has been a delay seeking medical help, the possibility of non-accidental injury must be considered. Associated injuries are present in up to 65%,⁴ including cutaneous bruising, intraocular haemorrhages, and multiple fractures of varying ages. All three cases described here had bony injury typical of physical abuse.²

Conventional investigations may add little to the evaluation. A raised amylase may indicate duodenal perforation or pancreatic damage. The serum amylase was measured in the second case and was normal. An intraperitoneal rupture of the duodenum may show the typical features of free intraperitoneal gas, but was not apparent in our second case. Retroperitoneal duodenal perforation is frequently difficult to diagnose using conventional radiology.⁵ The most sensitive method of detecting occult retroperitoneal trauma is CT scan with intravenous and oral contrast,⁶ and this should be performed

whenever the diagnosis is suspected if there is no clear indication for laparotomy. CT scanning also allows other injuries to be identified. However, findings are often subtle, and the diagnosis may still be missed,⁶ requiring continued clinical review in suspicious cases.

ACKNOWLEDGEMENTS

Thanks are extended to Dr Iona Jeffrey for advice on histological material.

Authors' affiliations

M P Champion, Department of Paediatric Intensive Care, Guy's Hospital, London, UK

C A Richards, H C Ward, Department of Paediatric Surgery, University Hospital, Lewisham, London, UK

S A Boddy, Department of Paediatric Surgery, St George's Hospital, London, UK

Correspondence to: Mrs C A Richards, Department of Paediatric Surgery, University Hospital, Lewisham High Street, London SE13 6LH, UK; catherine.richards@uhl.nhs.uk

Accepted 10 June 2002

REFERENCES

- 1 **Meadow R**. *ABC of child abuse*. London: BMJ, 1993.
- 2 **Merten DF**, Carpenter BLM. Radiologic imaging of inflicted injury in the child abuse syndrome. *Pediatr Clin N Am* 1990;**37**:815–37.
- 3 **Cripps NPJ**, Cooper GJ. Intestinal injury mechanisms after blunt abdominal impact. *Ann R Coll Surg Engl* 1997;**79**:115–20.
- 4 **Ledbetter DJ**, Hatch EI, Feldman KW, et al. Diagnostic and surgical implications of child abuse. *Arch Surg* 1988;**123**:1101–5.
- 5 **Toxopeus MD**, Lucas CE, Krabbenhoft KL. Roentgenographic diagnosis in blunt retroperitoneal duodenal rupture. *Radiology* 1972;**115**:281–8.
- 6 **Mirvis SE**, Gens DR, Shanmuganathan K. Rupture of the bowel after blunt abdominal trauma: diagnosis with CT. *AJR* 1992;**159**:1217–21.

ARCHIVIST

Pneumococcal infection in children with no spleen

Overwhelming infection remains a problem for children who have no spleen either because they were born without one or because they have undergone splenectomy. They are at risk from all encapsulated bacterial pathogens but more than half of invasive infections in these children are caused by *Streptococcus pneumoniae*. Data on invasive pneumococcal infections have been collected from eight children's hospitals in the United States since 1993 (Gordon E Schutze and colleagues. *Pediatric Infectious Disease Journal* 2002;**21**:278–82).

Between September 1993 and August 1999 a total of 2581 episodes of invasive pneumococcal infection were reported, including 26 episodes in 22 children with no spleen (12 congenital asplenia (9 with complex congenital heart disease), 10 post splenectomy). Six (27%) of these 22 children died, five of proved meningitis, and the sixth of suspected meningitis. Of 2476 children with a spleen who had invasive pneumococcal infection 33 (1.3%) died, 17 of meningitis.

Average age at first infection was 12.5 months (congenital asplenia) and 69 months (postsplenectomy). Nine of the 12 children over 2 years old had received the 23-valent polysaccharide pneumococcal vaccine and 18 of the 22 children had been given antibiotic prophylaxis. Presenting signs in the 26 episodes included shock (7), petechiae or purpura (7), disseminated intravascular coagulation (5), and respiratory distress (5). The clinical illnesses were bacteraemia alone (12), meningitis alone (8), bacteraemia with otitis media/sinusitis (3), bacteraemia with pneumonia (2), and meningitis with osteomyelitis (1). Almost half (46%) of isolated pneumococci were nonsusceptible to penicillin and almost 20% were nonsusceptible to ceftriaxone. The new heptavalent conjugate vaccine is expected to improve the protection provided for these children but five of the 26 isolates were of serotypes not present in the conjugate vaccine and one was of a serotype not present in the 23-valent polysaccharide vaccine.

The authors of this report endorse a policy of using both vaccines for children with no spleen. For those under 2 years they recommend the conjugate vaccine followed at 2 years by the polysaccharide vaccine. For those over 2 years they recommend an age-appropriate schedule of immunisation with the conjugate vaccine followed by the polysaccharide vaccine 6 to 8 weeks after the last dose of conjugate vaccine.