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Occult ruptured spleen—two unusual clinical presentations

P. G. MOORE
M.B., B.S., F.F.A.R.A.C.S.

J. G. GILLIES
M.B., B.S., F.R.C.S. (Eng), F.R.A.C.S.

O. F. JAMES
A.M., M.S., F.F.A.R.C.S., F.F.A.R.C.S.I., F.F.A.R.A.C.S.

NICHOLAS SALTOS
M.B., B.S., M.R.C.P., M.R.C.P.I., F.R.A.C.P.

Departments of Anaesthesia and Intensive Care, Surgery and Pulmonary Medicine, The Royal Newcastle Hospital, Newcastle 2300, Australia

Summary

Case reports of two patients with occult rupture of the spleen are presented. In one, blunt trauma appeared to involve only the neck and upper chest, resulting in two distinct tracheal injuries and no clinical indication of abdominal injury. On the 5th day after injury this patient strangulated an indirect inguinal hernia. At subsequent surgery, a ruptured spleen was also found. The second patient gave no history of trauma and presented in cardiac and respiratory failure after a 2-month illness characterized by abdominal pain. On clinical and biochemical assessments, he was considered to have pancreatitis complicated by pseudocyst formation. Laparotomy revealed intra-abdominal haemorrhage and a ruptured spleen. The diagnosis and complications of occult ruptured spleen are discussed.

KEY WORDS: scrotal swelling, tracheal injury, acute pancreatitis.

Introduction

The two patients presented in this paper emphasize the problems which may be encountered in diagnosing the underlying cause of a critical illness in patients presenting with systems failure in an Intensive Care Unit (ICU).

In particular, the second patient emphasizes the need for strong suspicion of underlying intra-abdominal disease in patients who present with acute respiratory, cardiac and renal failure.

Case reports

Case 1

A 27-year-old male was transferred to the ICU of The Royal Newcastle Hospital (RNH), 36 hr after injury in a motor vehicle accident. He was the driver of a car which was involved in a 'head-on' collision. He was not wearing a seat-belt and had sustained bruising to the upper central chest and the root of the neck from impact with the steering wheel. The initial mild swelling around the root of the neck greatly increased in size in the 12 hr before his admission. The swelling was associated with difficulty in swallowing and clotted blood was observed in his sputum. His only other obvious injuries were small lacerations on the nose, right elbow and both knees.

Further examination revealed a large swelling in the neck. He had inspiratory stridor, laboured respirations, a persistent cough with blood-stained sputum and pleuropericardial clicks (Hamman's sign). Chest examination was otherwise normal, his abdomen was soft and non-tender, systemic arterial blood pressure was 100/75 mmHg and the pulse 100/min. Chest radiological examination showed mediastinal and cervical air. Arterial blood gas analysis was normal. Bronchoscopy revealed two separate tracheal injuries, a proximal injury on the right anterolateral wall 2–3 cm below the vocal cords not thought to involve the full thickness of the tracheal wall. The tracheal lumen was narrowed in this area. A second longitudinal full thickness laceration was observed in the distal posterior wall 3–4 cm above the carina. In view of the airway narrowing and absence of pulmonary injury, a cuffed 'Portex' (polyvinyl) oral endotracheal tube was inserted past the proximal obstructing injury and the patient was allowed to breathe spontaneously. The tube was well tolerated.

He made satisfactory progress until 3 days after admission and 5 days after injury, when he became hypotensive (systemic arterial pressure 90/45 mmHg) and his pulse increased to 110/min. He indicated epigastric, left scrotal and groin pain and a large left

scrotal swelling was noted. He looked pale and distressed. The scrotal swelling was tense and tender but his abdomen was soft and not tender. A diagnosis of strangulated left inguinal hernia was made.

Under epidural anaesthesia with spontaneous breathing, a left inguinal transverse incision was made and an indirect hernial sac containing greater omentum and frank blood was found. Inguinal repair was followed by an upper midline laparotomy. This revealed approximately one litre of old blood and clot in the peritoneal cavity and a ruptured spleen. On the diaphragmatic surface was an area of subcapsular haematoma overlying a splenic laceration from which the haemorrhage appeared to arise. There were a number of smaller splenic lacerations at the superior pole and near the hilum as well. No other intra-abdominal injuries were found and splenectomy was performed.

His subsequent recovery was complicated by bilateral bronchopneumonia. Sputum culture grew *Staphylococcus aureus* sensitive to diethanolamine fusidate. He received intensive physiotherapy. On the 11th post-accident day the endotracheal tube was removed, bronchoscopy confirmed resolution of his tracheal injuries and he was discharged from hospital on the 17th post-accident day.

Case 2

A 42-year-old male was admitted to RNH with severe left hypochondrial and left posterobasal chest pain for 1 day and left shoulder tip pain for 2 days. Two months before admission he developed severe upper abdominal pain radiating to the right hypochondrium and loin. This was made worse by the ingestion of food or drink and partly relieved by sitting up. Although unwell he continued to work and gradually improved over the next 3 weeks. Radiological examinations of the chest, cholecystogram and intravenous pyelogram performed 2 weeks before admission were normal. Past history included pneumonia at the age of 19 years and haemorrhage from a pyloric ulcer at 36 years. This latter was treated by oral cimetidine.

On examination he was distressed with upper abdominal tenderness and guarding. His systemic arterial blood pressure was 100/60 mmHg, pulse 96/min.

Chest examination elicited dullness at both bases and absence of basal breath sounds. His haemoglobin was 12·5 g/dl, white cell count $23{\cdot}7 \times 10^9$/litre and serum amylase 1237 u/litre (normal <300 u/litre). Chest radiographic examination showed bilateral basal collapse and consolidation. A diagnosis of acute-on-chronic pancreatitis with bilateral pulmonary basal atelectasis was made and he was managed with intravenous fluids and morphine analgesia.

On the second hospital day he developed a left-sided pleural effusion from which 70–80 ml of heavily blood-stained fluid was drained. Later that day his condition deteriorated. He became confused and cyanosed with a respiratory rate of 40/min, pulse rate 120/min and arterial blood gases analysis whilst spontaneous breathing room air showed a Pa_{O_2} of 27 mmHg, Pa_{CO_2} 30 mmHg, pH 7·457. An oral endotracheal tube was inserted and ventilation with positive end-expiratory pressure (PEEP) was commenced. He was oliguric, his systemic arterial blood pressure was 95/60 mmHg, central venous pressure was 12 cmH_2O. He was given methylprednisolone 1 g IVI and commenced on dopamine/dobutamine infusion. Adequate oxygenation was achieved with mechanical ventilation with an F_IO_2 of 0·60 and PEEP of 18 cmH_2O. His haemoglobin level had fallen to 8·9 g/dl but bleeding studies were normal. Although the most likely diagnosis was still acute pancreatitis complicated by sepsis, pneumonia and cardio-respiratory failure, sufficient doubt existed to justify a laparotomy in order to determine and treat the specific nature of the upper abdominal pathology.

At laparotomy free blood and a perisplenic haematoma was found. This haematoma covered the diaphragmatic surface of the spleen which was small and was stripped of its capsule over the diaphragmatic surface. There was no major splenic laceration nor a splenic artery aneurysm. The only pancreatic abnormality was mild oedema without haematoma. Splenectomy was performed, the peritoneal cavity lavaged with saline and the abdomen closed leaving a drain in the left subphrenic space. Four units of whole blood were transfused during surgery and dopamine/dobutamine infusion ceased. His abdominal condition rapidly improved after operation and by the 5th post-operative day ventilatory support was no longer needed.

At this time in response to direct questioning, a retrospective history was obtained from his wife of noticing a haematoma of his left loin before the onset of his illness. The patient had no recollection of injury.

He was discharged from the Intensive Care Unit on the 7th and from hospital on the 12th day after operation.

Discussion

The spleen is the abdominal organ most commonly injured by blunt trauma (Di Vincenti, Rives and Laborde, 1968). When the spleen alone is injured, the mortality rate is less than 2% (Naylor, Coln and Shires, 1974). When the trauma to other organs is associated with splenic injury, the mortality rate is increased to about 10%.

The mortality rate is also increased in patients with

delayed splenic rupture, a condition which is estimated to occur in about 20% of patients with blunt splenic injury (Sizer, Wayne and Frederick, 1966). The use of peritoneal lavage in early assessment of abdominal blunt injury reduces delay in diagnosis of rupture of the spleen to less than 5% (Root *et al.*, 1965; Veith *et al.*, 1967). Some authors (Frey, Ernst and Lindenauer, 1967; Lim, Gleilman and Hunt, 1972) suggest that in those patients in whom clinical suspicion of rupture is high but in whom peritoneal lavage is normal, abdominal angiography may be helpful in confirming the diagnosis. However, angiography may be difficult to interpret and may not clarify the diagnosis.

While such manoeuvres may be of help when there are some indications of intra-abdominal injury, their aid is unlikely to be sought if there is nothing to suggest an intra-abdominal lesion or if other symptom complexes and investigations suggest other diagnoses. In our two cases, clinical suspicion of splenic injury did not exist. In the first patient, both history and examination failed to suggest any abdominal trauma. Presumably the steering wheel impact resulted in a subcapsular haemorrhage which was contained until the 5th day after injury when delayed ruptured and haemoperitoneum occurred. The increased effort of breathing and coughing associated with his pulmonary condition resulted in herniation of omentum and intra-peritoneal blood through a patent processus vaginalis. The overt appearance of strangulated hernia masked the unrecognized splenic rupture.

The second patient provided more of a problem of diagnosis although the abdominal symptoms and signs were in retrospect consistent with splenic rupture. The absence of a history of injury and the finding of a raised serum amylase further confused the clinical picture and failed to raise suspicion of splenic rupture. Acute traumatic pancreatitis can occur with a ruptured spleen. In these instances the body and tail of the pancreas is usually involved, the abdominal trauma is usually major and the ensuring pancreatitis is usually severe.

The development of remote organ failure after abdominal injury is well recognized particularly when intra-abdominal sepsis ensues (Root *et al.*, 1965; Polk and Shields, 1977; James and Moore, 1983). The occurrence of acute cardiac, respiratory or renal failure in patients previously well or recently injured should raise the suspicion of intraabdominal sepsis and perhaps lead on to exploratory laparotomy. In these circumstances definitive surgery or drainage of localized sepsis offers improved chances of survival.

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