

Case report

Spontaneous splenic rupture: a unique presentation of Q fever

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Spontaneous rupture of the spleen is rare but can occur in illnesses which alter normal splenic morphology. These include infections, typically infectious mononucleosis,¹ haematological malignancies,² and miscellaneous conditions such as rheumatoid arthritis, sarcoidosis and polyarteritis nodosa. It has not previously been reported in association with Q fever.

CASE REPORT

A 55-year-old female, from a rural area, following a four-day history of 'flu-like illness developed sudden onset severe epigastric pain radiating to her back and left shoulder. On admission to hospital she was hypotensive (blood pressure 80/60 mmHg) with a sinus tachycardia of 110/min. Abdominal examination was unremarkable. Haemoglobin was 10 g/dl, PCV 0.32, WCC $24.3 \times 10^3/l$, serum urea and electrolytes were normal and the serum amylase 120 Somogyi units. Supine abdominal X-ray showed a soft tissue mass in the left upper quadrant. Despite vigorous resuscitation she remained profoundly shocked.

At emergency laparotomy three litres of blood were found in the peritoneal cavity associated with a major splenic rupture. Splenectomy was performed and because several sizeable splenunculi were seen, no re-implantation of splenic fragments was attempted. Subsequent close questioning of the patient and her relatives failed to establish any preceding history of trauma. The postoperative period was complicated by persistent pyrexia, left-sided pleuritic chest pain and haemoptysis. ESR remained elevated at 55 mm/hr associated with a marked leucocytosis and elevated serum transaminases (AST 251 μ/l , ALT 334 μ/l).

Chest X-ray on admission had shown an area of oval shadowing in the left mid-zone which progressed postoperatively to collapse and consolidation of the left lower lobe. Repeated cultures of sputum and blood were negative. An atypical pneumonia was diagnosed radiologically and the clinical findings resolved with tetracycline therapy. Serological tests on acute and convalescent sera using the complement fixation test (Microtitre system) demonstrated a rising antibody to Q fever phase 2 antigen from < 1:10 to 1:80, thus confirming recent infection with *Coxiella burnetii*. Histopathology of the spleen demonstrated

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features of a non-specific infective process without evidence of leukaemic or neoplastic infiltration, connective tissue disorder, emboli or infarctive changes. Four months postoperatively the patient remained well with no evidence of residual Q fever.

DISCUSSION

Spontaneous splenic rupture, first described by Atkinson in 1874³ is rare. The mechanism is unknown although disruption of the splenic capsule and blood vessels by disease processes such as leukaemic deposits, microabscesses, rheumatoid arthritis and polyarteritis have been suggested. In such circumstances a sudden increase in portal pressure produced by coughing, vomiting or defaecation may precipitate splenic congestion and rupture. No obvious precipitating event occurred in this patient.

Q fever was first reported in Northern Ireland in 1962.⁴ Since then there has been a high incidence of the condition in the province,⁵ with 262 cases diagnosed up to 1986 when this case occurred. The condition commonly presents as a mild flu-like illness often associated with pneumonitis, although up to 30% may demonstrate an acute hepatic picture. A recent Australian report⁶ suggests that splenomegaly is present in over 30% of patients particularly if endocarditis is present. This case report is the first of spontaneous splenic rupture.

Correct preoperative diagnosis of this complication is difficult, often being confused with other intra-abdominal catastrophes.⁷ The diagnosis should be considered in any patient with an illness known to involve the spleen, who presents with unexplained abdominal pain and cardiovascular collapse. A high index of suspicion should reduce diagnostic delay, accelerate appropriate surgical intervention and reduce mortality.

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