Case report

Splenic rupture as a presenting feature of endocarditis

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Abstract

We describe the first case of infective endocarditis presenting with spontaneous splenic rupture. Our patient, a known intravenous drug user presented with hypovolaemic shock secondary to splenic rupture. The patient was resuscitated and underwent an emergency splenectomy. Subsequent clinical examination revealed a systolic murmur and a diagnosis of mitral valve infective endocarditis was made after echocardiography. Splenic tissue, blood cultures and mitral valve tissue all cultured Enterococcus faecalis. The patient had a successful mitral valve replacement and was discharged home after 44 days. To our knowledge this is the first reported case of enterococcal endocarditis presenting with splenic rupture. This case highlights the need to consider endocarditis in spontaneous splenic rupture particularly in those patients in a high risk group, such as IV drug users, especially if they lack a clear history of trauma.

Keywords: Splenic rupture; Infective endocarditis; Enterococcus faecalis

1. Case report

A 49-year-old female presented with a vague one week history of increasing abdominal pain and distension, dysuria, haematuria and malaise. The patient was a known intravenous drug user and had Hepatitis C. On admission the patient was afebrile, hypotensive (BP 75/50 mmHg) and tachycardic (HR 105/min). There was generalised abdominal tenderness, worse in left upper quadrant. Initial investigations revealed a haemoglobin of 51 g/l (115—160 g/l), microscopic haematuria and renal failure; urea 48 (2.1—7.1 mmol/l), creatinine 850 (50—100 μmol/l). The patient received balanced fluid resuscitation with aliquots of crystalloid and packed red cells. CT abdomen revealed a large amount of intra-abdominal free fluid adjacent to the liver and spleen and extending into the paracolic gutters. There was severe disruption of the spleen, which was split transversely in two regions (Fig. 1). On direct questioning, there was no history of recent abdominal trauma, and examination revealed no external evidence of trauma. The patient continued to deteriorate requiring further resuscitation with blood, clotting products and vasopressors. An urgent laparotomy revealed 2 l of intra-abdominal blood from a splenic subcapsular bleed and parenchymal tear. A spleenectomy was performed. Pathological examination of the spleen demonstrated multiple capsular disruptions, subcapsular wedge infarcts, extensive necrosis, abscess formation and rupture (Fig. 2).

Subsequent examination on the first postoperative day revealed a long systolic murmur. Trans-thoracic echocardiogram revealed severe mitral regurgitation with echo-dense lesions on both anterior and posterior mitral valve leaflets (1.0 × 0.7 cm and 0.5 × 0.4 cm, respectively). Left ventricular size and function was normal. Subsequently Enterococcus faecalis was cultured from splenic tissue. Antibiotic therapy of vancomycin and ciprofloxacin was commenced after further blood cultures had been taken (as the patient had a strong history of penicillin allergy). The patient decompensated with fast atrial fibrillation and pulmonary oedema. An urgent mitral valve replacement was performed using a 27 mm MedtronicTM mosaic stented bioprosthetic valve. Multiple blood cultures taken prior to mitral valve replacement and mitral valve tissue grew E. faecalis. Antibiotics were modified to vancomycin and gentamicin. A postoperative transoesophageal echocardiogram revealed a functionally and anatomically normal mitral valve bioprosthesis. The patient required aggressive organ support postoperatively with ventilation, inotropes and renal replacement therapy. She made a good recovery and was discharged from the intensive care unit after 10 days and from hospital on day 44.

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2. Discussion

Whilst splenic involvement in infective endocarditis (IE) is well recognised, this is, to the best of our knowledge, the first reported case of splenic rupture as the presenting feature of IE. Most reported cases of spontaneous splenic rupture in the setting of IE involve splenic rupture occurring after a diagnosis of IE has been made and often after the affected valve has been replaced [1–4]. Hence anticoagulation associated with either cardiopulmonary bypass or mechanical valve warfarinisation may be implicated in these cases. Splenic rupture in enterococcal endocarditis is also rare.

Historically the rate of enterococcus as a causative organism in native valve endocarditis is approximately 5–8% in patients aged 16–60 years, and more common in those aged over 60 years, at 14–17% [5–7]. However, more recent evidence demonstrates an increasing incidence of enterococcus as a causative organism in endocarditis in younger patients and as a nosocomial infection [5,6]. The mortality rate for enterococcal endocarditis remains high and has been reported to be as high as 24% in the recent literature [8]. Embolisation is variably reported as occurring in between 21 and 40% cases of endocarditis and is a significant cause of mortality particularly in the form of cerebrovascular accident [7]. The international collaboration on endocarditis found an embolisation rate of 26% for enterococcus endocarditis which is significantly less than staphylococcus aureus endocarditis and similar to streptococcal endocarditis [6]. The incidence of splenic embolism is reported as between 5 and 12% [5]. There is controversy as to the risk factors for embolism in endocarditis. Retrospective data suggests that the size of the vegetation and mobility are important factors; however, this is an inconsistent finding. Importantly, embolism becomes less frequent with length of treatment and has been demonstrated to occur in 75% of cases prior to the onset of therapy [7,9].

There are a number of possible mechanisms for splenic rupture in endocarditis. Histopathological studies have recognised that rupture may occur secondary to simple infarction but it also been documented to occur in relation to splenic abscess formation after embolisation. The incidence of splenic abscess formation post endocarditis is approximately 5% [10]. In our case pathological examination revealed evidence of suppurative necrosis suggestive of abscess formation.

In summary, while enterococcus is a relatively common cause of IE the combination of presumed embolism and splenic rupture is extremely rare and indeed the presentation of enterococcal endocarditis with splenic rupture has not been reported previously. In this instance the patient presented with spontaneous splenic rupture of unknown aetiology. This case highlights the need to consider endocarditis in spontaneous splenic rupture particularly in those patients in a high risk group, such as IV drug users, especially if they lack a clear history of trauma.

Conflict of interest statement

None declared.

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