



Splenic Rupture after Infective Endocarditis by *Enterococcus faecalis*: Case Report

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Authors' contributions

This work was carried out in collaboration between all authors. Authors TAM, ACAB and PGMBS wrote the draft of the manuscript. Authors TAM, ACAB, PGMBS, FQA managed the literature searches and contributed to the correction of the draft. Authors TAM, RLH, MCS, JCTG and VF provided the case, the figures and supervised the work. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aims: In patients with infective endocarditis, with risk of embolization, early identification of parenchymal changes may suggest the risk of splenic rupture.

Presentation of Case: A 68-year-old male presented with a history of 2 months of fever and also left upper quadrant pain initiated 2 days before admission. Transesophageal echocardiogram demonstrated the presence of two mobile vegetations on the ventricular side of the aortic valve; the largest diameter being 2.1 cm. *Enterococcus faecalis* was isolated in blood culture after a diagnosis of subacute aortic valve infective endocarditis. He complained of abdominal pain. An abdominal computed tomography scan revealed infarction of the upper region of the spleen (septic embolism). Therapy with penicillin and gentamicin was initiated, but the patient developed symptoms of heart failure that led to a surgical treatment, and aortic bioprosthesis was implanted on day 14. On day 5 postoperatively, the patient developed sudden hemorrhagic shock signs due

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to splenic rupture and underwent emergency splenectomy. A pathological examination revealed areas of splenic laceration of the capsule, splenic infarction areas, and the absence of abscesses. Splenic rupture is a complication much rarely occurring due to infectious endocarditis caused by *E. faecalis*.

Conclusion: This case highlights the importance of conducting serial imaging, particularly in symptomatic patients, for the early detection of parenchymal changes that may suggest the risk of rupture.

Keywords: Splenic rupture; endocarditis; *Enterococcus faecalis*; septic embolism.

1. INTRODUCTION

Splenic rupture is a severe and often fatal event. Although trauma is the most commonly reported [1,2] etiology that due to septic emboli from the endocarditis of the aortic valve and/or mitral valve is rarely described. The diagnosis and early treatment of bleeding complications are fundamental, because hemorrhagic shock is often fatal [3].

2. PRESENTATION OF CASE

A 68-year-old male Caucasian patient, with a history of hypertension, hyperuricemia, and a permanent pacemaker for symptomatic bradycardia, was admitted to the emergency department with fever, sweating, pale skin, a holodiastolic murmur in the aortic valve area, and pain on deep palpation of the left upper quadrant. He had no recent history of surgery or urinary tract infections. Two months prior to admission, he recounted having a fever which did not improve with antipyretics. Two days after admission, the patient's symptoms worsened, with dyspnea on mild exertion and intense pain in the left upper quadrant radiating to the back. Additional tests including blood cultures, transesophageal echocardiography (TEE), and abdominal tomography (due to patient's abdominal pain) were performed. The blood culture identified penicillin- and gentamicin-sensitive *Enterococcus faecalis*. TEE showed two vegetations on the ventricular face of the aortic valve, measuring 21 mm at their largest diameter, and moderate aortic insufficiency. Abdominal CT scan showed rupture of the upper segment of the spleen (Fig. 1). Antibiotic therapy guided by the antibiogram (penicillin and gentamicin) was administered for 6 weeks, but the patient continued to exhibit symptoms of heart failure related to symptomatic aortic insufficiency. On day 14 after admission, surgical treatment was indicated with the aortic valve being replaced with a bioprosthesis. On postoperative day 5, the patient had symptoms of

sudden intense abdominal pain in the left upper quadrant, profuse sweating, and hypotension that were apparently unrelated to the surgical procedure. Emergency abdominal tomography showed signs of splenic rupture (Fig. 2). The patient underwent splenectomy with a large volume of bloody collection (estimated at 3,000 mL) from the abdominal cavity without evidence of purulence. He received 10 units of concentrated red blood cells and hemodynamic support (vasopressor), which restored his hemoglobin levels from 5.3 g/dL to 10.3g/dL without any new episodes of bleeding.



Fig. 1. Computed tomography scan: Hypodense image (wedge) in the upper region of the spleen, compatible with splenic infarction (arrow)

3. DISCUSSION

Splenic rupture due to septic emboli with ischemic infarction in the presence of infective endocarditis (IE) due to *E. faecalis* is a rarely described entity [4,5]. Although, infective endocarditis related to splenic rupture is associated with high mortality rate [5,6] in our case, despite the clinical presentation of hemorrhagic shock, the clinical outcome was favorable. *E. faecalis* is a Gram positive bacterium that is susceptible to penicillin in most

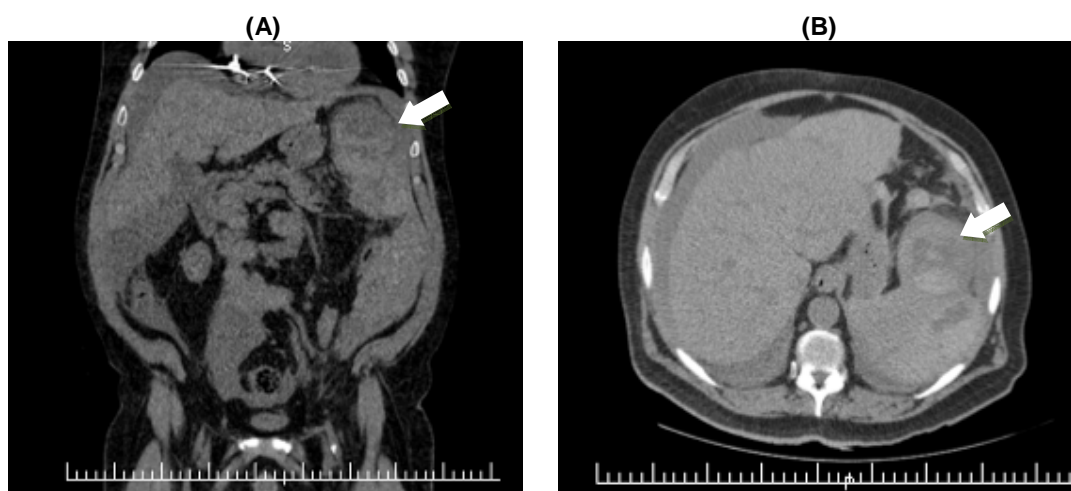


Fig. 2. Computed Tomography scan: A) Irregularities of the splenic contour and massive heterogeneous collections suggestive of rupture (arrow). B) Solution of continuity of the splenic parenchyma

cases, with a minimal inhibitory concentration (MIC) of < 8 mg/dL. IE due to *E. faecalis* generally presents as a subacute infection associated with central venous catheters in intensive care units. Similarly to other descriptions [7,8], the rate of enterococcus related with native valve endocarditis is more common (14-17%) in patients over 60 years than in younger. Embolic complications occur in 20–50% of cases, and are usually due to *Staphylococcus aureus* [9]. The clinical presentation varies depending on the organ involved: central nervous system (38%), spleen (30%), kidneys (13%), lung (10%), peripheral arterial system (6%), mesenteric system (2%), and coronary arteries (1%)[10]. Vegetations > 15 mm, mobile and pedunculated, have the greatest risk of embolization mainly being observed in IE due to *S. aureus* [11]. In this case, one of the vegetations was 21 mm, with a high embolic potential. Antibiotic therapy was guided by the antibiogram, with an MIC of < 2 mg/dL, suggesting high sensitivity to the antibiotic. Despite treatment to reduce the recurrence of the inflammatory response and septic embolism, a spontaneous rupture of the spleen occurred at week 3 of treatment. There are three pathophysiological mechanisms of splenic rupture in bacterial endocarditis: rupture of an intrasplenic vessel with hematoma formation rupture of a mycotic aneurysm, subcapsular dissection related with infarcted area, or rupture of a splenic abscess secondary to occlusion of a splenic vessel by embolized vegetations [12]. In our case, as there was an image of splenic

infarction in the initial CT scan (Fig. 1), hemorrhagic transformation of the infarcted area was the most likely mechanism of splenic rupture.

Clinicians should have a high degree of suspicion in patients with IE presenting in shock and with evidence of intraperitoneal hemorrhage. Timely diagnosis and surgical management are crucial to reduce the mortality of this complication [7]. Therefore, splenic rupture associated with hemorrhagic shock requires surgical emergency (splenectomy). Postoperatively, these patients are at an increased risk of infection from encapsulated bacteria (*Streptococcus pneumoniae* and *Neisseria meningitidis*), which is why prophylactic vaccination is recommended [13].

4. CONCLUSION

This case highlights the importance of both clinical surveillance and imaging to detect the presence of splenic infarction, particularly in patients with abdominal pain, and for the early detection of parenchymal changes that may suggest the risk of rupture.

CONSENT

All authors declare that written informed consent was obtained from the patient for publication of this case report and accompanying images. After listening to a transcript of this case report in

native language, the patient gave written consent to the authors to publish this case report.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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