

Endocarditis Caused by Enterococcus Leading to Spondylodiscitis: Case Report and Literature Review

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Introduction

Spondylodiscitis (SD) is rarely observed as a complication of infective endocarditis (IE) and the frequency of this association may range from 0.6 to 2.2%. IE patients were screened for SD and 15% were diagnosed with such spinal impairment, according to the literature.¹ The symptoms of SD associated with endocarditis can be as mild as back pain and myalgia or as severe as septic arthritis. The most feared complication of SD is disability due to residual neurological injury or severe pain that can occur in up to one third of the cases. Differential diagnosis with chronic rheumatic diseases is often difficult.^{1,2}

Enterococcus faecalis is the third most frequent cause of IE, behind *Streptococci* and *Staphylococci*, and rarely causes osteoarticular infections. Similarly, *Enterococcus faecalis* is a rare cause of SD associated with IE.³

This case presents a rare association between IE by *Enterococcus faecalis* and SD. It also presents the description, evolution and complications of the clinical picture.

Case Report

GFS, male, 58 years old, with onset of hyporexia and weight loss about five months before admission, complained of chills associated with sharp diffuse abdominal pain and dyspnea. Cardiac auscultation revealed systolic murmur (3+/6+) more audible in the mitral area and diastolic murmur (1+/6+) more audible in the aortic area. Abnormal inflammation markers (CRP and ESR) and other non-specific laboratory tests with abnormalities. The patient had a history of two ischemic strokes. The last event was concomitant with the onset of symptoms. Abdominal computed tomography scan showed splenic infarction and low kidney uptake. Transthoracic echocardiography at the bedside revealed vegetation in the mitral and aortic valves. After collection of blood samples and initiation of antibiotics with ceftriaxone and vancomycin, transesophageal echocardiography was performed, detecting mitral vegetation of 13 x 3.7 mm

(Figure 1A) and aortic valve of 14 x 5 mm (Figure 1B). Three days later, both blood samples revealed growth of *Enterococcus faecalis*.

Surgical planning was initiated in the cardiac intensive care unit. The patient complained of back pain refractory to opioid analgesics. Nuclear magnetic resonance imaging of the lumbar spine showed P1 signal hypointensity of the surfaces near vertebral discs L5 and S1 which is enhanced by paramagnetic contrast agents. Slight diffuse protrusion of disc L5-S1 with slight compression of the front face of the dural sac and decreased left neural foramen amplitude with signs of inflammatory/infectious SD (Figure 2).

Gentamicin was added to the initial treatment with antibiotics and three days after the start, the patient reported no further back pain. Bivalvar replacement surgery was performed and turned out to be lengthy due to heavy wear of mitral and aortic valves. In the post-operative period, the patient had refractory cardiogenic shock and died.

Discussion

Association of SD with endocarditis was first described in 1965 by Sèze et al. This association has a variable frequency in the literature, which has been estimated to range between 0.6 and 15%.⁴ The clinical characteristics described in this case are quite overlapping to that found in review studies, including age (>55 years) and sex (mainly males) and the symptoms presented are also similar to those available in the literature: localized pain is usually the case before diagnosis of IE, and the lumbar area is the most affected one.^{2,4}

In our case, IE leading to SD was caused by *Enterococcus faecalis*. Osteoarticular infections caused by *Enterococcus* are rare, with few cases described in the literature. This particularity stems from Enterococcal virulence factors, particularly adhesion molecules that are part of their cell wall and have a particular tropism for cardiac valves and for renal epithelial cells, which is not true regarding the bone.⁵⁻⁷

However, the need for valve replacement seems to be more frequent in patients with IE who develop SD. Prognostic factors include associated comorbidities (heart failure, neurological events, renal failure or HIV infection), development of perivalvular infection or myocardial abscess, as well as the intrinsic virulence of the agent, which certainly determine morbidity and mortality and high rates of recurrence.⁶

Of the laboratory parameters, high ESR is useful in the diagnosis of septic SD as it demonstrates an association with infectious processes, such as IE, and it should be interpreted in conjunction with the clinical picture and neurological and radiological abnormalities. Disc space narrowing

Keywords

Endocarditis/complications; *Enterococcus faecalis*; Echocardiography, Transesophageal; Mitral Valve/surgery.

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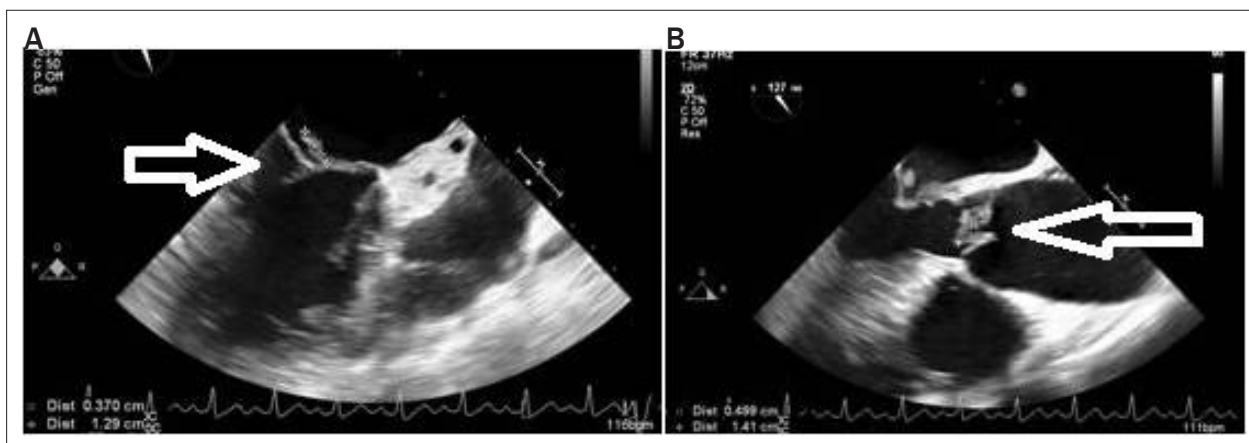


Figure 1 – A: Arrow indicating vegetation in mitral valve. B: Arrow indicating vegetation in aortic valve.

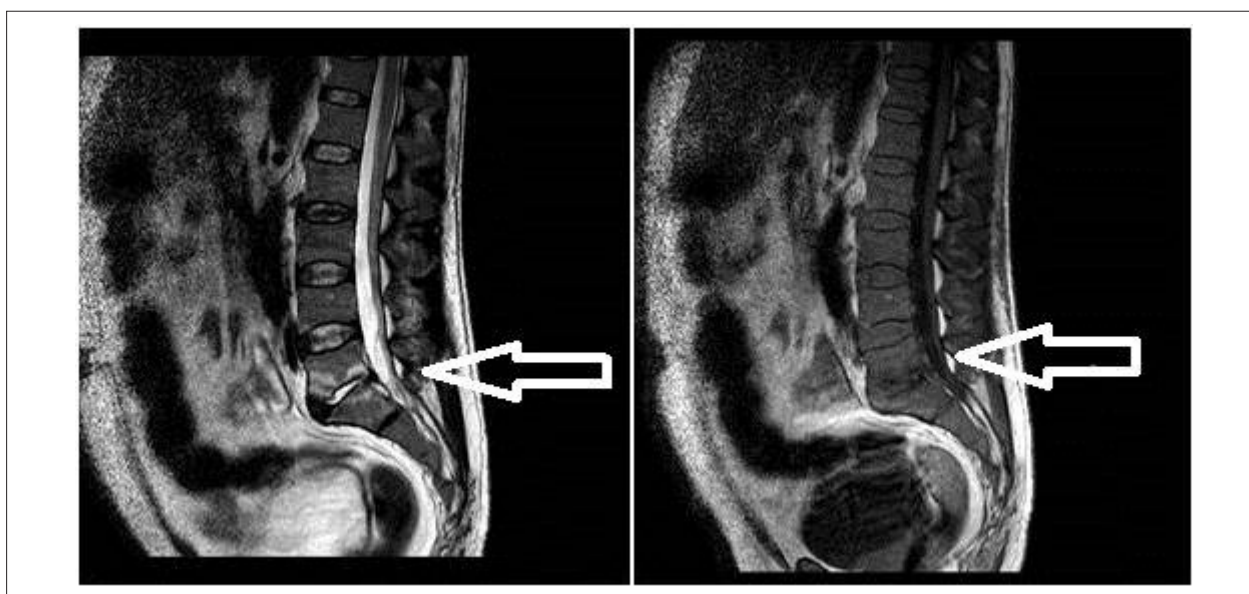


Figure 2 – Arrows indicating intervertebral disc injury and vertebral body destruction.

is characteristic and can be seen on plain radiography from two to four weeks after the onset of symptoms. Radiography can reveal reactive sclerosis of adjacent vertebral plateaus (eight weeks), osteogenesis (12 weeks) and bone fusion (six months). Tomography can show details such as lytic lesions in disc space and paraspinal, epidural and psoas muscle abscess.⁷

High-resolution computed tomography (CT), bone scintigraphy and nuclear magnetic resonance imaging (NMR) are important in early diagnosis, especially in assessing the extent of the infection and diagnosing paraspinal abscess. High-resolution computed tomography may show early bone lesions, while NMR has a diagnostic sensitivity of 96%. These are the diagnostic methods of choice and for outpatient follow-up of vertebral complications.^{8,9}

Hematogenous SD can occur in 1:100,000 to 1:25,000 in the adult population (4% to 16% of all bone infections of hematogenous origin), especially in males, in adults older than 50 (41% to 75%) and preferably in the lumbar spine. Differential diagnosis of SD with renal colic is often difficult. However, we have noticed that in SD, pain is continuous, with painful palpation of the spinous processes while in renal colic, the pain is reported to be in the costovertebral angle, usually associated with urinary symptoms and fever. Drug treatment should be guided by the cultures. Antibiotic therapy must be longer in cases of SD associated with IE (6 weeks to 3 months).⁹⁻¹¹

As to the pathogenesis, some authors have suggested that the dorsal pain is due to microembolia of immune complexes with or without bacteria. In SD associated with IE, the mortality rate is still relatively high, while in SD alone it is almost zero.¹²

Case Report

Conclusion

The presence of SD in patients affected by IE is a rare event and it should be suspected when there is concomitant persistent lumbar pain with a compatible clinical picture. Once the most common causes of lumbar pain are ruled out, such as musculoskeletal impairment and renal lesions, diagnosis of SD should be readily delivered with imaging tests, as it represents poor prognosis. Despite the importance of diagnosis of SD, initiation of antimicrobial therapy should not be delayed as the prognosis of patients is much more associated with valve destruction and hematogenous embolization than with SD.

Authors' contributions

Data acquisition: Guimarães Neto WP, Silveira LFD, Brite MRS; Manuscript drafting: França JCQ; Critical review of

the manuscript for important intellectual content: Godoy MF, Araújo RB.

Potential Conflicts of Interest

There are no relevant conflicts of interest.

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