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CASE REPORT
Valvular heart disease

Vertebral osteomyelitis secondary to infective endocarditis detected by repeated magnetic resonance imaging: a case report

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Background Patients with infective endocarditis (IE) experience various symptoms, a major one being back pain, which is occasionally caused by concomitant vertebral osteomyelitis (VO). Magnetic resonance imaging (MRI) is generally used to detect VO; however, the sensitivity of detection using MRI is very low in the early stages of VO.

Case summary A 60-year-old man visited our hospital with complaints of fever and persistent back pain over the past 7 days. A holosystolic heart murmur was auscultated, and an echocardiography revealed a vegetation on the posterior mitral leaflet. Blood cultures were positive for *Streptococcus sanguinis*. He was diagnosed with IE and treated with antimicrobials. A lumbar spine MRI on Day 1 showed no clear signs of vertebral infection, but the back pain continued and gradually worsened. Magnetic resonance imaging retest on Day 8 showed high signal intensity within the lumbar vertebral bodies and the disk on T_2-weighted sequences, indicating VO. Intravenous antimicrobial therapy was extended, followed by oral antimicrobials, and a corset was put on to protect the lumbar spine to prevent bone degradation.

Discussion For persistent back pain in IE patients, repeat MRIs at regular intervals of time can detect possible vertebral infection even if signs of vertebral infection were absent on the initial MRI.

Keywords Infective endocarditis • Back pain • Echocardiography • Magnetic resonance imaging • Vertebral osteomyelitis • Case report

ESC curriculum 4.11 Endocarditis • 2.2 Echocardiography • 4.3 Mitral regurgitation

Learning points

- Magnetic resonance imaging (MRI) is a useful imaging method with high sensitivity for establishing the diagnosis of vertebral osteomyelitis (VO) associated with infective endocarditis. However, the advantage tends to be lost in the early phases of the disease.
- Magnetic resonance imaging should be repeated after some interval to detect concomitant VO in a case of infective endocarditis with persistent back pain, even if there were no signs of vertebral infection in an initial MRI. A high index of suspicion for VO should be kept in such a setting.

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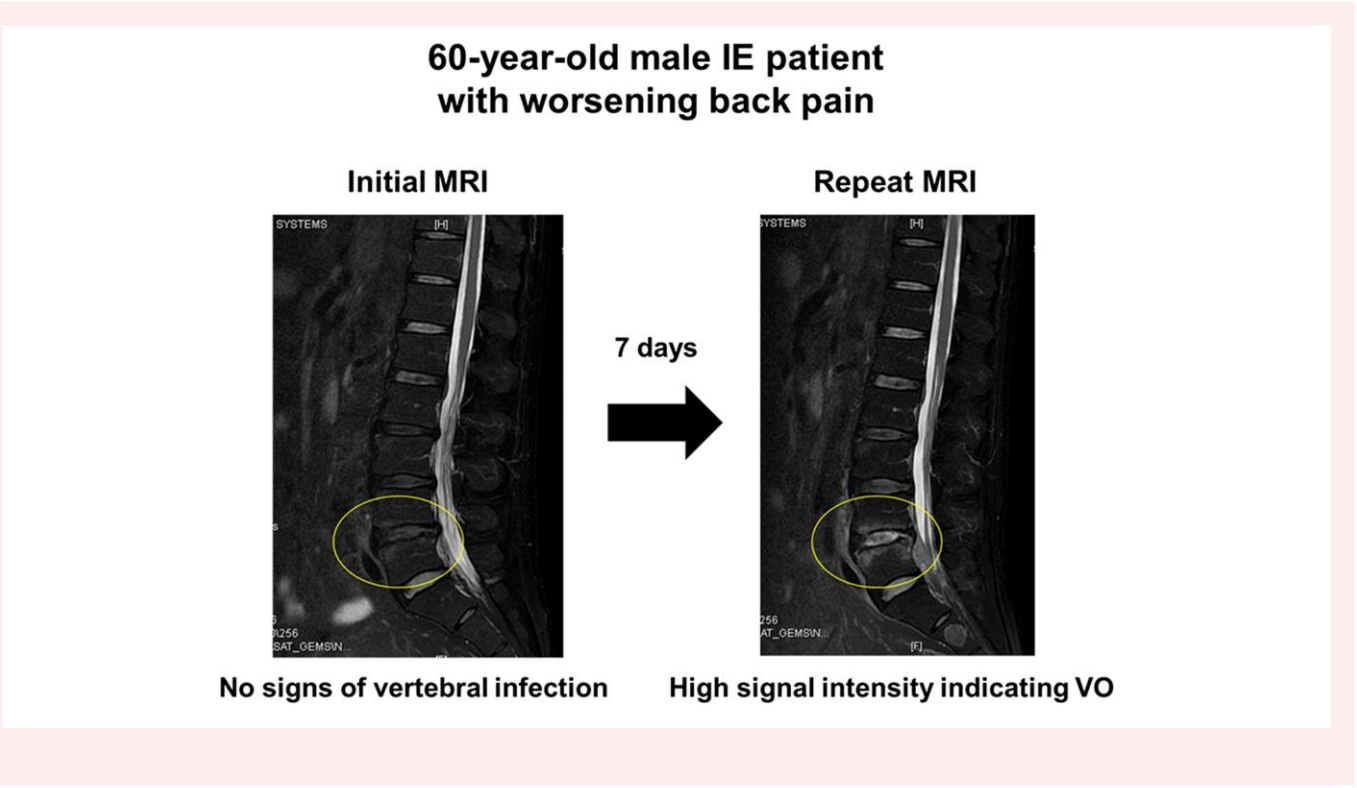
Introduction

Back pain is a common symptom in patients with infective endocarditis (IE) and is usually caused by concomitant vertebral osteomyelitis (VO).[1] Infection of the spine, if not treated properly, may cause bone destruction, spinal cord compression, and subsequent neurologic deficits. Therefore, appropriate diagnosis is critical for the favourable clinical outcomes in patients with VO. Vertebral osteomyelitis can be diagnosed using magnetic resonance imaging (MRI) with high sensitivity and specificity[2]; however, this imaging modality may fail to reveal early signs of VO. Herein, we report a case of IE with back pain caused by VO, which was diagnosed following repeated MRI evaluation. With accurate diagnosis, the patient could be treated successfully without any neurologic manifestations.

Summary figure



Case presentation

A 60-year-old man presented to our hospital with a fever of 38.9°C and worsening back pain over the last 7 days. His medical history included hypertension and arrhythmia (details unknown). He had never undergone transthoracic echocardiography (TTE). His regular medication included amlodipine, telmisartan, and hydrochlorothiazide. His blood pressure was 105/71 mmHg, pulse rate was 107 b.p.m., respiratory rate was 20 breaths/min, and oxygen saturation was 97%. Cardiac auscultation detected a holosystolic murmur over the mitral area. Blood examination demonstrated elevated C-reactive protein level (10.91 mg/dL, normal range 0–0.5 mg/dL, *Figure 1*) and slightly elevated liver enzymes [alanine aminotransferase 47 U/L (normal range 8–40 U/L) and aspartate aminotransferase 37 U/L (normal range 7–38 IU/L)] but normal creatinine level (1.00 mg/dL, normal range 0.61–1.04 mg/dL). His blood count showed mild thrombocytopenia [leucocytes 66×10^9/L (normal range 40–100×10^9/L), haemoglobin 15.9 g/dL (normal range 13.2–17.2 g/dL), and platelet 140×10^9/L (normal range 150–400×10^9/L)]. His chest radiograph was normal. Transthoracic echocardiography (*Figure 2* and *Videos 1* and *2*) and transoesophageal echocardiography (TOE, *Figure 3* and *Video 3*; Supplementary material online, *Video S1*) showed moderate-to-severe mitral regurgitation (MR; regurgitant volume: 30 mL/beat; regurgitant fraction: 50%; a wall-impinging jet that refluxes into nearly half of the left atrium) due to a rupture of the chordae tendineae attached to the left atrial aspect of the P2 segment (middle scallop) with an oscillating mass, indicating a vegetation (up to 16 mm on TOE). Four sets of blood cultures showed growth of gram-positive cocci within 24 h. The patient satisfied two major criteria as per the modified Duke criteria: four sets of positive blood cultures and an echocardiogram with oscillating intracardiac mass, which classified him as having definite IE.[3] He did not claim any symptom of heart failure (New York Heart Association Class I). Upon admission to our hospital with a diagnosis of IE, he was empirically treated with sulbactam/ampicillin (12 g/day) and ceftriaxone (2 g/day), which were deescalated to penicillin G (2400 million units/day) and gentamicin (160 mg/day) on Day 6 after the identification of *Streptococcus sanguinis* in the blood cultures. He was haemodynamically stable without any signs of heart failure, and the infection was well controlled. Therefore, antibiotic therapy was continued under careful observation with valvular surgery on hold.

To examine the cause of his back pain, a lumbar spine MRI was performed on Day 1, but no remarkable findings of vertebral infection were detected (*Figure 4A*). The fever and C-reactive protein level subsided on Day 4 (*Figure 1*), and repeated blood cultures performed 3 days after starting antimicrobials were negative, suggesting effectiveness of the antimicrobial therapy. Nevertheless, the patient's back pain persisted and gradually worsened. A repeat lumbar spine MRI performed on Day 8 revealed high signal intensity within the margins of

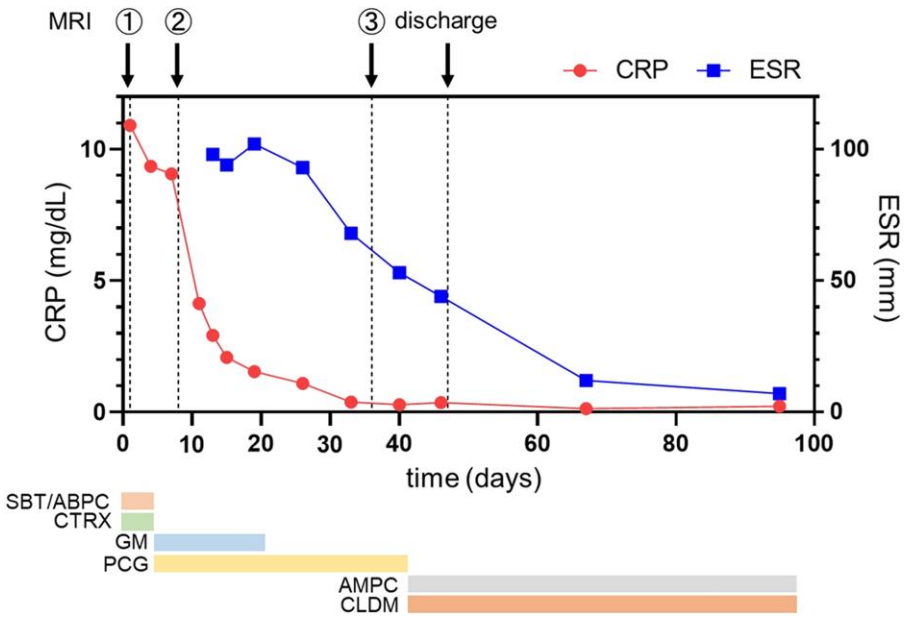


Figure 1 Clinical course of the patient. MRI, magnetic resonance imaging; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; SBT/ABPC, sulbactam/ampicillin; CTRX, ceftriaxone; GM, gentamicin; PCG, penicillin G; AMPC, amoxicillin; CLDM, clindamycin.

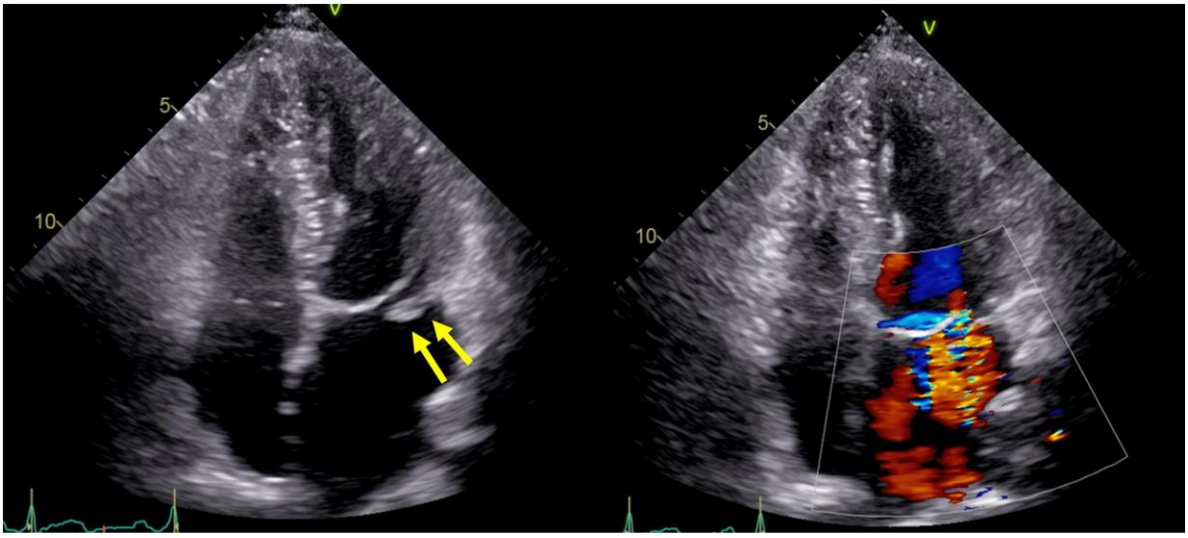
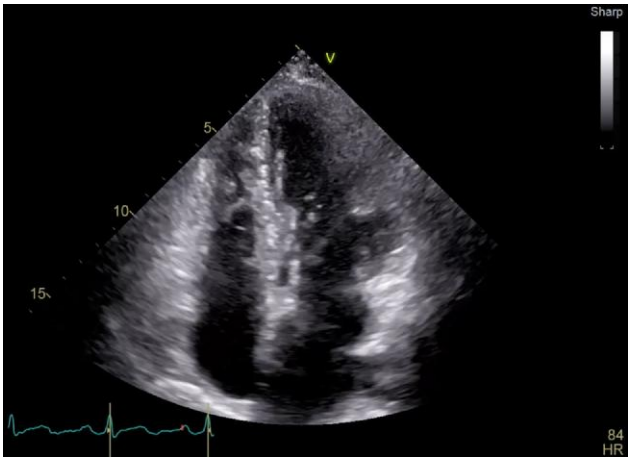
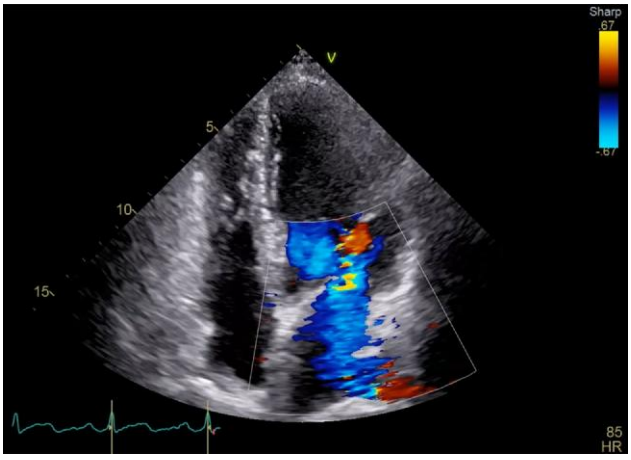


Figure 2 Initial transthoracic echocardiography showing moderate-to-severe mitral regurgitation, ruptured chordae tendineae attached to the P2 segment, and a vegetation (arrows).

the L4 and L5 vertebral bodies and the intervertebral disk on T_2-weighted images, accompanied by a nodal mass at the dorsal aspect of the L5 vertebral body (*Figure 4B*). The patient was diagnosed with VO at the L4–L5 level accompanied with an epidural abscess. He was recommended bed rest wearing a customized corset for prevention of bone degradation. The patient was treated with penicillin G and gentamicin for the first 2 weeks and then with penicillin G alone for the next 4 weeks. His lumbar pain gradually subsided, and on Day 36, a lumbar spine MRI revealed resolution of the lumbar spine abscess, although the area of high signal intensity persisted (*Figure 4C*). The patient was discharged on Day 47. Since the patient had an epidural abscess (which is a risk factor for recurrent VO),[4] a further 8-week treatment with oral antibiotics was recommended, after which his inflammatory markers returned to normal on Day 95 (*Figure 1*). Follow-up TTE (*Figure 5*; Supplementary material online, *Videos S2 and S3*) and TOE also showed the prolapsing P2 segment with a vegetation (up to 7 mm on TOE), severe MR (regurgitant volume: 70 mL/beat; regurgitant fraction: 62%), and a rupture of the chordae tendineae of the mitral valve. Although



Video 1 Initial transthoracic echocardiography showing ruptured chordae tendineae attached to the P2 segment, and a vegetation.



Video 2 Initial transthoracic colour doppler echocardiography showing moderate-to-severe mitral regurgitation.

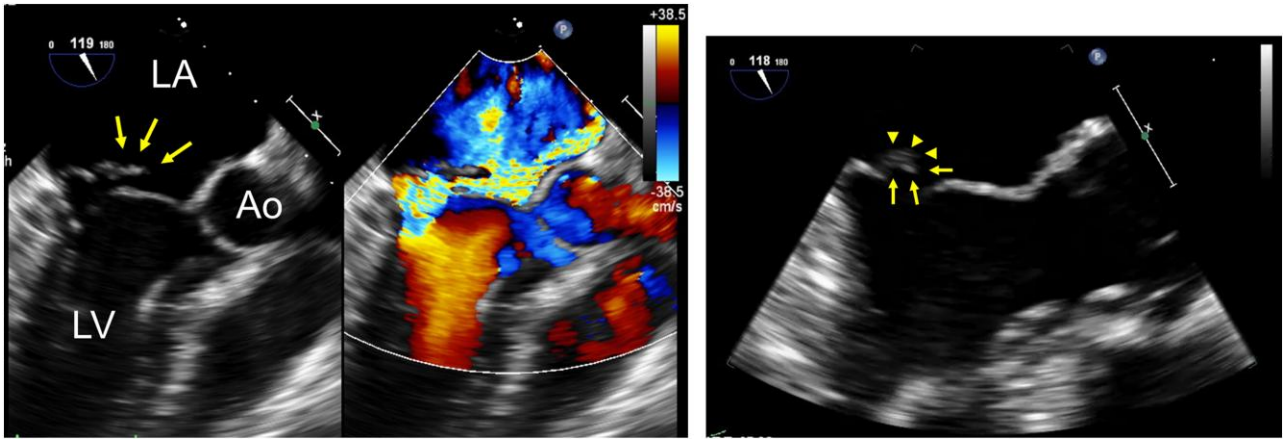
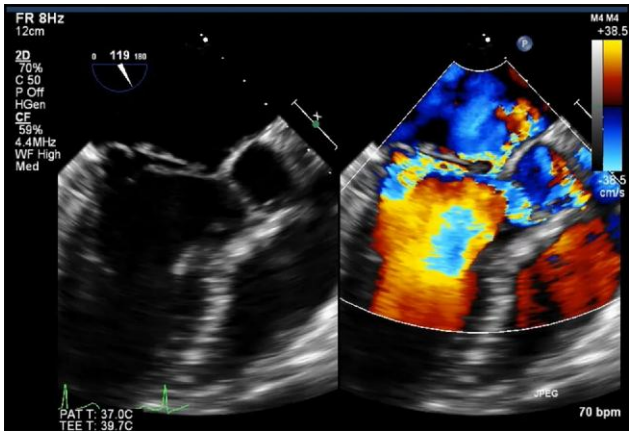


Figure 3 Transoesophageal echocardiography showing severe mitral regurgitation and ruptured chordae tendineae attached to the P2 segment (arrows) and a vegetation (up to 16 mm, arrowheads). Ao, aorta; LA, left atrium; LV, left ventricle.



Video 3 Transoesophageal echocardiography showing severe mitral regurgitation and ruptured chordae tendineae attached to the P2 segment, and a vegetation (up to 16 mm).

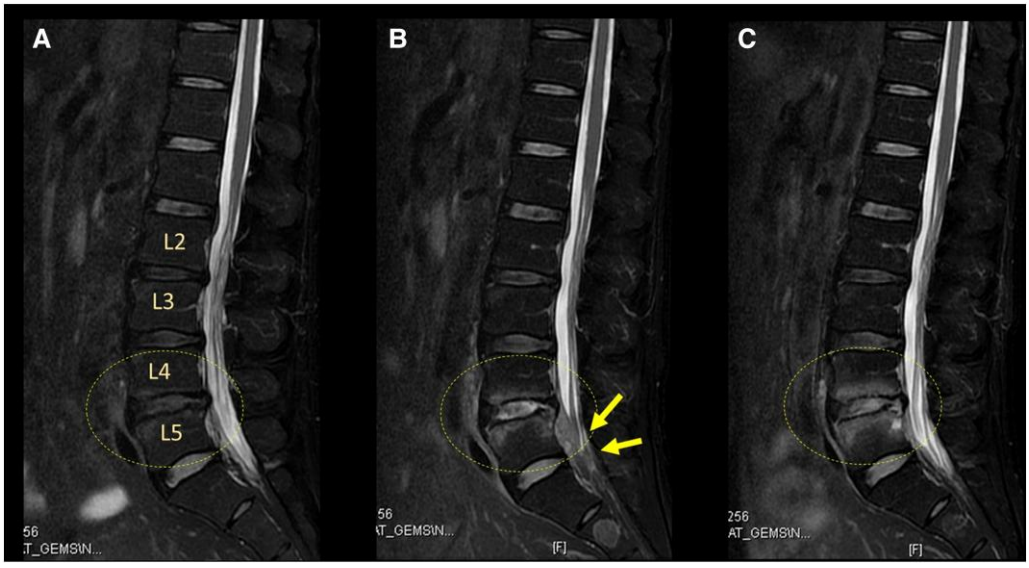


Figure 4 Magnetic resonance imaging findings. (*A*) On Day 1, no remarkable signs of infection are noted. (*B*) On Day 8, high signal intensity changes within the margins of the L4 and L5 vertebral bodies and the intervertebral disk on T_2-weighted images are noted. Epidural abscess is also seen (arrows). (*C*) On Day 36, the epidural abscess has disappeared. The area of high signal intensity has expanded.

surgical treatment was considered plausible, the patient did not consent to the surgery and has been visiting an outpatient clinic periodically for follow-up.

Discussion

In this report, we presented the case of a 60-year-old man with IE and back pain, which persisted despite effective antimicrobial therapy and was eventually found to be caused by VO. Patients with IE often complain of musculoskeletal symptoms, such as myalgia, articular pain, and low back pain.[1] Causes of back pain in patients with IE include renal or splenic infarction,[5] microembolization of immune complexes,[6] and vertebral infection such as VO, which is thought to be the result of bacterial embolization or haematogenous spread.[7] The incidence rate of IE is 1.5–11.6 per 100 000 person-years, and VO complicates IE with a frequency of 4.6–19%.[3,5,7–9] In patients with IE, the hospital mortality rate is higher among those with VO (12.7%) compared to those without (7.1%).[8] *Staphylococcus aureus* is reportedly the most frequent causative microorganism of IE with osteoarticular infection including VO.[9] In turn, more than half of patients with VO caused by *Streptococcus viridans* develop IE.[8] A diagnosis of concomitant VO is crucial in terms of the treatment. Bed rest wearing a customized corset is required for protection of spinal bodies from bone degradation,[10] and the duration of antibiotic therapy should be prolonged compared with native valve IE (6–8 and 2–6 weeks, respectively).[2,3]

In the present case, the echocardiography revealed moderate-to-severe MR and vegetation at the ruptured chordae tendineae of the

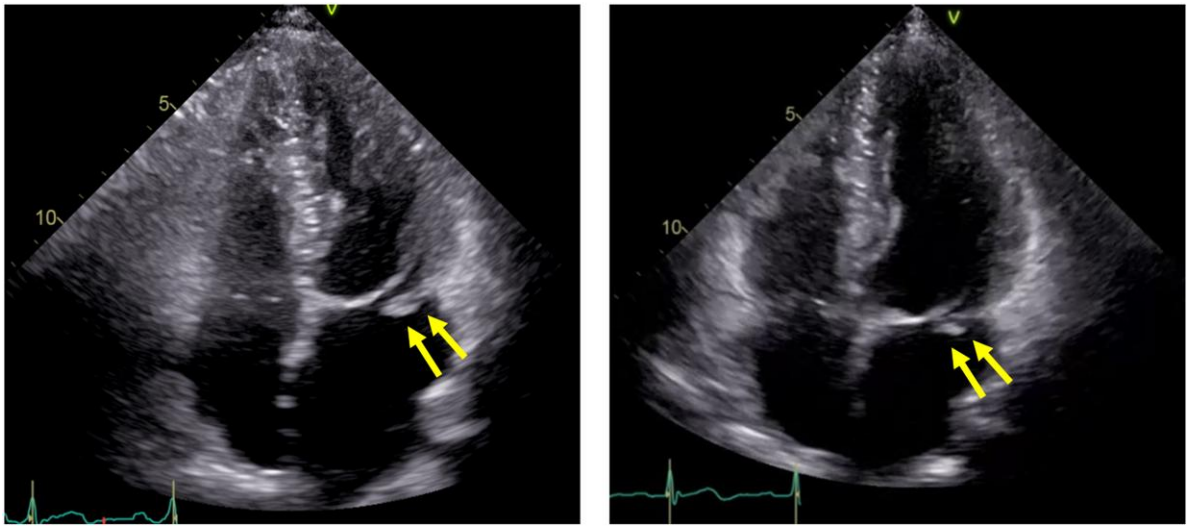


Figure 5 Initial (on Day 4) and follow-up (on Day 42) transthoracic echocardiography comparing the vegetation before and after antibiotic therapy.

posterior mitral leaflet. The rupture of the chordae tendineae was presumably present before the onset of IE because no heart murmur suggestive of valvular diseases that caused bacterial attachment had been detected during visiting a clinic before the onset. We assume that the patient had asymptomatic idiopathic rupture prior to the onset of IE, since mitral valve tendon ruptures are often idiopathic and asymptomatic,[11] and he did not have a history of connective tissue diseases or traumatic injury, which can be potential causes of mitral valve prolapse. Additionally, antibiotic therapy was continued under careful observation with valvular surgery on hold because he was haemodynamically stable without any signs of heart failure and the infection was well controlled. The European Society of Cardiology guidelines suggest that indications for surgery are recommended in patients with IE who develop refractory pulmonary oedema, cardiogenic shock, locally uncontrolled infection, or persistent vegetation >10 mm after one or more embolic episodes despite appropriate antibiotic therapy.[3] Meanwhile, subsequent surgical intervention likely contributes his better prognosis.[3,12]

Spine MRI is a useful imaging method for the detection of VO with high diagnostic value. The sensitivity of MRI is superior to that of computed tomography.[2,13] However, it should be noted that the sensitivity of the modality is limited in the early stages of VO.[14] Reportedly, 22.2% of patients with early VO (<14 days of symptoms) were at risk of being overlooked.[15] Repeating MRI after an interval of 8–22 days was reported to be useful in revealing signs of spinal infection.[14] Thus, in cases where patients have persistent back pain and a spine MRI does not reveal signs of VO, a repeat examination after a suitable interval may be necessary to enable a diagnosis to be made.

Conclusion

When treating patients with IE, it is important to routinely ask about back pain that may reveal VO. Such pain may be the result of an existing concomitant VO, which requires adequate treatment that includes absolute rest, use of a corset, and antimicrobial therapy for a sufficient duration. Even if signs of VO are not visible on an initial MRI in patients with IE with a short history of back pain, repeating MRI at regular intervals is effective in detecting VO. The timely diagnosis of VO in patients with IE ensures that correct treatment is provided, which improves patient's clinical outcomes.

Lead author biography

Masaki Hashimoto, MD, graduated from the University of Tsukuba, College of Medicine, in 2015. He completed his residency at Showa General Hospital, and he currently works at The University of Tokyo Hospital.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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Data availability

The data underlying this article will be shared upon reasonable request to the corresponding author.

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