



## Case Report

# Rapid diagnosis of prosthetic valve endocarditis from Janeway lesions in a transcatheter aortic valve implantation patient



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## ABSTRACT

Percutaneous transcatheter aortic valve implantation (TAVI), first introduced in 2002, is a viable solution for previously inoperable or high-risk patients with aortic stenosis, providing the benefit of valve replacement without the associated risks of surgery. When these patients develop prosthetic valve endocarditis (PVE), management is complicated, owing to their often, atypical presentations and baseline comorbidities. Moreover, it is often difficult to detect vegetations in such patients, even with transesophageal echocardiography. Here, we describe a case of post-TAVI PVE that was successfully treated medically after a rapid diagnosis was made based on physical examination. Our experience shows that physical examination continues to be important for rapid diagnosis of infective endocarditis, even in the era of structural heart disease intervention.

**<Learning objective:** Diagnosis of endocarditis in transcatheter aortic valve implantation patients can be difficult and may be delayed, as echocardiography is often inconclusive because of the unique physiology of these patients. We should maintain a low threshold for investigation of infective endocarditis in these patients, and more emphasis should be placed on physical examination to ensure rapid diagnosis and favorable clinical outcome.>

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## Introduction

In 2002, percutaneous transcatheter aortic valve implantation (TAVI) was first introduced as a viable solution for inoperable or high-risk patients with severe symptomatic aortic stenosis (AS) [1]. As TAVI became widespread, prosthetic valve endocarditis (PVE) began to draw notice as a critical potential sequela [2]. In a study of the largest multicenter registry of TAVI patients, the rate of PVE was 0.67% after 1.1 years of follow-up, similar to that among patients who underwent surgical aortic valve replacement. However, the 1-year mortality rate associated with post-TAVI PVE was 66%, which is higher than for any other kind of infective endocarditis (IE) [3]. TAVI patients often have multiple comorbidities, which can

significantly complicate management and increase PVE morbidity and mortality [4,5]. Rapid diagnosis with appropriate treatment is crucial for these patients, but the presence of multiple comorbidities, along with the prosthetic nature of the implanted valve, may make this difficult. We here describe a case of PVE after implantation of a CoreValve prosthetic valve (Medtronic, St Paul, MN, USA) that was successfully treated as a result of rapid diagnosis based on physical examination.

## Case report

The patient was a 79-year-old male emergently admitted to our hospital with persistent fever, disturbance of consciousness, and malaise. He had a history of chronic lung disease and coronary artery bypass surgery for angina, and 20 months prior to presentation, he had undergone transfemoral TAVI with a 29-mm Medtronic CoreValve prosthesis for severe symptomatic aortic stenosis. He had type II diabetes mellitus and denied the presence of common risk factors associated with IE, including recent dental

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treatment, previous rheumatic fever, and intravenous drug misuse. On admission, he appeared generally ill, with a temperature of 39.3 °C, a Glasgow Coma Scale score of 13, and a blood pressure of 154/80 mmHg. Initial laboratory tests showed leukocytosis with a predominance of neutrophils, as well as an elevated C-reactive protein level (21.6 mg/dL). Transthoracic echocardiography (TTE) showed no obvious vegetation and only trivial paravalvular aortic regurgitation, with no significant changes compared with the original post-TAVI TTE. T2-weighted brain magnetic resonance imaging (MRI) showed only age-appropriate old lacunar infarcts in the right caudate nucleus. On the second hospital day, physical examination revealed Janeway lesions on the fingers (Fig. 1), but no conjunctival hemorrhage, Osler's nodes, or Roth's spots. Transesophageal echocardiography (TEE) did not show any obvious vegetation (Fig. 2).

Although the patient did not fulfill any of the major modified Duke criteria for IE, 3 minor criteria (a fever of 39 °C, the predisposing effect of the prosthetic valve, and Janeway lesions) were present, indicating its possibility. Therefore, intravenous administration of vancomycin and gentamicin was initiated immediately. On the third hospital day, 3 of 4 blood cultures taken on the admission day were found to be positive for methicillin-susceptible *Staphylococcus aureus*, and vancomycin was switched to cefazolin. On the fourth hospital day, diffusion-weighted brain MRI showed high signal intensity in the bilateral frontal lobe and cerebellar hemisphere (Fig. 3), which suggested

multiple acute cerebral infarcts caused by embolisms. A second TEE, performed 2 weeks after admission, showed mobile vegetation on the leaflets of the aortic prosthesis but indicated a well-functioning prosthetic valve with trivial paravalvular aortic regurgitation (Fig. 2). A third TEE, 3 weeks after admission, confirmed resolution of the vegetation without impaired prosthesis function. A series of MRIs found no obvious endocarditis-related cerebral aneurysms. The clinical outcome was good, with resolution of the fever, return to normal levels of inflammatory markers, and disappearance of the peripheral stigmata of IE. After 6 weeks of intravenous cefazolin, the patient was discharged. Over a follow-up period of 1 year, there was no recurrence of PVE.

## Discussion

The clinical course of the present patient provided important clinical suggestions regarding post-TAVI PVE. Our findings suggested that the modified Duke criteria for IE might also be useful for the diagnosis of post-TAVI PVE; however, detecting vegetation, even with TEE, is often difficult in such patients. Physical examination is important and especially critical for rapid diagnosis. In this case, the presence of Janeway lesions was an excellent clue to the diagnosis and aided in the successful treatment of our patient.

The number of TAVI procedures performed yearly worldwide has grown exponentially since the first TAVI was performed in 2002 [1], making post-TAVI PVE an increasingly important problem. Surgical intervention is not feasible in the majority of post-TAVI PVE patients owing to their multiple comorbidities. A recent study reported that at least 1 complication requiring reintervention according to the guidelines occurred in 86.8% of patients with post-TAVI PVE, including systemic emboli in 12.2%. However, the rate of reintervention was as low as 11.3%—one of the lowest rates of reintervention in PVE cases ever reported—and the in-hospital mortality rate was 47.2%, which increased to 66% at the 1-year follow-up [3]. Thus, a delay in diagnosis can be disastrous. The modified Duke criteria for IE may well be useful for the diagnosis of post-TAVI PVE, although no study has yet confirmed their usefulness. However, our experience demonstrates that signs visible on echocardiograms are not always necessary to make a diagnosis. Furthermore, as mentioned above, detecting vegetations with echocardiography, especially small ones, can be quite difficult in TAVI patients; it has been reported that the sensitivity and specificity of TEE and TTE for the diagnosis of IE are lower in patients with prosthetic valves than in those with native valves [6]. In the TAVI procedure, the calcified native valve is not removed, and the native leaflets are compressed in the Valsalva sinus. Moreover, reflectance and shadowing due to aortic root calcification and the metal frame of the prosthesis make it difficult to detect vegetations echocardiographically in TAVI patients, even with TEE. A large multicenter registry reported that echocardiographic findings revealed the presence of vegetations in only 77% of patients with post-TAVI PVE; however, this rate may have been overestimated because doubtful cases were not included [3].

Fever (71.7%) and heart failure (58.5%) have been reported to be the most common symptoms of IE [3]. Additionally, peripheral stigmata are less frequent than they once were [7,8], with Janeway lesions present in 2.4–4.6% of cases, Osler's nodes in 2.9–4.4% of cases, Roth's spots in 1.3–1.9% of cases, and splinter hemorrhages in 8.0% of cases [7,9]. However, these prevalence rates may be underestimated, as standard procedures do not include systematic dermatological examinations or photography for retrospective assessment [8]. In the present case, the patient's Janeway lesions, which were visible before IE were confirmed by echocardiography and MRI, were a critical clue, enabling rapid diagnosis and successful treatment. Our experience shows that physical examination for

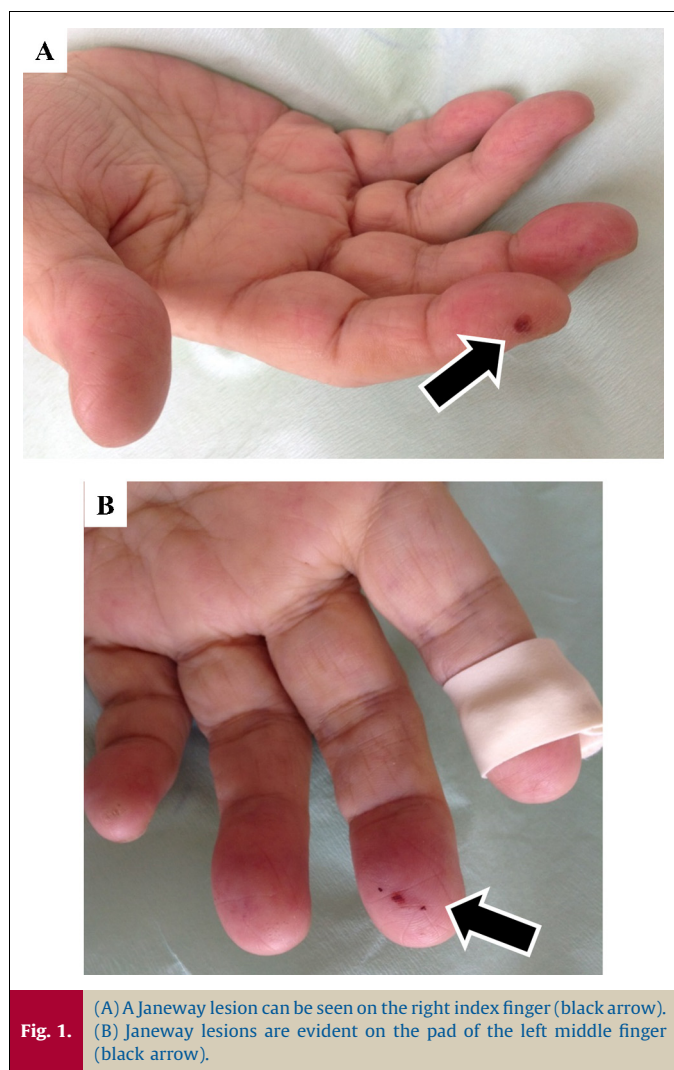
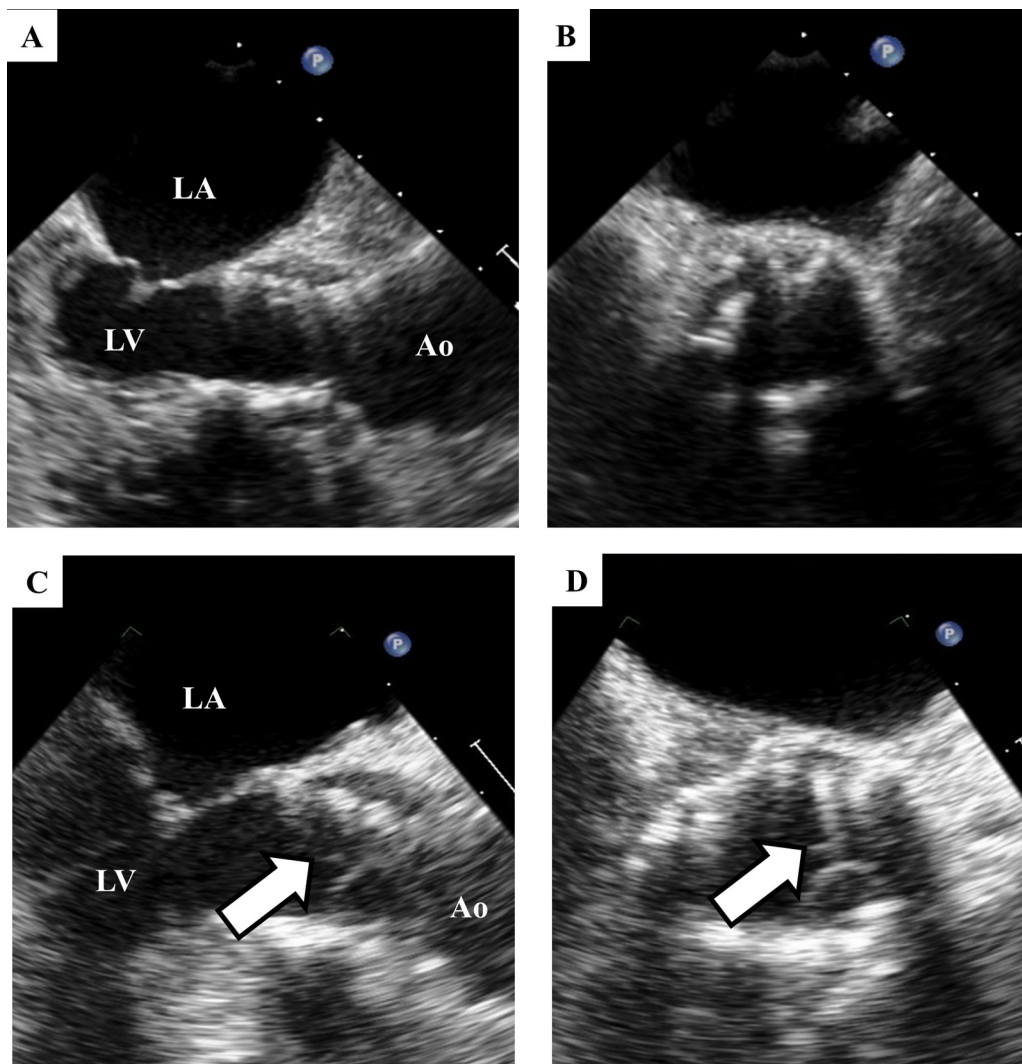
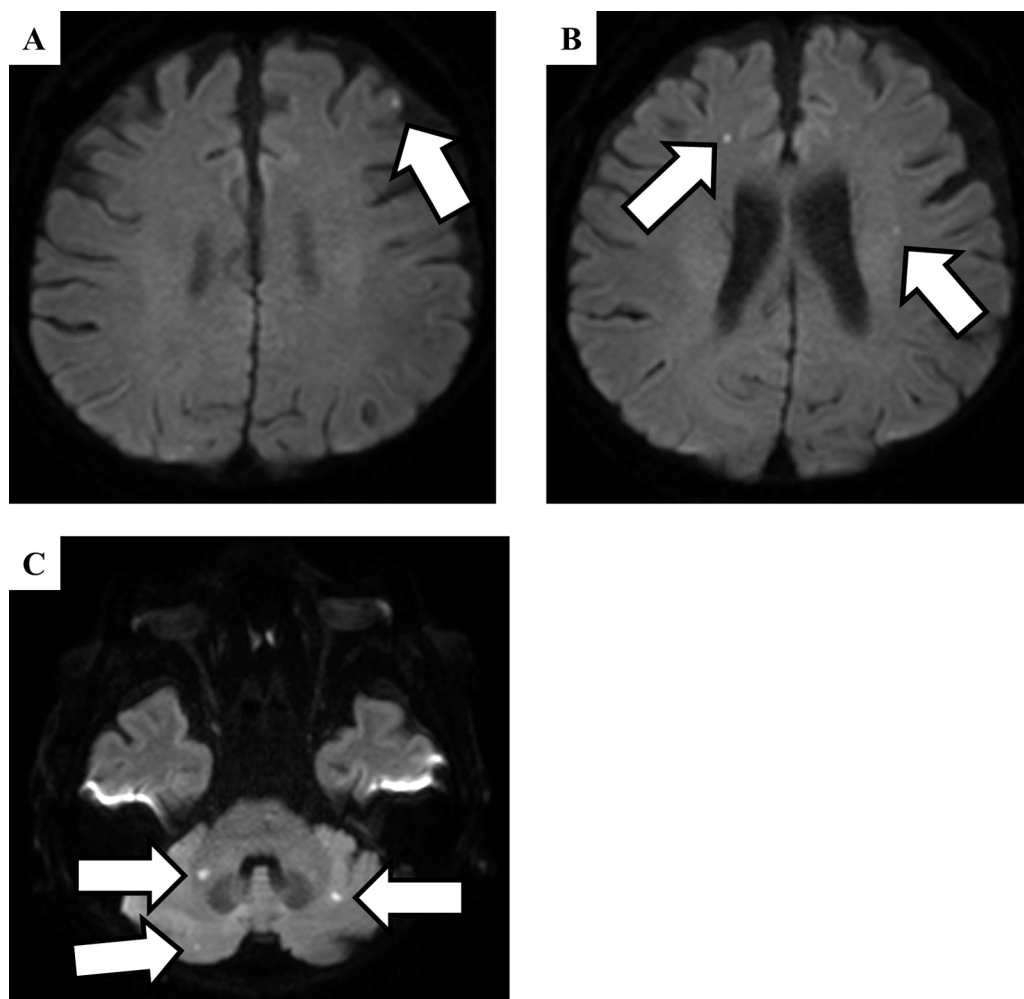


Fig. 1. (A) A Janeway lesion can be seen on the right index finger (black arrow). (B) Janeway lesions are evident on the pad of the left middle finger (black arrow).

**Fig. 2.**

(A and B) The first transesophageal echocardiography (TEE), performed on the second hospital day, did not show any obvious vegetation. (C and D) A second TEE, performed 2 weeks after admission, confirmed mobile vegetation on the leaflets of the aortic prosthesis but indicated a well-functioning prosthetic valve with trivial paravalvular aortic regurgitation (white arrows). LA, left atrium; LV, left ventricle; Ao, aorta.



**Fig. 3.** (A–C) Diffusion-weighted brain magnetic resonance images on the fourth hospital day showed bright signal intensity in the bilateral frontal lobe and cerebellar hemisphere (white arrows).

peripheral stigmata continues to be important for rapid diagnosis of IE, particularly post-TAVI PVE, even in the era of structural heart disease intervention.

### Conflict of interest

The authors declare that there is no conflict of interest.

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