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Marked changes in bioprosthetic valve thrombosis by anticoagulation therapy

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Case description

A 74-year-old woman presented to our hospital with dyspnoea on exertion. She underwent mitral valve replacement (MVR) with a 27-mm-stented porcine valve and coronary artery bypass surgery for ruptured mitral chordae tendineae following inferior acute myocardial infarction 2 years ago. Postoperatively, she had been treated with vitamin K antagonist (VKA) and aspirin for

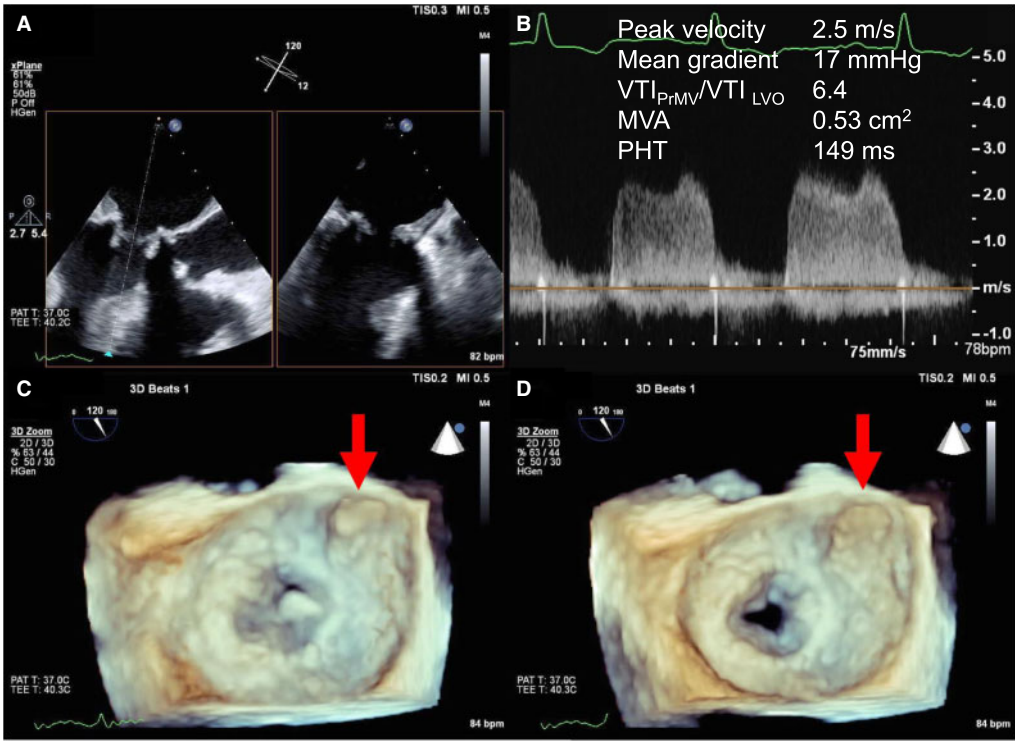

Figure 1 Simultaneous multiplane imaging of two-dimensional transoesophageal echocardiography images (*A*) and three-dimensional transoesophageal echocardiography images in systole (*C*) and diastole (*D*) on admission. The red arrows indicate thrombus in the atrial septum. The peak early mitral velocity and the mean transmitral gradient increased to 2.5 m/s and 17 mmHg, respectively (*B*). LVO, left ventricular outflow tract; MVA, mitral valve area; PHT, pressure half time; PrMV, prosthetic mitral valve; VTI, velocity-time integral.

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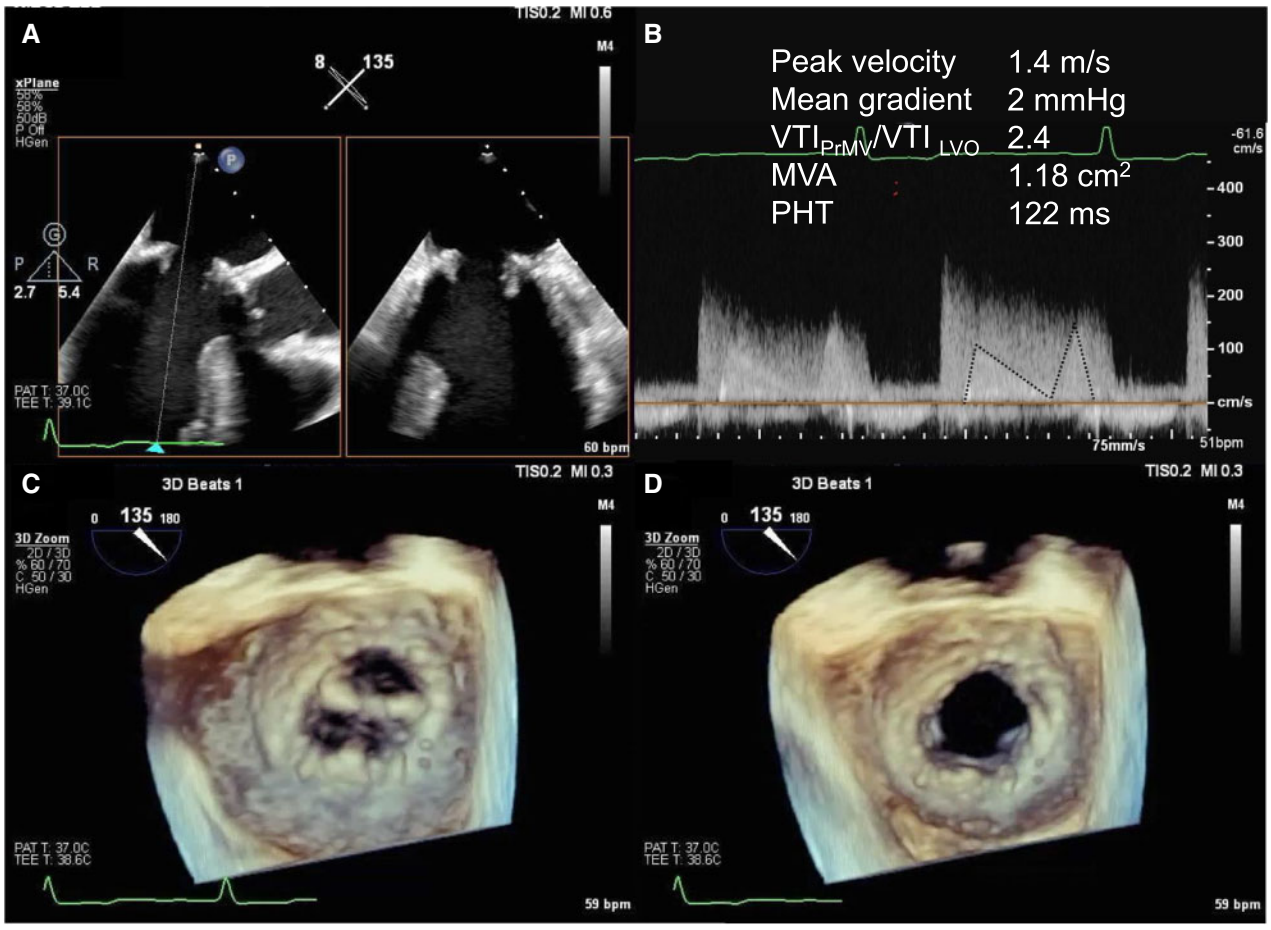

Figure 2 Simultaneous multiplane imaging of two-dimensional transoesophageal echocardiography images (*A*) and three-dimensional transoesophageal echocardiography images in systole (*C*) and diastole (*D*) after 3-month treatment with vitamin K antagonist. The mitral leaflet thickness significantly decreased and valve opening markedly improved. In addition, thrombus in the atrial septum disappeared. The peak early mitral velocity and the mean transmitral gradient markedly decreased to 1.4 m/s and 2 mmHg, respectively (*B*). Because she had moderate aortic regurgitation, the Doppler spectrum of the mitral valve flow overlapped with the Doppler spectrum of the aortic regurgitation flow (*B*).

3 months, and VKA was discontinued thereafter. Even though serial transthoracic echocardiography examination demonstrated a gradual increase in the mitral transvalvular gradient and the inferior wall motion abnormality, she did not have any symptoms and both ventricular function was preserved. Upon admission, transoesophageal echocardiography (TOE) revealed thickened mitral leaflets with restricted motion and low echoic mass extended to the left atrial septal wall (*Figure 1C and 1D*, Supplementary material online, *Video S1*). The peak mitral velocity and mean transmitral gradient increased to 2.5 m/s and 17 mmHg, respectively (*Figure 1B*). Additionally, the mitral valve area calculated by the continuity equation was 0.53 cm^2. The bioprosthetic valve thrombosis (BPVT) was suspected because it occurs mostly within 2 years after valve replacement,[1] VKA was initiated instead of surgery or thrombolysis because she was hemodynamically stable without thromboembolic event.[2,3] Three months later, follow-up TOE demonstrated normal leaflet thickness and opening (*Figure 2C and D*, Supplementary material online, *Video S2*). The peak mitral velocity and mean transmitral gradient markedly decreased to 1.4 m/s and 2 mmHg, respectively (*Figure 2B*). The mitral valve area calculated by continuity equation recovered to 1.18 cm^2. Bioprosthetic valve thrombosis is not uncommon, with an incidence of 0.74%.[1] Haemodynamic, haemostatic, and surface factors are considered as potential mechanisms of BPVT. She did not have laboratory findings of thrombotic disorders, maintained cardiac function and sinus rhythm throughout the clinical course. As we currently lack a precise understanding of the mechanism leading to BPVT, she was ultimately diagnosed with unprovoked BPVT. Careful follow-up is necessary even in the chronic phase after bioprosthetic valve replacement.

Supplementary material

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

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and

associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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