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Clinical valve thrombosis post-transcatheter aortic valve implantation with hypoattenuating leaflet thickening in computed tomography: anticoagulation is the answer

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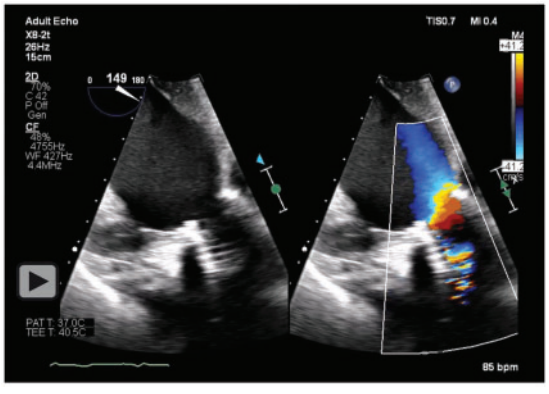
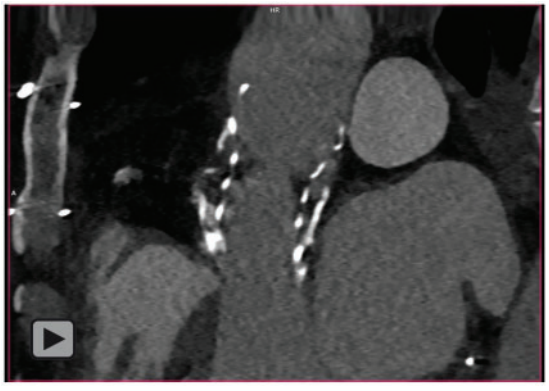
A 59-year-old woman presented to the emergency room in acute pulmonary oedema. Her past medical history included aortic valve replacement in 2005 for severe aortic insufficiency with a mechanical prosthesis, which was followed 2 years later by an aortic homograft replacement due to prosthetic endocarditis. In 2017, the patient developed homograft dysfunction with severe stenosis and worsening left ventricular (LV) systolic dysfunction, with a left ventricular ejection fraction (LVEF) of 29%. After heart team discussion, it was decided to perform transfemoral transcatheter aortic valve implantation (TAVI) - Corevalve Evolut PRO no. 29. Because LV dysfunction persisted after TAVI (LVEF 31%) and the patient had left bundle branch block (QRS 150 ms), a cardiac resynchronization therapy-defibrillator was implanted.

After admission, the patient responded well to medical therapy which included non-invasive ventilation for 24 h. Device interrogation denoted over 98% biventricular pacing, and no atrial fibrillation was detected. Transthoracic and transoesophageal echocardiography showed TAVI dysfunction with leaflet thickening and an increased mean transvalvular pressure gradient of 38 mmHg with no significant regurgitation (*Video 1*) and a LVEF of 26%. Leaflet lesions were further evaluated with cardiac computed tomography (CT), which showed bioprosthesis dysfunction with extensive hypoattenuating leaflet



Video 1 Transesophageal echocardiography denoting obstructive TAVI dysfunction with leaflet thickening.



Video 2 Cardiac CT showing extensive leaflet thickening suggestive of thrombosis.

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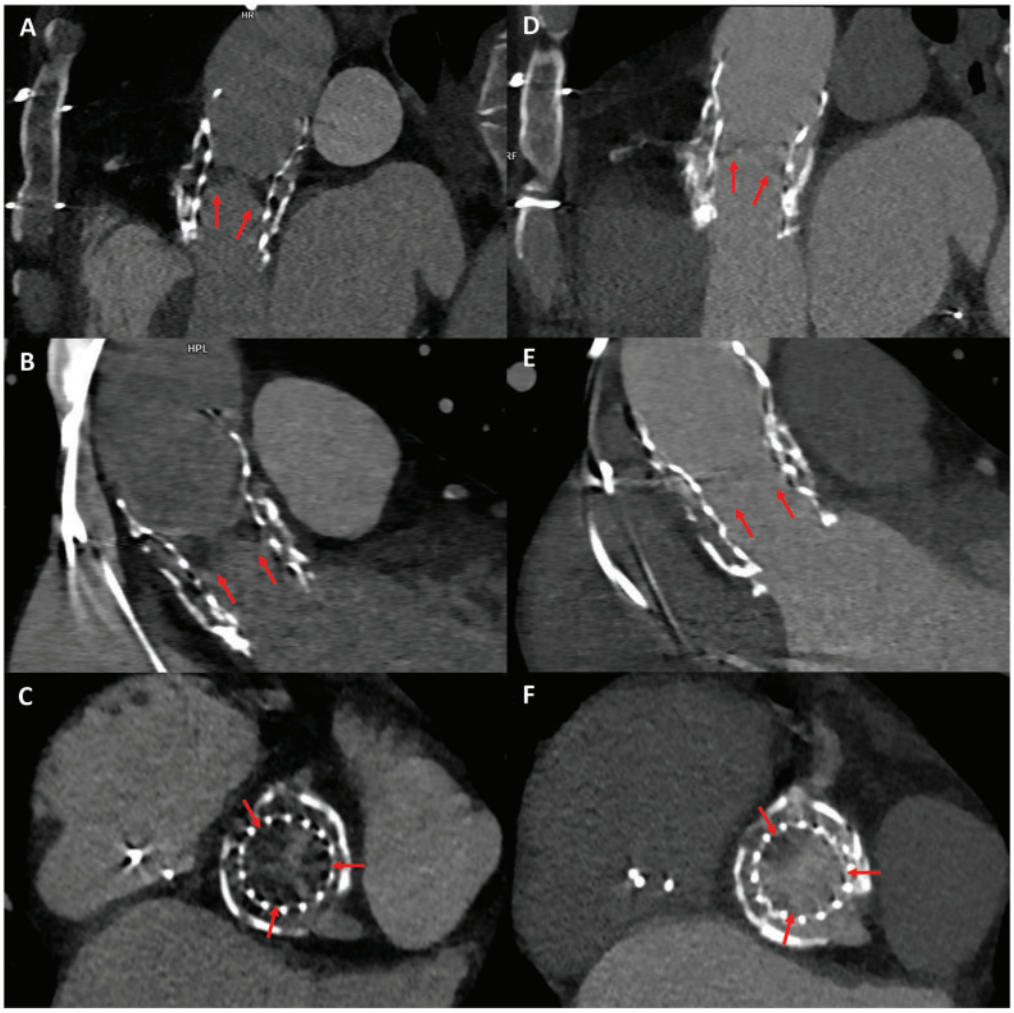

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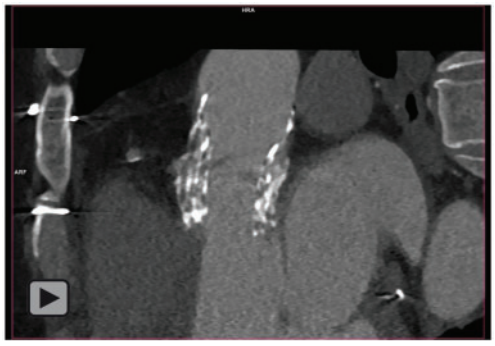

Video 3 Cardiac CT confirming significant resolution of thrombosis after 7 weeks of anticoagulation.

thickening compatible with thrombosis (*Panels A–C*, red arrow; *Video 2*), despite having been treated with aspirin since TAVI was performed. Warfarin was started, which was associated with clinical improvement and significant resolution of the leaflet lesions after 7 weeks of therapy (*Panels D–F*, red arrow; *Video 3*), and reduction of mean transprosthetic gradient to 11 mmHg.

Multiple mechanisms have been proposed for TAVI thrombosis, including (i) small valve size and under-expansion, (ii) aggressive post-dilation, (iii) geometric deformation of aortic valve stent, (iv) suboptimal antiplatelet/antithrombotic therapy, or (v) underlying thrombophilia, which was not investigated in this patient.

This case highlights the importance of cardiac CT in the initial assessment and follow-up of patients with symptomatic bioprosthetic valve dysfunction. Moreover, it demonstrates that they can have a significant clinical improvement with anticoagulation therapy and illustrates the challenge of optimizing the antithrombotic strategy in patients with no previous formal indication for anticoagulation.

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.