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CASE REPORT
Heart failure

Case report: a transcatheter aortic valve replacement failure secondary to COVID-19 infection

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Background Valve thrombosis is a well-documented cause of bioprosthetic valve failure. Case reports have been published of prosthetic valve thrombosis secondary to COVID-19 infection. This is the first case report of COVID-19 associated valve thrombosis in a patient with transcatheter aortic valve replacement (TAVR).

Case summary A 90-year-old female with atrial fibrillation on therapeutic apixaban and status-post TAVR presented with COVID-19 infection and was found to have severe bioprosthetic valvular regurgitation with features suggestive of valve thrombosis. She underwent valve-in-valve TAVR with resolution of valvular dysfunction.

Discussion This case report contributes to a growing body of evidence describing the occurrence of thrombotic complications in patients with valve replacement and COVID-19 infection. Increased vigilance and continued investigation are warranted to better characterize thrombotic risk and to inform optimal antithrombotic strategies during COVID-19 infection.

Keywords Case report • Prosthetic valve thrombosis • Aortic valve • Valve replacement • Acute heart failure • Thrombosis • Pulmonary oedema

ESC Curriculum 2.2 Echocardiography • 4.10 Prosthetic valves • 9.9 Cardiological consultations • 4.1 Aortic regurgitation • 4.2 Aortic stenosis

Learning points

- Recognize bioprosthetic aortic valve thrombosis as a possible diagnosis in a patient with history of transcatheter aortic valve replacement (TAVR) presenting with acute decompensated heart failure and COVID-19 infection.
- Recognize that there is no optimal anticoagulation regimen for post-TAVR patients when infected with COVID-19 and these patients may remain at increased risk for bioprosthetic valve thrombosis even when on anticoagulation.

Introduction

Valve thrombosis is well described in patients with bioprosthetic aortic valves, with one meta-analysis demonstrating an overall incidence of 8% (4% in patients taking direct oral anticoagulants) and resulting in new-onset aortic regurgitation in 50% of patients.[1,2]

Clinical presentation for valve thrombosis ranges from normal gradients on imaging to elevated gradients with symptoms of heart failure.[3]

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Though visualization of thrombotic mass on echocardiography is uncommon, associated characteristics include cusp thickness, reduced cusp mobility, and increased mean transvalvular gradient of >50% from baseline.[4]

Case reports are emerging describing acute mechanical and bioprosthetic valve thrombosis in the setting of COVID-19 infection. The first documented case of bioprosthetic aortic valve thrombosis was in a surgically implanted valve in a patient taking apixaban.[5] Two cases have been reported of mitral valve replacement failure in patients anticoagulated with low molecular weight heparin or vitamin K antagonists.[6,7]

We present the case of a 90-year-old female status-post transcatheter aortic valve replacement (TAVR) on therapeutic apixaban who presented with acute onset of bioprosthetic aortic valve regurgitation which we attribute to valve thrombosis in the setting of COVID-19 infection.

Timeline

9 April 2015	Transcatheter aortic valve replacement (TAVR)
8 December 2021	Routine transthoracic echocardiogram (TTE) with properly functioning bioprosthetic aortic valve
1 February 2022	Presentation to clinic with chest pain, referral to an emergency department
2 February 2022	Point-of-care ultrasound demonstrating holodiastolic colour flow across bioprosthetic aortic valve
3 February 2022	Formal TTE revealing severe aortic regurgitation
8 February 2022	Discharge from hospital
11 February 2022	Transoesophageal echocardiogram confirming severe aortic regurgitation
24 February 2022	Valve-in-valve TAVR
25 March 2022	Post-operative clinic visit, TTE revealing proper prosthetic valve placement and function

Case presentation

A 90-year-old female with atrial fibrillation treated with therapeutic apixaban and aortic stenosis status-post TAVR with a 29 mm self-expanding Medtronic core-valve placed 7 years prior presented to the clinic for the evaluation of acute onset chest pain and elevated blood pressure on home monitoring of 190/90 mmHg. The patient described the chest pain as substernal, severe, radiating posteriorly, and lasting for 45 min before resolving spontaneously.

The patient had a routine echocardiogram performed 2 months prior showing a properly functioning bioprosthetic aortic valve with normal haemodynamic performance (*Figure 1*). The Doppler measured peak transaortic gradient was 4 mmHg and the mean transaortic gradient was 2 mmHg.

Vital signs in the clinic (blood pressure of 110/41 mmHg, heart rate of 76 beats/min, oxygen saturation of 95% on room air) and physical examination were noted as unremarkable.

The patient was referred to the emergency department for assessment of the coronary anatomy and to rule out aortic dissection (*Figure 2*). During imaging, the patient developed acute respiratory failure with severe pruritis and urticaria and was admitted for inpatient management.

Evaluation in the emergency department was notable for the absence of hives, stridor, or other associated signs or symptoms of anaphylaxis.

Lab evaluation in the emergency department demonstrated a troponin-I value of 0.22 ng/mL (reference <0.04 ng/mL), B-type natriuretic peptide (BNP) of 514 pg/mL (reference <100 pg/mL; patient baseline of 22–71 pg/mL), and a positive COVID-19 polymerase chain reaction rapid test.

Chest x-ray was notable for diffuse interstitial pulmonary oedema. Electrocardiogram demonstrated sinus tachycardia with the left bundle branch block unchanged from prior. Computed tomographic (CT) angiography of the chest demonstrated trace bilateral pleural effusions, enlarged pulmonary arteries, and ruled out acute aortic dissection and pulmonary embolism.

The patient was hospitalized for acute decompensated heart failure and treated with non-invasive positive pressure ventilation, nitroglycerine infusion, and intravenous furosemide 40 mg twice daily. The patient's respiratory status improved by Day 1 of the hospital admission. The peak BNP was 1649 pg/mL.

Point-of-care ultrasound (POCUS) performed with a handheld device on hospital Day 2 was suggestive of a significant diastolic flow across the TAVR valve and directed further investigation for the diagnosis of prosthetic valve failure (*Figure 3*).

Transthoracic echocardiogram (TTE) on hospital Day 3 revealed severe aortic regurgitation with an eccentric and anteriorly directed jet and holodiastolic flow reversal in the descending thoracic aorta (*Figure 4*). A peak measured transaortic gradient was 9 mmHg and the mean transaortic gradient was 5 mmHg. At the time of the patient's hospitalization, the hospital system where the patient was admitted was experiencing staffing shortages and resource limitations related to an ongoing community COVID-19 surge. This served to delay access to the patient's echocardiogram. The hospital policy at the time was to delay routine transoesophageal echocardiography (TEE) until patients were designated as COVID-19 recovered.

During hospitalization, the patient was treated for COVID-19 with intravenous remdesivir 100 mg for 4 days and intravenous dexamethasone 6 mg for 7 days. Initially, the patient was managed for 2 days with continuous heparin infusion for presumptive treatment of acute coronary syndrome but was restarted on therapeutic apixaban 5 mg twice daily. The patient was discharged after 7 days and was transitioned from intravenous furosemide to oral torsemide 20 mg twice daily.

Three days after hospital discharge, TEE confirmed proper positioning of the bioprosthetic stent-valve with leaflet thickening, abnormal cusp mobility, restriction of the non-coronary cusp prosthetic leaflet, and an eccentric regurgitant jet consistent with severe aortic regurgitation (*Figure 5*). The acute TAVR valve regurgitation was subsequently treated with a valve-in-valve TAVR 17 days post-discharge.

The patient was seen in the clinic 30 days after valve-in-valve TAVR. The patient denied chest pain and dyspnoea. A TTE demonstrated bioprosthetic stent-valve in the aortic position with normal leaflet mobility and without paravalvular aortic regurgitation.

Discussion

In this case report, we describe a patient on therapeutic anticoagulation hospitalized for acute decompensated heart failure secondary to acute bioprosthetic aortic valve failure in the setting of COVID-19 infection.

Imaging revealed increased cusp thickness, reduced cusp mobility, and increased mean transvalvular gradient >50% from baseline, providing a 90% specificity for the diagnosis of valve thrombosis.[4] Additionally,

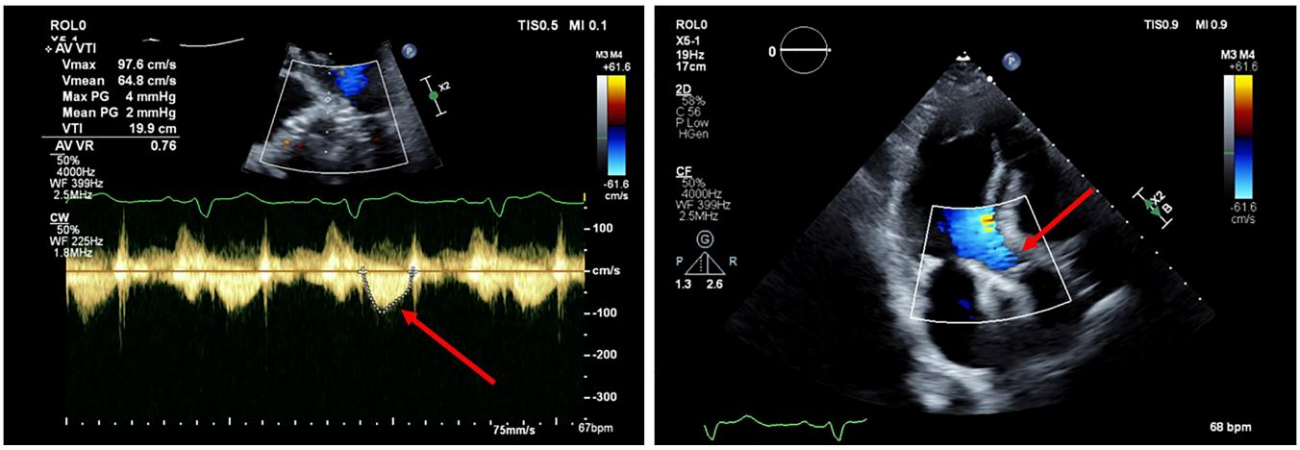

Figure 1 Transthoracic echocardiogram 2 months prior to initial presentation demonstrating well-seated transcatheter bioprosthesis with a normal spectral Doppler profile.

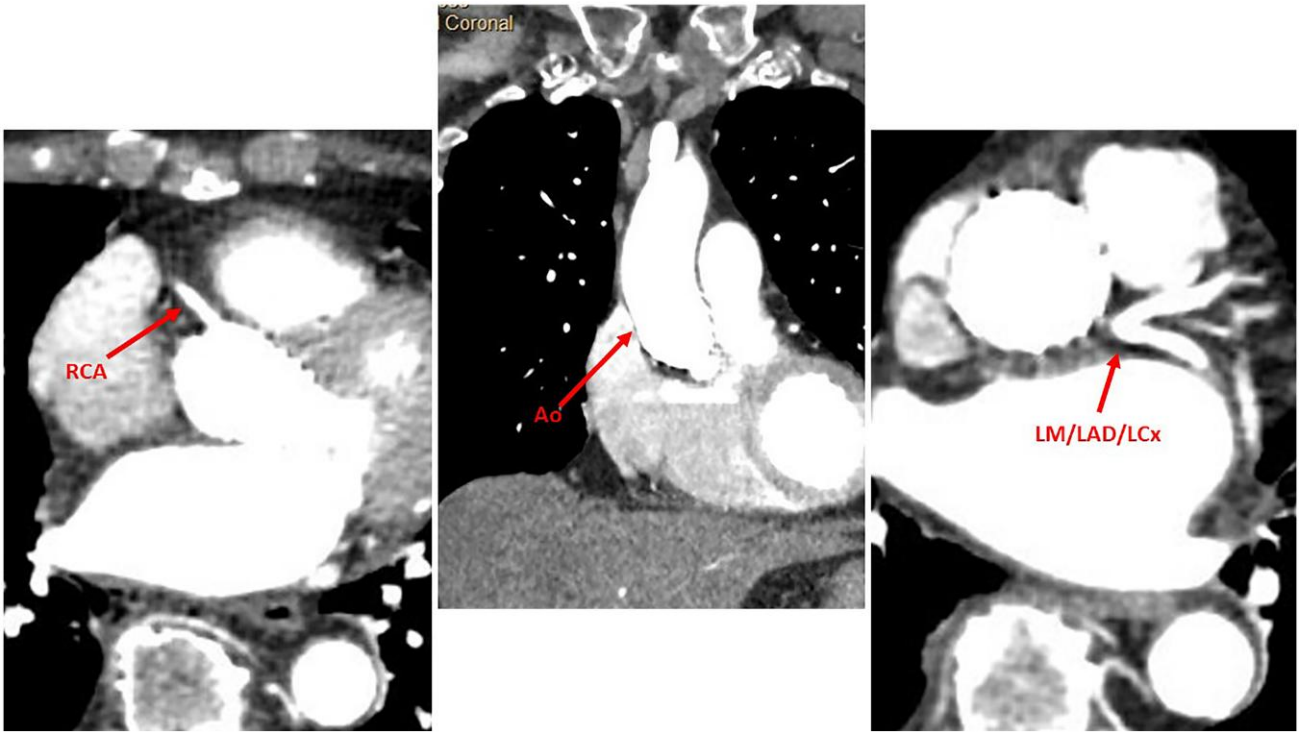

Figure 2 Computed tomography with contrast performed in the emergency department demonstrating the absence of aortic dissection and patent proximal coronary arteries. From left to right: right coronary artery, aorta (Ao), left main, LAD, and left circumflex coronary arteries (LM/LAD/LCx).

bacterial endocarditis was excluded as a diagnosis by an absence of TEE findings of valvular and perivalvular vegetations and negative blood cultures. The patient's echocardiogram performed 2 months prior to presentation demonstrated normal TAVR functioning suggesting structural failure as a less likely cause of the patient's clinical presentation. This represents the first published case of TAVR thrombosis secondary to COVID-19.

The precise mechanism for presumed COVID-19-related TAVR thrombosis and subsequent prosthesis failure remain unknown. It is plausible that increased thrombogenicity above the range of therapeutic efficacy with the targeted factor Xa inhibition of apixaban, or suboptimal anticoagulation related to disease–drug interaction, or medication non-adherence may be mechanisms for valve thrombosis and subsequent prosthetic valve failure.[8]

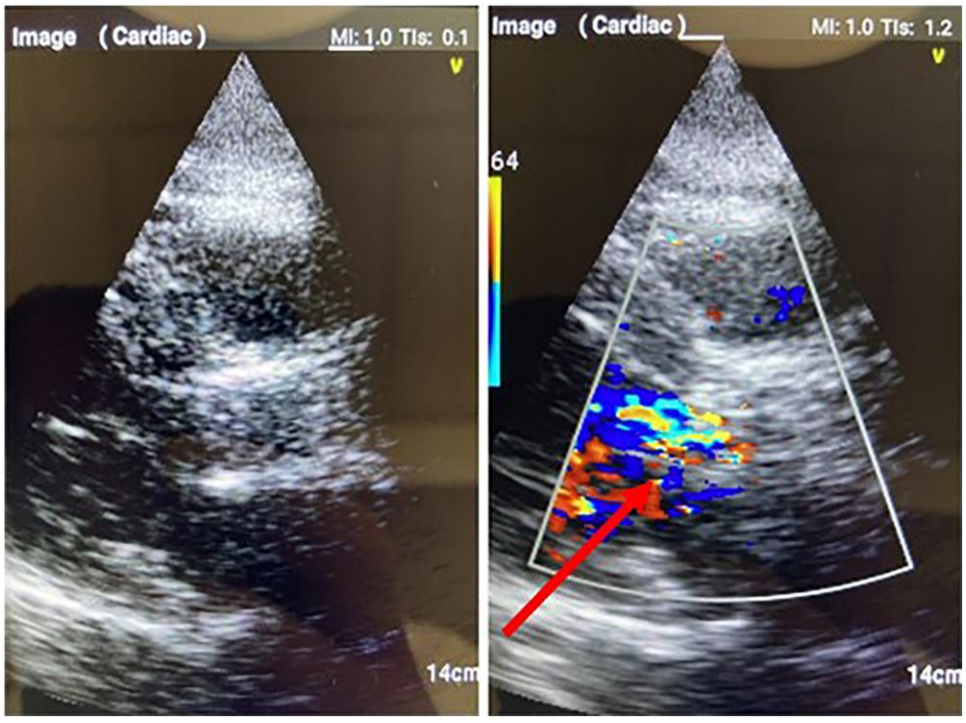

Figure 3 Point-of-care ultrasound images on hospital Day 2 demonstrating significant diastolic colourflow suggestive of severe aortic insufficiency: systole (left) and diastole (right) (arrow).

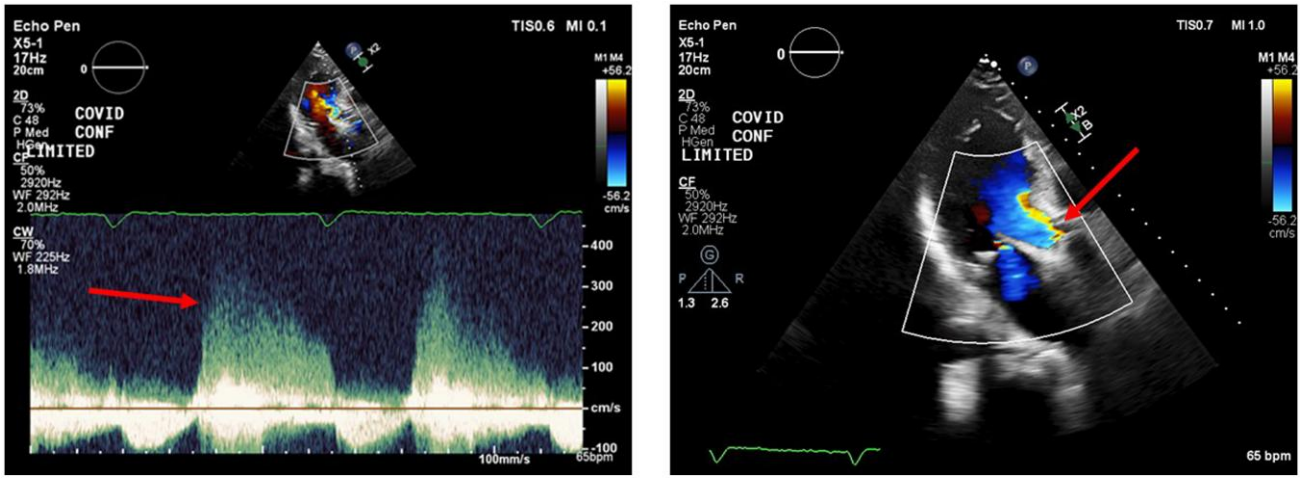

Figure 4 Transthoracic echocardiography demonstrating significant diastolic colourflow and dense spectral Doppler signal consistent with severe aortic insufficiency (arrows).

Thrombotic risk associated with COVID-19[9] was first recognized in the early stages of the COVID-19 pandemic. This case report contributes to a growing body of evidence describing the occurrence of thrombotic complications specifically in patients with valvular heart disease and COVID-19 infection regardless of anticoagulation. Increased vigilance in clinical management and continued investigation is warranted to better characterize thrombotic risk and to inform optimal antithrombotic strategies during COVID-19 infection. Finally, challenges associated with clinical assessment and obtaining formal inpatient echocardiography in actively infected COVID-19 patients[10] highlight the value of POCUS in these patients' clinical management.

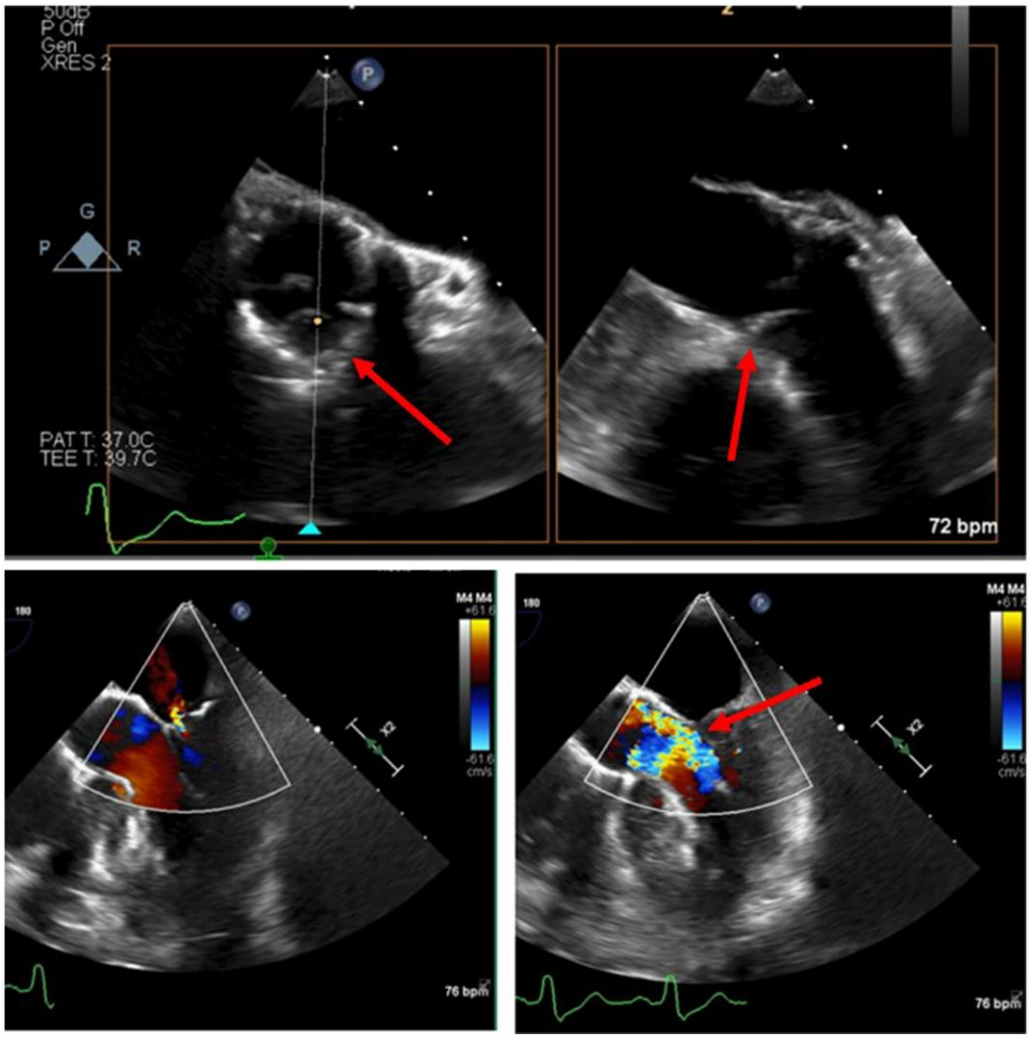

Figure 5 Transoesophageal echocardiography demonstrating leaflet thickening and eccentric diastolic colourflow jet consistent with severe aortic insufficiency (arrows).

Lead author biography

Dr P. Ryan Tacon is a third-year resident in Internal Medicine at the Cedars-Sinai Medical Center in Los Angeles, CA, USA. He is interested in a broad range of cardiovascular subspecialties and will be pursuing a career in cardiology after completing his internal medicine residency.

Supplementary material

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

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors 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.

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