

Acute myocardial infarction and stroke secondary to valve thrombosis following transcatheter aortic valve replacement—what can happen when antiplatelet agents are stopped

Mau Chu Lam¹, Gajen Kanaganayagam², Yousif Ahmad², Michael Fertleman³, Ben Ariff⁴

¹Department of Diagnostic Radiology, Princess Margaret Hospital, Kwai Chung, Hong Kong, China; ²Department of Cardiology, Imperial College Healthcare NHS Trust, London, UK; ³Care of the Elderly, St. Mary's Hospital, Paddington, London, UK; ⁴Imaging Department, Imperial College Healthcare NHS Trust, London, UK

Correspondence to: Dr. Mau Chu Lam, Department of Diagnostic Radiology, Princess Margaret Hospital, 2-10 Princess Margaret Hospital Road, Kwai Chung, Hong Kong, China. Email: lmc660@ha.org.hk.

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Introduction

An 84-year-old woman was hospitalised for a fall with subsequent agitation and vomiting. She had a past medical history of coronary artery bypass grafting performed 18 years ago. In the 8 months prior to her admission, she underwent transcatheter aortic valve replacement (TAVR) for severe aortic stenosis (AS). Post procedure, the patient had been instructed to take dual antiplatelet therapy (DAPT) for 6 months but had stopped these herself earlier than the recommendation.

On physical examination she was noted to have dysphasia, right upper limb weakness and hypotonia. An electrocardiography (ECG) showed dynamic inferior ST elevation and her serum troponin level was elevated.

A cardiac gated computed tomography (CT) of the aorta and coronary arteries was performed to exclude an aortic dissection and assess the coronary arteries. The CT demonstrated thrombus extending from the TAVR into the right coronary artery (*Figures 1,2*). A CT brain and magnetic resonance imaging (MRI) showed an acute infarct in the left parietal lobe. (*Figures 3,4*)

She was treated with antiplatelets and anticoagulation with intravenous (IV) heparin. Her ECG changes and neurological deficits resolved subsequently.

Discussion

AS is the commonest valvular heart disease leading to

surgery or catheter intervention in Europe (1). Surgical aortic valve replacement is recommended for symptomatic severe AS (2). Older persons often have multiple comorbidities, rendering them at high surgical risk. TAVRs, bioprosthetic valves inserted into the aortic valve position percutaneously are a treatment option for these patients (2).

The rate of thromboembolism among bioprosthetic aortic valve patients is 1.9% per patient-year despite anti-thrombotic therapy (2), underscoring the importance of the prevention of thromboembolism in TAVR patients. Current guidelines on post TAVR anti-thrombotic therapy, however, are not uniform and the strength of recommendations is not strong. The 2014 AHA/ACC guideline on valvular heart disease states that lifelong aspirin plus clopidogrel for 6 months “may be reasonable” (Class IIb, level of evidence C) (3). The 2017 AHA expert consensus document on TAVR recommends standard anti-thrombotic therapy should be clopidogrel 75 mg daily for 3–6 months with lifelong aspirin 75–100 mg daily (4). ESC guidelines recommend a combination of low-dose aspirin and a thienopyridine in the first 3–6 months after TAVR, followed by a lifelong single antiplatelet; although single anti-platelet therapy may be considered in those at high risk of bleeding (1). Position statements from the Canadian Cardiovascular Society (CCS) (DAPT for 1–3 months) (5) and ACCF/AATS/SCAI/STS (DAPT for 3–6 months) (6) give similar recommendations.

Whilst guideline bodies all recommend lifelong aspirin



Figure 1 Multiplanar reformatted CT image at the bioprosthetic valve level shows hypodense thrombus at the non-coronary cusp (black arrow) and thrombus extending into right coronary artery (white arrow). CT, computed tomography.

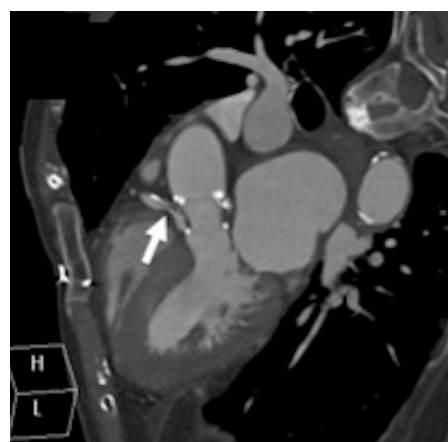


Figure 2 Multiplanar reformatted oblique CT image of the left ventricular outflow tract shows hypodense thrombus extending into right coronary artery (arrow). CT, computed tomography.

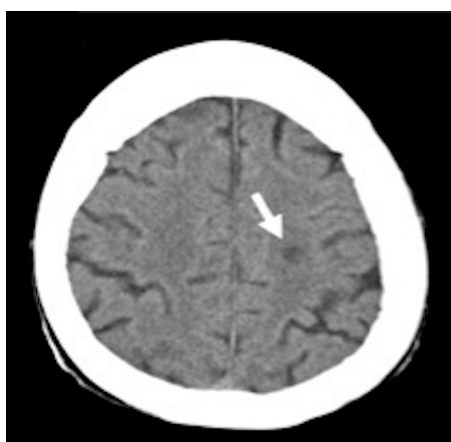


Figure 3 CT brain image shows a hypodense focus at left parietal lobe (arrow). CT, computed tomography.

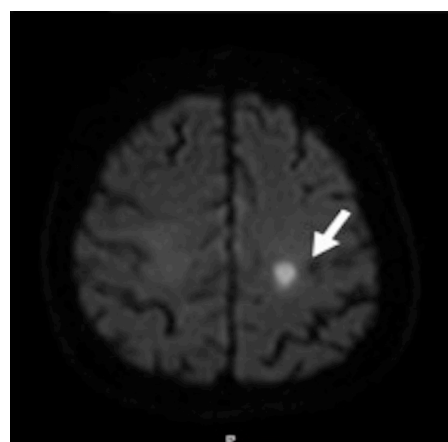


Figure 4 The corresponding lesion (arrow) shows hyperintensity on DWI image, suggesting restricted diffusion, and is compatible with an acute infarct. DWI, diffusion weighted imaging.

after TAVR, the recommended duration of DAPT varies, making it unclear as to what the optimal duration of DAPT is. The role of new oral anticoagulants post-TAVR is currently being explored. It is possible that these may be superior to antiplatelet therapy in the prevention of valve thrombosis.

There are currently no specific guidelines on perioperative antiplatelet therapy in TAVR patients, making the management of patients undergoing procedures difficult. Bridging heparin is only recommended in patients with mechanical prosthetic valves (1,3,7). We are unaware of

any centre that recommends the use of bridging heparin for TAVR patients who need suspension of antiplatelet therapy. A recent study has, however, shown that anticoagulation with novel oral anticoagulants and warfarin, but not DAPT was effective in the prevention and treatment of subclinical leaflet thrombosis, which itself was associated with increased rates of transient ischemic attacks (TIAs) and strokes (8). Currently, we recommend a balance of risks and reintroduction of the antiplatelet therapy as soon as possible.

Bioprosthetic valve thrombosis, as in our case, is uncommon. The first-line treatment of bioprosthetic valve

thrombosis is anticoagulation with a vitamin K antagonist and/or unfractionated heparin (1).

In summary, this case highlights the potential effects of stopping antiplatelets agents in TAVR and emphasises the importance of post TAVR antithrombotics. A balance must be made between risks of bleeding and thrombosis. Until antiplatelets can be restarted, there is no role for preoperative bridging heparin either via subcutaneous or intravenous routes.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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