CASE STUDY

Acute Myocardial Infarction due to Coronary Artery Embolism in a Patient with a Tissue Aortic Valve Replacement

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Abstract

Acute occlusive embolism to the coronary arteries resulting in acute myocardial infarction (AMI) is an uncommon occurrence. Although cases of patients with mechanical prosthetic heart valves resulting in this phenomenon have been reported in the setting of inadequate anticoagulation, reported cases resulting years after tissue aortic valve replacement (AVR) are rare. We report the case of a 50-year-old man who underwent a tissue AVR four years earlier and presented to the Emergency Department (ED) with an ST-segment elevation myocardial infarction. ED door-to-balloon time was delayed (at 115 minutes) because of pre-existing left bundle branch block on electrocardiogram. Emergent coronary angiography demonstrated complete occlusion of the left anterior descending coronary artery by a coronary embolus. The patient was successfully treated with percutaneous transluminal coronary angioplasty and aspiration thrombectomy, and subsequently underwent a transesophageal echocardiogram demonstrating thrombus on the tissue aortic valve prosthesis. This case demonstrates that coronary embolism resulting in AMI, while rare, can occur in patients years after tissue AVR surgery.

Case Report

A 50-year-old man who underwent a tissue aortic valve replacement (AVR) (23 mm Magna pericardial tissue heart valve) four years earlier for bicuspid aortic valve and aortic stenosis, presented to the Emergency Department (ED) complaining of sudden onset chest discomfort with radiation to his left arm. The discomfort began 20 minutes before arrival in the ED while he was lifting heavy weights. Medical history was notable for a coronary angiogram immediately prior to his AVR without significant coronary artery disease.
paroxysmal atrial fibrillation prior to surgery that was treated with a MAZE procedure at the time of AVR (without recurrence of atrial fibrillation), hypertension, and gout. His medications included losartan and allopurinol (but not aspirin for unclear reasons).

The patient was a well-developed, middle-aged man who appeared to be in no acute distress. Vital signs demonstrated an oral temperature of 98.6°F (37°C), pulse 60 beats/minute, blood pressure 115/72 mm Hg, respirations 20 breaths/minute, with an oxygen saturation of 100% on room air. Physical examination, including the heart and lungs, was entirely normal. A 12-lead electrocardiogram (ECG) was obtained in the ED (Figure 1, panel A), which demonstrated a normal sinus rhythm, left bundle branch block (LBBB) (present on previous ECG, figure 1, panel B) with new ST-segment concordance with the QRS waves and slightly more prominent ST-segment elevations in leads aVL and V5. The patient was initially felt not to meet criteria for an ST-segment elevation myocardial infarction (STEMI). Laboratory tests were significant for a troponin I of 0.45 ng/mL (normal range 0.00-0.09 ng/mL). Treatment for presumed acute coronary syndrome was initiated with oral aspirin, clopidogrel, and sublingual nitroglycerin. The patient was started on intravenous unfractionated heparin and eptifibatide and considered for expeditious cardiac catheterization. Approximately 90 minutes following ED arrival, the patient went into cardiac arrest with the monitor demonstrating ventricular fibrillation. Cardiopulmonary resuscitation immediately commenced, and he was successfully defibrillated to normal sinus rhythm with one biphasic shock of 200 joules with return of spontaneous circulation. The patient was given intravenous amiodarone 150 mg over 10 minutes, and was rushed to the cardiac catheterization laboratory (pulse 56 beats/minute, blood pressure 106/54 mm Hg at time of transfer). Angiography demonstrated a complete occlusion of the proximal left anterior descending artery, with angiographic features strongly suggestive of an embolic event (Figure 2, panel A), and akinesis of the mid to distal anterior wall and apex of the left ventricle. Successful percutaneous transluminal coronary angioplasty with balloon angioplasty and aspiration thrombectomy (6 French Export AP Catheter; Medtronic, Inc; Minneapolis, MN, USA) was undertaken with successful recanalization of the left anterior descending artery (Figure 2, panel B). Gross and microsopic pathological examination of the aspirated material by the pathologist confirmed the presence of thrombus. The patient was continued on unfractionated heparin, and warfarin therapy was initiated. A transesophageal echocardiogram (TEE) performed on hospital day #2 demonstrated 0.5 x 0.5 cm echodensities associated with the leaflets (but not the struts) of the tissue aortic valve prosthesis, consistent with thrombi (Figure 3, panel A, arrows). These thrombi were adjacent to the left main ostium; notably, no thrombi were seen in the left atrium. The patient was discharged neurologically intact on hospital day #5 with instructions to continue warfarin, enoxaparin (until warfarin therapeutic), aspirin, losartan and simvastatin as an outpatient. A repeat TEE performed six weeks later demonstrated near resolution of the aortic valve thrombi (Figure 3, panel B, arrows).

**Discussion**

In patients with significant aortic valve disease, replacement with prosthetic mechanical valves or with tissue valves (porcine or pericardial xenograft) remains the current standard treatment.1 In clinical practice, valve selection is based on several factors, including patient age, valve position (aortic vs mitral), patient comorbidities, the risks and benefits of anticoagulation, and

![Panel A](image1.png)

![Panel B](image2.png)

Figure 2. Coronary angiogram from a 50-year-old man demonstrating acute occlusion of the proximal left anterior descending artery by the coronary thrombus (panel A, arrow), and following successful percutaneous transluminal coronary angioplasty, suction thrombectomy and recanalization of the left anterior descending coronary artery (panel B, arrow).
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Mechanical prosthetic valves are thrombogenic, and patients who receive them require long-term anticoagulation with warfarin (with its associated increased risk of bleeding).1,2 Bioprosthetic (tissue) valves are felt to be less thrombogenic and usually do not require patients to continue chronic anticoagulation therapy; however, structural degeneration of these valves limits their durability.3,4

Coronary artery embolism is an uncommon cause of acute myocardial infarction (AMI).5 Several recent case reports cite prosthetic mechanical heart valves (in the aortic or mitral positions) in the setting of subtherapeutic anticoagulation as the cause of the coronary artery emboli and AMI.6,9 Other causes of coronary embolism resulting in AMI include infective endocarditis,10,11 atrial thrombus in the setting of atrial fibrillation,12 atrial myxomas,13 embolism of cotton pledgets following insertion of porcine valve bioprosthesis,14 calcific emboli from calcific aortic stenosis,15 and even emboli originating from biologic glues used in surgical repair of acute aortic dissection.16

Chronic tissue AVR thrombosis, while rare, has been reported.7-9 However, to our knowledge, such thrombotic involvement of a chronic tissue valve resulting in coronary embolism and AMI has yet to be reported in the literature. In our case the coronary embolism was found to be thrombus by angiographic and pathological examination, demonstrated to be related to a tissue AVR (by TEE), resolved after treatment with anticoagulation, and not associated with clinical or pathologic findings consistent with aortic valve endocarditis.

The prevalence of coronary artery embolism resulting in AMI is unknown, but is believed to be relatively uncommon.20 In one study, 55 out of 419 patients (13%) with autopsy-studied infarcts showed evidence of coronary artery embolism.21 It has been observed that most emboli involve the left coronary system, which could be because of the preferential flow into the artery related to coronary flow volume characteristics and aortic valve morphology.22 In patients with a presumed embolic coronary event, TEE has been recommended to identify any potential endocardial source of emboli.23

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Percutaneous transluminal coronary angioplasty and stenting have been successfully used to treat patients with AMI resulting from coronary artery embolism.24 Conversely, catheter aspiration embolectomy (as used in this case), has been successfully employed by several authors to aspirate thrombus from a native coronary artery.8,25-28 In our case, following successful aspiration thrombectomy of the clot, the patient was continued on unfractionated heparin (as a bridge to anticoagulation) and started on warfarin to decrease the risk of further thromboembolic events.

Our patient presented with STEMI in the setting of a known LBBB. The presence of a preexisting LBBB on the ECG may conceal the changes of AMI, and delay both its recognition and treatment.29,30 The Sgarbossa criteria are the most validated ECG criteria for diagnosing STEMI in the presence of LBBB.29,30 These ECG criteria include: 1) ST-segment elevation >1 mm concordant with QRS complexes; 2) ST-segment depression >1 mm in lead V1, V2, and 3) ST-segment elevation >5 mm discordant with QRS complex. The application of the Sgarbossa criteria has resulted in low sensitivities and high specificities for identifying patients with known LBBB and STEMI.29-31 The

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Figure 3. Transesophageal echocardiogram from 50-year-old man demonstrating echodensities associated with the aortic valve leaflets consistent with thrombus (panel A, arrows), compared with the same view following six weeks of warfarin anticoagulation, now demonstrating near resolution of the thrombi (panel B, arrows).
low sensitivity limits their utility in clinical practice, as diagnostic tests for AMI need to be highly sensitive to detect most cases.29

Our patient was not taking aspirin or any antipla telet agent at the time of his STEMI. The 2006 American Heart Association/American College of Cardiology Guidelines for Management of Patients with Valvular Heart Disease recommend long-term aspirin for all recipients of bioprosthetic heart valves (Class I, level of evidence C).32 The fact that our patient was not using an antiplatelet agent may have played a role in development of the AVR thrombus and subsequent coronary artery embolism.

We report the case of a man presenting with a STEMI resulting from acute coronary embolism in the setting of a tissue AVR performed four years earlier. The thrombus was successfully aspirated during coronary angiography, with the source of the coronary thrombus arising from the tissue AVR leaflets, as visualized by TEE. Following warfarin anticoagulation for six weeks, repeat TEE demonstrated near resolution of the AVR thrombi. Our case demonstrates that although rare, embolism from tissue prosthetic valves to the coronary artery can occur, resulting in life-threatening AMI. In such cases, emergent coronary angiography (with aspiration thrombectomy, percutaneous transluminal coronary angioplasty, and/or coronary artery stenting), TEE, and treatment with anticoagulation therapy are effective management strategies.

Disclosure Statement
The author(s) have no conflicts of interest to disclose.

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The Hot Organ

In the midst of [the lungs] is seated a hot organ, the heart, which is the origin of life and respiration. It imparts to the lungs the desire of drawing in cold air, for it raises a heat in them; but it is the heart which attracts. If, therefore, the heart suffer primarily, death is not far off.

— On Pneumonia, Aretaeus, the Cappadocian, 2nd Century AD Greek physician