

CASE PRESENTATION

A rare cause of acute ST-elevation myocardial infarction: a case of coronary embolism secondary to calcified bicuspid aortic valve

Anda Bularga¹, Andrew R Chapman¹, John Hung¹, Nicholas L. Mills¹, Marc R. Dweck¹

Abstract: Coronary embolism is an uncommon cause of acute myocardial infarction, which can have a similar clinical presentation to a plaque rupture event with acute onset of ischaemic symptoms, ST segment elevation on electrocardiogram (ECG) and significant elevation in cardiac troponin, requiring immediate intervention. We report the case of a middle-aged female with a background history of previous non-ST elevation myocardial infarction, bicuspid aortic valve with severe stenosis and metastatic breast cancer. The patient underwent emergency coronary angiography following acute onset central chest pain and evidence of anterior ST segment elevation on ambulance 12-lead ECG. The procedure revealed complete occlusion of the mid left anterior descending coronary artery with immediate flow restoration following embolus aspiration and subsequent normal appearance of the left anterior descending coronary artery. Gross examination of the aspirated specimen resembled a calcified hard lump, which was further confirmed on microscopic examination revealing calcified fibrous tissue most likely an embolus from the calcified bicuspid aortic valve. The patient had evidence of near transmural myocardial infarction in the distribution of the left anterior descending coronary on cardiac magnetic resonance imaging (MRI). She made full recovery and was discharged on short-term dual antiplatelet therapy followed by lifelong aspirin and further assessment for aortic stenosis management.

Keywords: myocardial infarction, coronary angiography, aortic valve stenosis.

Rezumat: Embolismul coronarian este o cauză puțin frecventă a infarctului miocardic acut. Acesta însă se poate manifesta cu un tablou clinic similar rupturii plăcilor aterosclerotice, cu instalarea acută a simptomelor de ischemie, supradenivelare de segment ST și creșterea semnificativă a troponinelor, necesitând o intervenție promptă. Prezentăm cazul unei femei de vârstă medie cu antecedente pozitive de infarct miocardic acut fără supradenivelare de segment ST, valvă aortică bicuspidă cu stenoză în stadiu avansat și cancer mamar metastatic. Pacienta a fost supusă unei angiografii coronariene de urgență în urma instalării acute a durerilor retrosternale și elevării segmentului ST (pe deviațiile anteroseptale) ale electrocardiografei. Investigația a demonstrat ocluzia completă a arterei coronariene stângi, ramura interventriculară și reluarea imediată a circulației coronariene în urma aspirării embolismului cu recăpătarea aspectului normal al arterei coronariene. Examinarea macroscopică a specimenului aspirat a arătat o masă dură cu aspect calcificat, iar examinarea microscopică ulterioară a demonstrat un țesut fibros calcificat, cel mai probabil cu origine valvulară (valva aortică bicuspidă calcificată). Rezonanța magnetică cardiacă a evidențiat un infarct miocardic (aproape) transmural în teritoriul de distribuție arterei coronariene stângi, ramura interventriculară. Pacienta s-a recuperat complet și a fost externată cu un tratament antiplachetar dual de scurtă durată, urmat de tratament continuu cu aspirină și reevaluarea tratamentului stenozei valvei aortice.

Cuvinte-cheie: infarct miocardic, angiografie coronariană, stenoză valvulară aortică.

INTRODUCTION

Coronary embolism is a rare, but important non-atherosclerotic cause of acute myocardial infarction and can present as an acute ST segment elevation myocardial infarction (STEMI) requiring emergency percutaneous coronary intervention for individualised diag-

nosis and management¹. The 4th universal definition of myocardial infarction classifies coronary embolism as a mechanism for myocardial injury related to acute myocardial ischaemia due to reduced myocardial perfusion². As such the pathophysiological mechanism of coronary embolism is classified under the umbre-

¹ BHF Centre for Cardiovascular Science, University of Edinburgh, United Kingdom

► Contact address:

Anda Bularga, MD, BHF Centre for Cardiovascular Science, Chancellors Building, Royal Infirmary of Edinburgh, Edinburgh EH16 4SA, United Kingdom.
E-mail: anda.bularga@ed.ac.uk

Ila term of type 2 myocardial infarction secondary to oxygen supply-demand imbalance^{2,3}.

The underlying causes of coronary embolism are multifactorial with atrial fibrillation as a common underlying predisposing clinical condition and a systemic hypercoagulable state as a potential risk factor⁴. Three types of coronary embolisms are described: direct embolisms originating from cardiac structures such as the left atrial appendage, aortic or mitral valves, cardiac neoplasms; paradoxical embolisms that arise from the venous circulation and pass through interatrial shunts and iatrogenic embolisms secondary to cardiac procedures⁴.

We present a rare case of a spontaneous calcific embolus from a native bicuspid aortic valve causing total occlusion of the left anterior descending coronary artery.

CASE REPORT

We report the case of a 53 years old female who presented with sudden onset central chest pain at rest described as „chest tightness” with no preceding symptoms of exertional angina. She had a background history of a previous non-ST elevation myocardial infarction, six years ago in the context of minor coronary plaque disease, which was managed medically with secondary prevention therapy; severe bicuspid aortic stenosis with preserved left ventricular systolic function for which she was receiving regular follow up and metastatic breast cancer on palliative treatment. The patient was a smoker, with no significant family history, her most recent cholesterol profile showed a total cholesterol of 3.5mmol/L with LDL choleste-

rol of 2.0mmol/L on regular statin therapy. An echocardiogram performed one month prior to this acute admission revealed progressive severe bicuspid aortic valve stenosis with a mean gradient of 72mmHg and peak gradient of 114mmHg across the valve and preserved left ventricular systolic function (Figure 1). The patient reported mild breathlessness on exertion and was referred for further investigations, multidisciplinary team discussion and consideration of valve intervention one month prior to this admission.

On initial assessment the patient was haemodynamically stable. Cardiovascular examination revealed first heart sound followed by an ejection systolic murmur radiating to the carotids with absent second heart sound. An ambulance trace showed evidence of ST segment elevation in the anteroseptal leads (VI-V4) with reciprocal ST segment depression in the lateral leads (Figure 2). Initial high-sensitivity cardiac troponin I was elevated at 1.319ng/L with a peak troponin of >50.000ng/L at 12 hours following symptom onset. She received emergency medical therapy with antiplatelet therapy and was taken directly to the cardiac catheterization laboratory for a primary percutaneous coronary intervention. The procedure was performed two hours after the initial symptom onset. The angiogram procedure showed an abrupt complete occlusion of the mid left anterior descending artery with thrombolysis in myocardial infarction (TIMI) grade zero flow in the distal segment, with no evidence of plaque disease proximal to the lesion or in the other coronary arteries (Figure 3).

The lesion was aspirated using an Export Advance catheter on first pass with immediate flow restorati-

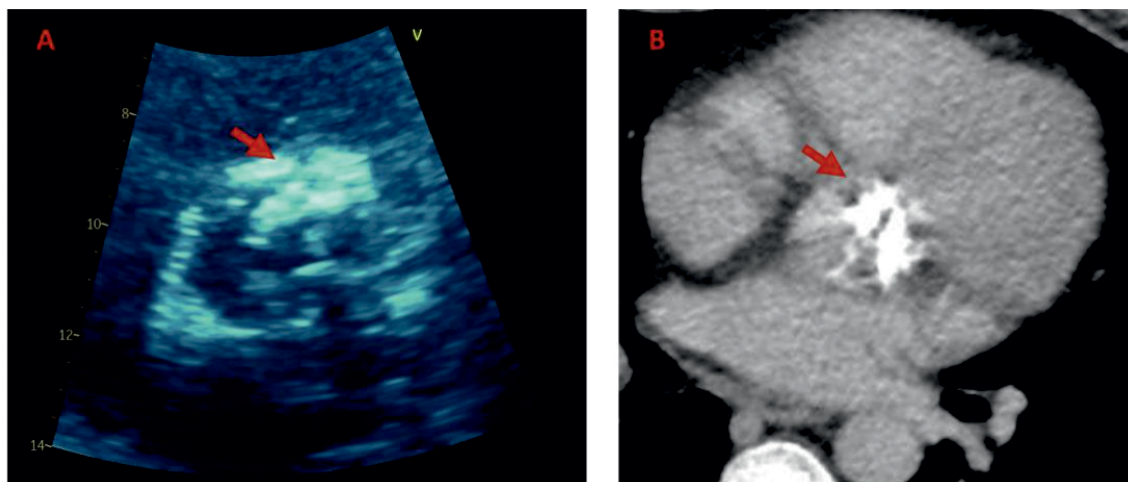


Figure 1. Echocardiogram image (A) and computed tomography image (B) showing severe calcific disease of the native bicuspid aortic valve. Scan were performed one month prior to the acute admission.

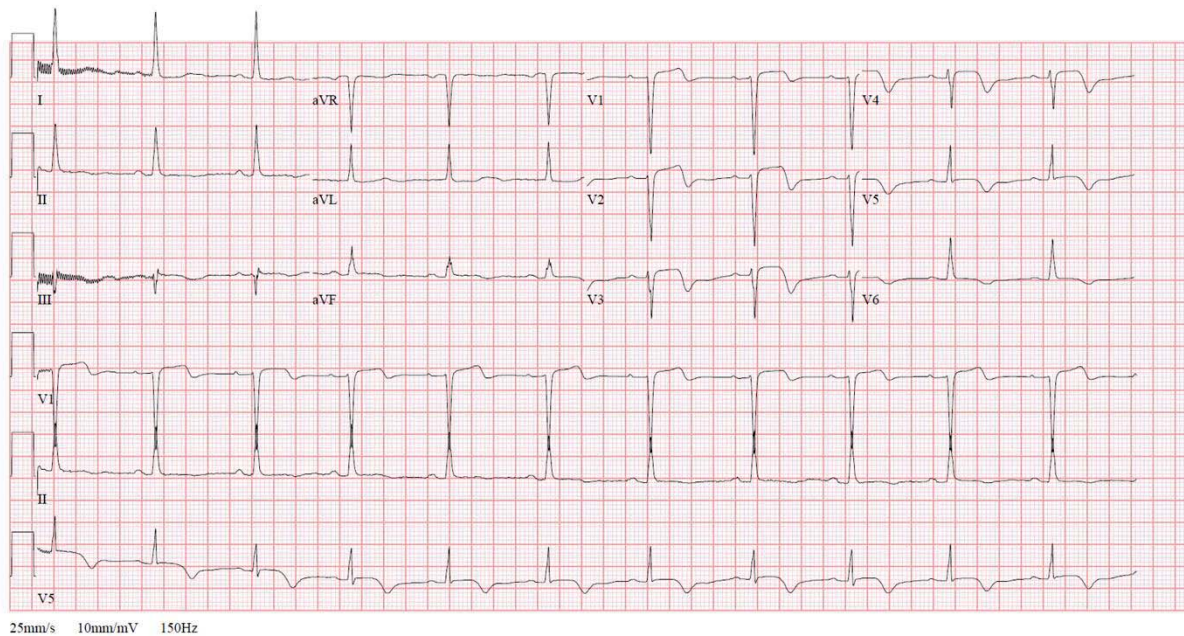


Figure 2. Electrocardiogram performed following coronary angiogram and embolus aspiration showing Q waves and residual ST segment elevation in the anteroseptal leads with inverted T waves in the lateral leads.

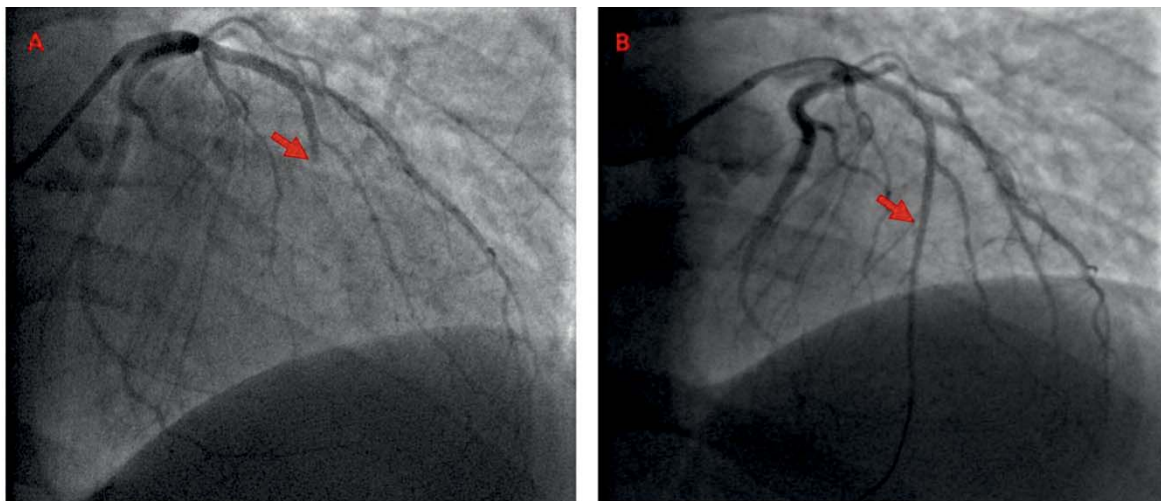


Figure 3. Coronary angiogram images showing abrupt occlusion of the left anterior coronary anterior (A) and flow restoration following embolus aspiration (B).

on in the distal vessel and normal appearances of the left anterior descending coronary artery (TIMI grade three flow) (Figure 3), therefore angioplasty with stent deployment was not required. On further inspection the aspirated tissue appeared as hard, ivory-white fibrous tissue resembling calcified deposits. The sample was sent for histopathological examination. Microscopic evaluation revealed granular proteinaceous material, which was focally calcified and surrounded by fibrin with occasional inflammatory cells and no other atypical features identified. The histopathology report did

not reveal evidence of organized thrombus, and, therefore, this was presumed to be embolic debris from the native calcified aortic valve.

Following the emergency percutaneous coronary intervention procedure, the patient was monitored on the coronary care unit for the next 48 hours and she made full recovery. She was managed with dual antiplatelet therapy for three months followed by aspirin monotherapy lifelong. The patient underwent cardiac magnetic resonance imaging (MRI) as part of a research study evaluating the mechanisms of myo-

cardial injury and role of coronary artery disease in patients with type 2 myocardial infarction (DEMAND-MI, NCT 03338504). The cardiac MRI scan, which was performed four days following the acute event, showed evidence of severe hypokinesis of the antero-septal wall extending towards the apex with near transmural late gadolinium enhancement in the same territory, consistent with myocardial infarction (Figure 4). There was evidence of mild to moderate left ventricular systolic dysfunction with ejection fraction of 47% and severe flow acceleration through a thickened aortic valve innkeeping with the background history of severe aortic stenosis.

The patient was discharge from hospital with plans for timely follow up and discussion of aortic valve stenosis management following multidisciplinary team consensus in view of her underlying comorbidities and metastatic cancer disease. Following thorough assessment and discussion with the Oncology team and in view of her progressive shortness of breath on follow up the patient underwent prosthetic aortic valve replacement six months following this acute admission.

DISCUSSION

Coronary embolism is an uncommon, but important and serious pathophysiological cause of acute myocardial infarction with potential adverse clinical consequences⁵. The reported prevalence varies according to angiographic or autopsy studies ranging between 4% and 13%^{6,7}. This pathological phenomenon is a recognised distinct mechanism for acute total occlusion

of a coronary vessel and can happen in the presence of absence of underlying coronary disease. It can have clinical presentations of varying severity including ST segment elevation myocardial infarction requiring emergency management and intervention similar to acute coronary syndrome due to atherosclerotic plaque rupture⁶. In clinical practice it is difficult to distinguish coronary embolism from atherothrombotic acute coronary syndrome, prior to diagnostic coronary angiography, hence the initial management and assessment of these patients is similar^{5,6}. Intracoronary imaging by means of optical coherence tomography (OCT) or intravascular ultrasound may be considered in selected cases to help evaluate for potential evidence of plaque rupture or erosion versus an embolic event^{4,5}.

The current universal definition of myocardial infarction classifies coronary embolism as a mechanism of reduced myocardial perfusion due to coronary flow disruption in the absence of plaque rupture or erosion (type 1 myocardial infarction)². As such coronary embolism is defined as a distinct mechanism of decreased oxygen supply under the umbrella term of type 2 myocardial infarction². Type 2 myocardial infarction is a heterogeneous condition with poor clinical outcomes encompassing a range of cardiac and systemic pathophysiological processes⁸. Arguably, coronary embolism has many similarities to a type 1 myocardial infarction, both from the clinical presentation, diagnostic approach and management perspectives. This case illustrates some of these similarities – the

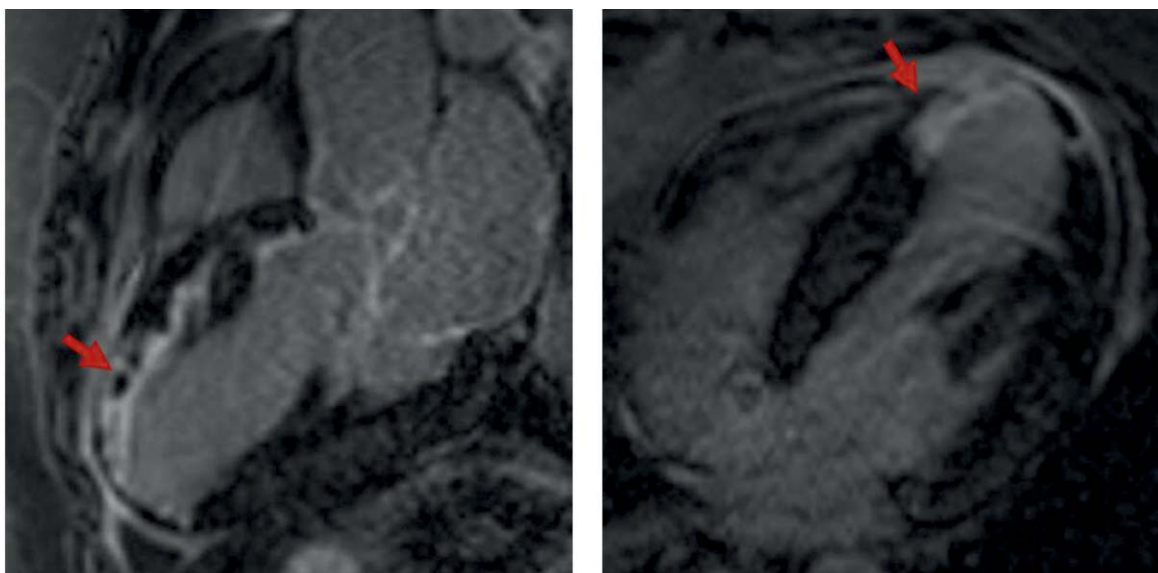


Figure 4. Cardiac MRI long-axis views showing near transmural late gadolinium enhancement in the mid anteroseptum extending to the apex.

patient presented with an acute anteroseptal ST-elevation myocardial infarction. In view of her background history of a previous acute coronary syndrome in the context of mild plaque disease and cardiovascular risk factors there was a strong suspicion of an atherothrombotic event. She received antiplatelet therapy and underwent emergency percutaneous coronary intervention, which revealed total occlusion of the left anterior coronary artery with no significant coronary disease in the proximal vessel or other coronary arteries. Thrombus aspiration restored normal flow and macroscopic evaluation of the specimen showed distinct features in keeping with a calcified mass. The patient did not undergo further intracoronary imaging following clot aspiration; however, this may play a role when the suspicion of a plaque rupture event is high.

Determining the underlying cause of the coronary embolism is in itself an important step, which can have implications on long-term management particularly when considering regular antithrombotic therapy, follow up and prevention of recurrent events⁴. Whilst a range of embolisation sources and associated predisposing clinical conditions are recognised, some of these are more common than others. In a number of recently published cohort studies atrial fibrillation was reported as the most common cause of coronary embolism with a period of monitoring being potentially warranted in the absence of further aetiological evidence^{5,6}. Clinical history and structural cardiac imaging are important when considering direct, paradoxical emboli or iatrogenic causes.

In this case the underlying aetiology was strongly suspected following coronary intervention which revealed abrupt occlusion of the left anterior descending coronary artery with normal appearance of the artery following embolus aspiration without significant residual coronary disease and evidence of collateral circulation. Furthermore, gross examination of the aspirated embolus, which resembled calcified material was in keeping with the known calcific bicuspid aortic valve disease with severe stenosis. In considering alternative embolic sources, patient's underlying history of metastatic breast cancer increases predisposition to a procoagulant state and indeed would be a plausible alternative cause. However, the histopathological evaluation of the aspirated embolus confirmed the suspected aetiology showing evidence of calcified fibrous material and absence of organised thrombus formation.

The risk of calcific embolisation from aortic stenosis is recognised and can affect cerebral, renal, coronary and retinal circulation⁹ and thought to be more common with bicuspid valves and severe stenotic disease due to turbulent flow through the diseased valve and calcium debris rupture^{10,11}. In this case the coronary embolus caused clinically significant anteroseptal myocardial infarction as evidenced by extensive late gadolinium enhancement in the affected coronary territory. Although rare, calcified embolus from valve disease should be a differential cause of coronary embolus and this case highlights the importance of thorough evaluation of the embolism source to guide medical management and further evaluation of the severity of valvular heart disease. Whilst septic emboli in the context of infective endocarditis is an indication for surgical valve replacement¹², emboli from calcific, degenerative native valve disease are not a recognised indication for valve intervention, however careful assessment and consideration should be given in individual cases.

CONCLUSION

This case shows a rare cause of an anteroseptal ST-elevation myocardial infarction due to total occlusion of the left anterior coronary artery caused by calcified material from severe calcific disease of a native bicuspid aortic valve. Whilst many pathophysiological causes of coronary embolisms can be identified, thorough, individualised assessment of these patients is necessary to determine the underlying mechanism of the coronary embolism for appropriate immediate and long-term management.

Conflict of interest: none declared.

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