

CASE REPORT

An uncommon complication of atrial fibrillation

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Abstract

Coronary embolism is a well-recognized cause of myocardial infarction. It is often under diagnosed and cardiologists need to be vigilant for this diagnosis. A 77-year-old man presented with chest pain with an ECG showing a new diagnosis of atrial fibrillation. Owing to ongoing chest pain coronary angiography was performed and revealed an acute occlusion of the left circumflex artery with coronary blood flow restored following aspiration of a large red thrombus. Following this the coronary vessel looked smooth with no residual coronary lesions requiring angioplasty or plaque rupture to justify the thrombosis. The clinical picture and angiographic data suggested the coronary embolus was secondary to the newly diagnosed atrial fibrillation.

INTRODUCTION

Coronary embolism is one cause of acute myocardial infarction with angiographically normal coronary arteries [1]. The thrombus may be seen during angiography and can be aspirated with restoration of coronary flow. In other cases, it may not be seen at all but presumed to be the cause depending on individual clinical characteristics. Atrial fibrillation is increasingly being recognized as cause of coronary embolism. We present a case of a patient with a new diagnosis of atrial fibrillation and myocardial infarction presumably due to coronary embolism successfully treated with thrombus aspiration.

CASE REPORT

A 77-year-old retired army service man with past history of hypercholesterolaemia and previous tuberculosis presented to the Emergency Department having briefly lost consciousness. On recovering, he was aware of a chest discomfort with radiation to the back and neck. He was an ex-smoker. Initial assessment revealed an irregular pulse with a rate between 30 and 50 bpm and a blood pressure of 103/56 mmHg. Cardiac examination was unremarkable with no murmurs heard and the jugular venous pressure was not raised. Auscultation of the chest was also

unremarkable. He had different blood pressures between left (103/56 mmHg) and right arms (76/59 mmHg). His ECG revealed atrial fibrillation, a new finding, partial right bundle branch block and left axis deviation but no other ischaemic changes. A CT aortogram excluded aortic dissection and pulmonary embolus. Blood tests revealed an initial troponin T of 96 ng/l (normal < 14), which subsequently rose to 1088 ng/l. He was treated as an acute coronary syndrome. Echocardiography revealed mild left ventricular impairment with lateral wall hypokinesia and no evidence of intra-cardiac thrombus. Whilst on the ward he complained of intermittent chest discomfort and we proceeded to coronary angiography 48 h after admission, which revealed an unobstructed left main stem and minor irregularities within the left anterior descending artery. The right coronary artery was co-dominant and unobstructed. However, the left circumflex (LCx) artery was co-dominant and there was a large area of solid thrombus covering a 2 cm stretch of the artery (Video 1). Given the history of atrial fibrillation we felt that this most likely represented an embolic occlusion. A guide wire was placed across the occlusion and the thrombus was aspirated successfully with an aspiration catheter. A significant amount of red thrombus was obtained. Subsequent angiographic images of the LCx revealed a completely smooth normal looking artery without any area of

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Video 1: Coronary angiography showing a large area of solid thrombus in the LCx artery.



Video 2: Coronary angiography post thrombus aspiration showing smooth LCx artery with no area of stenosis.

stenosis or irregularity (Video 2). The patient remained well following his procedure and was discharged on apixaban 5 mg twice daily, atorvastatin 80 mg daily, bisoprolol 2.5 mg daily, clopidogrel 75 mg daily and ramipril 1.25 mg daily.

There was no evidence of an atrial septal defect or suspicion of a patent foramen ovale on the trans-thoracic echocardiogram thus making the diagnosis of an embolic coronary embolus secondary to atrial fibrillation and possibly thrombus in the left atrial appendage the most likely cause of his myocardial infarction.

DISCUSSION

Acute myocardial infarction secondary to coronary embolism is a rare but well-recognized phenomenon, now classified as a Type 2 MI (i.e. myocardial infarction due to ischaemic imbalance) in the most recent third universal definition of myocardial infarction [2]. Although, thrombus seen during angiography despite an obvious plaque lesion can still be due to the classic plaque rupture of atherosclerosis (i.e. concealed atherosclerosis with outward plaque expansion) [1], coronary embolism has been found to be responsible for acute myocardial infarction between 10 and 13% of cases in autopsy studies [1]. Atrial fibrillation as a cause of acute coronary embolism has been shown in one early autopsy study to be responsible for 24% of coronary embolic cases [3]. Other causes of coronary embolism include atrial myxoma, mural thrombus, infective endocarditis and calcium dislodged from calcified valves during cardiac procedures [1]. Coronary embolism has also been described during left atrial ablation for atrial fibrillation that was successfully treated by thrombectomy [4]. Even more rare, paradoxical coronary embolism can also cause myocardial infarctions via a patent foramen ovale with venous thromboembolism constituting most of the reported cases [5], though tumour [6], air [1, 5] and foreign material [7] have been reported.

Often, though not demonstrated in our case, one clue of coronary embolism being the cause is the presence of multiple territory myocardial infarcts demonstrated either during angiography or on imaging such as cardiac MRI [8, 9]. It seems, as in our case, that in cases of coronary embolism, aspiration with a

thrombus aspiration catheter is enough to improve coronary blood flow without the need to proceed to any form of angioplasty and is a successful way to treat these patients [10].

Subsequent pharmacotherapy following an embolic MI, however, can be more challenging. The European Society of Cardiology (ESC) in their most recent guidelines, though recognizing coronary embolization from atrial fibrillation as a rare cause of MI, it offers no guidance as to subsequent drug treatment [11]. The ESC advises a triple therapy of aspirin, clopidogrel and warfarin for 3–6 months followed by warfarin and clopidogrel for 12 months [12] in patients with AF and acute MI undergoing coronary intervention, as there is clearly a benefit from being both on antiplatelet agents as well as an anticoagulant agent.

However, in cases of embolic MI, one could hypothesize that there is no benefit from being on an antiplatelet agent. In the absence of guidelines or indeed clinical trials, as most of the literature on embolic MIs is based on case reports and case series, the subsequent treatment is left to the discretion of the treating physician. Some clinicians opt for warfarin only [10, 13] whereas others give warfarin and one antiplatelet agent [14, 15]. In our case, we opted for the latter.

In conclusion, atrial fibrillation can be a cause of coronary embolism leading to myocardial infarction and cardiologists need to be vigilant to make the diagnosis as immediate interventional treatment may differ and the patient will require different pharmacological therapy post-infarct. Clues to the diagnosis include the clinical history, ECG, the presence of multiple simultaneous territory infarcts and successful treatment with thromboaspiration only with angiographically normal coronary arteries.

CONFLICT OF INTEREST STATEMENT

None declared.

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