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A Suspected Case of Acute Embolic Myocardial Infarction Following Direct-Current Cardioversion of Atrial Fibrillation

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 66

Final Diagnosis: A suspected case of acute embolic myocardial infarction following direct current cardioversion of atrial fibrillation

Symptoms: Exertional shortness of breath

Medication: —

Clinical Procedure: Direct current cardioversion

Specialty: Cardiology

Objective: Unusual clinical course

Background: Non-atherosclerotic causes of ST-segment elevation myocardial infarction (STEMI) are uncommon, and there are few case reports of acute myocardial infarction secondary to coronary artery embolism.

Case Report: A 66-year-old man presented with shortness of breath and leg swelling. Diagnoses of congestive heart failure and atrial fibrillation were made. He was electrically cardioverted to normal sinus rhythm. Coronary angiogram was performed to rule out ischemic etiology of new-onset systolic heart failure, and anticoagulation therapy was interrupted for cardiac catheterization. His coronary angiogram showed 60% angiographic but hemodynamically insignificant stenosis by fractional flow reserve in the left anterior descending artery. The following day, the patient developed chest pain and ST-segment elevation in the anterolateral leads of the ECG. An emergent coronary angiogram showed thrombotic occlusion of the left anterior descending artery distal to the mid-left anterior descending artery lesion that was found on the initial angiogram. Successful thrombus aspiration was performed, and the patient was discharged to home on oral anticoagulation therapy with rivaroxaban. Most likely, the cause of thrombotic occlusion of the left anterior descending artery was an atrial fibrillation-related thromboembolic phenomenon due to interruption of anticoagulation therapy soon after direct-current cardioversion.

Conclusions: Subtherapeutic anticoagulation therapy soon after direct-current cardioversion of atrial fibrillation can lead to potentially fatal coronary artery embolism and acute myocardial infarction.

MeSH Keywords: Atrial Fibrillation • Electric Countershock • Myocardial Infarction

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/911469

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Background

Non-atherosclerotic causes of ST-segment elevation myocardial infarction (STEMI) are uncommon, and there are few case reports of acute myocardial infarction secondary to coronary artery embolism [1–9]. Atrial fibrillation, left ventricular thrombus, septic emboli from infective endocarditis, tumors, and paradoxical embolism due to patent foramen ovale can be the source of emboli to coronary arteries [8]. This case highlights a rare but potentially fatal complication of subtherapeutic anticoagulation therapy soon after direct-current cardioversion of atrial fibrillation. Our patient developed acute embolic occlusion of the left anterior descending artery following cardioversion, most likely due to interruption of anticoagulation therapy.

Case Report

A 66-year-old morbidly obese white man with a past medical history of hypertension and hyperlipidemia presented with gradually worsening exertional shortness of breath and leg swelling over the last week. The patient had gained 30 lb over the past month. He was afebrile with an irregularly irregular pulse rate of 152 beats per minute, blood pressure of 123/56 mmHg, respiratory rate of 16 breaths per minute, and oxygen saturation 95% on room air. The cardiovascular examination was remarkable for bilateral basilar rales and lower-extremities edema. An initial 12-lead ECG showed atrial fibrillation with rapid ventricular response (Figure 1A). B-type natriuretic peptide (BNP) level was elevated to 771 pg/ml (reference range 0–100 pg/ml). A transesophageal echocardiogram (TEE) showed moderately reduced biventricular systolic function. The left atrium appeared to be enlarged, with no thrombus in the left atrial appendage or left atrium itself. Mild atherosclerotic plaque in the descending aorta was seen. Successful direct-current cardioversion to normal sinus rhythm (Figure 1B) was performed, and anticoagulation with weight-based unfractionated intravenous heparin infusion and antiarrhythmic therapy with amiodarone were initiated.

The following day, a coronary angiogram was performed to exclude significant coronary artery disease as the etiology of new-onset systolic heart failure. The coronary angiogram showed a 60% stenosis, but it was hemodynamically insignificant by fractional flow reserve (FFR 0.88) study in the middle segment of the left anterior descending (LAD) artery (Figure 2A, Video 1). To achieve adequate hemostasis at the catheterization access site, intravenous anticoagulation therapy was interrupted for approximately 10 hours following the cardiac catheterization.

The next day, the patient developed acute substernal chest pain radiating to the left arm, and ST-segment elevation in the anterolateral leads was seen on ECG (Figure 1C). The initial troponin-I level was 0.06 ng/ml (reference range 0.00–0.04), CKMB 3.5 ng/mL (reference range 0.0–5.0 ng/mL), and CK 205 IU/L (reference range 30–223 IU/L). A decision was made to perform an emergent coronary angiogram, which showed a stable, previously seen stenosis in the middle segment of the LAD; however, acute thrombotic occlusion of the distal LAD was now present (Figure 2B, Video 2). Following thrombus aspiration and intracoronary nitroglycerin administration, complete restoration of the flow in the LAD was achieved (Figure 2C, Video 3). No other new angiographic lesions were found. The remaining hospital course was uneventful, and the patient was discharged to home on aspirin, clopidogrel, and oral anticoagulation therapy with rivaroxaban.

Discussion

Atrial fibrillation, left ventricular thrombus, septic emboli from infective endocarditis, tumors, and paradoxical embolism due to patent foramen ovale can be the source of emboli to coronary arteries [8]. On a postmortem examination of 419 patients, 55 patients (13%) had coronary artery embolic infarcts [10]. In a recent retrospective study of 1776 patients with *de novo* acute myocardial infarction, the prevalence of coronary artery embolism was 2.9% [11]. Furthermore, no predilection to right or left coronary arteries was observed. This finding was in contrast to a previous study which showed that the left coronary system was more prone to emboli, possibly due to the preferential flow [10].

The prevalence of thromboembolic events after direct-current cardioversion of atrial fibrillation is 2.0%, and is higher in diabetic and heart failure patients (9.8%) [12,13]. Most of the embolic events occur within 3 days after cardioversion [14]. The duration of atrial fibrillation prior to the direct-current cardioversion is an important risk factor for early formation of left atrial appendage thrombus in the case of subtherapeutic anticoagulation. In the cases of atrial fibrillation of more than 48 hours' duration following direct-current cardioversion, there is electromechanical dissociation in the left atrium due to tachycardia-mediated stunning of the left atrium [15]; this causes stasis of the blood, leading to thrombogenesis and embolization [16]. In our patient, anticoagulation therapy interruption following the cardiac catheterization is the most likely explanation of the coronary artery embolism [14]. The sensitivity and specificity of TEE reaches 99% for exclusion of left atrial appendage thrombus [17], a frequent source of emboli; yet it is possible that in rare cases, micro-emboli remain undetected. Another possible pathophysiologic mechanism of myocardial infarction in our patient is the atrial fibrillation-induced inflammation. Many inflammatory markers have been used to predict cardiovascular events, and they play an

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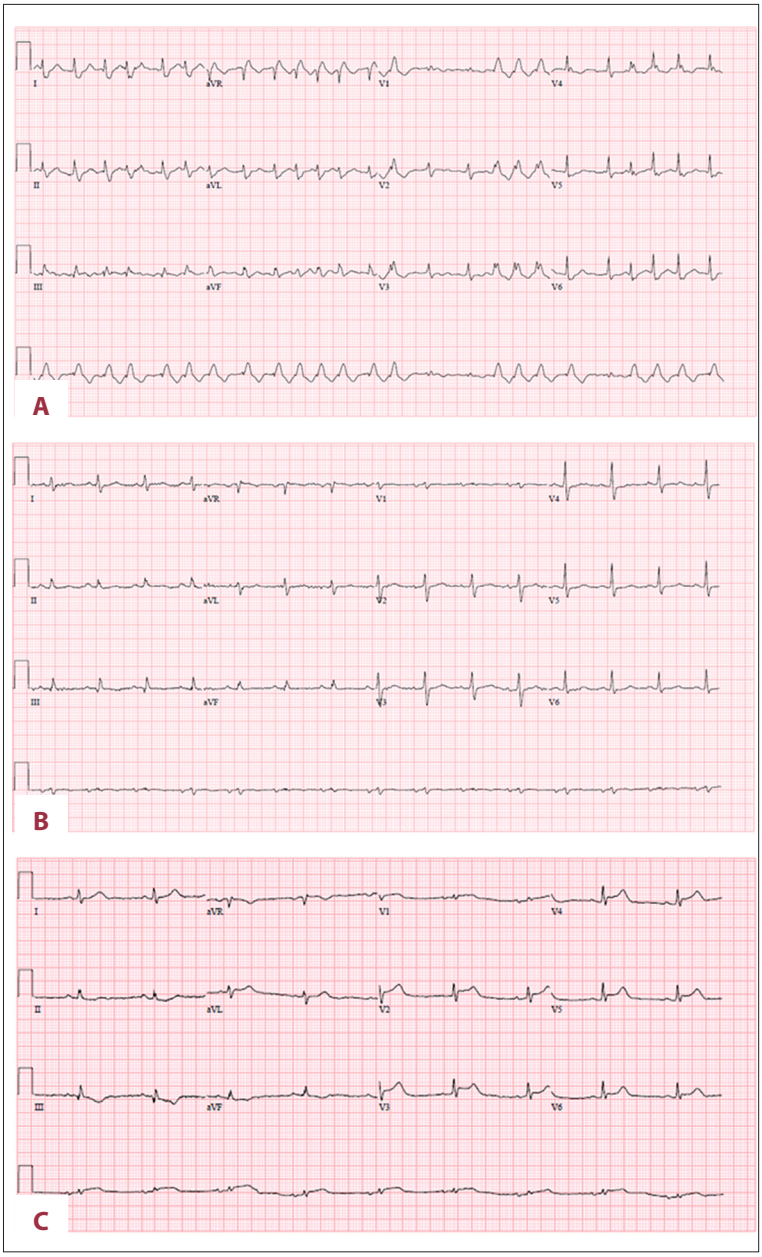

Figure 1. 12-lead ECG shows (**A**) atrial fibrillation with rapid ventricular response, (**B**) normal sinus rhythm (post cardioversion), and (**C**) ST-segment elevation in the anterolateral leads.

important role in the pathophysiology of AF [18]. For example, plasma stromal cell-derived factor-1 has been found to be increased in patients with atrial fibrillation and coronary artery disease and is associated with recruitment of the inflammatory cells and adverse remodeling [19].

The key to successful management of coronary artery embolism-related acute myocardial infarction is prompt thrombus aspiration [9]. The use of intracoronary thrombolysis with r-TPA or urokinase in addition to balloon angioplasty has also been described [1,6]. Optical coherence tomography (OCT) or intravascular ultrasound (IVUS) can be performed after catheterization to rule out possible underlying eccentric plaque rupture or erosion [20]. Histopathological examination shows that the left atrial thrombus contains fibrin, platelets, and red blood cells [3]. Long-term treatment with oral anticoagulation and antiplatelet therapy are recommended in these patients [5]. A coronary artery embolism recurrence rate as high as 8.7% has been reported during the 5-year follow-up. Moreover, a higher 5-year risk of all-cause and cardiovascular mortality compared to other subgroups of patients with acute myocardial infarction has been reported [11].

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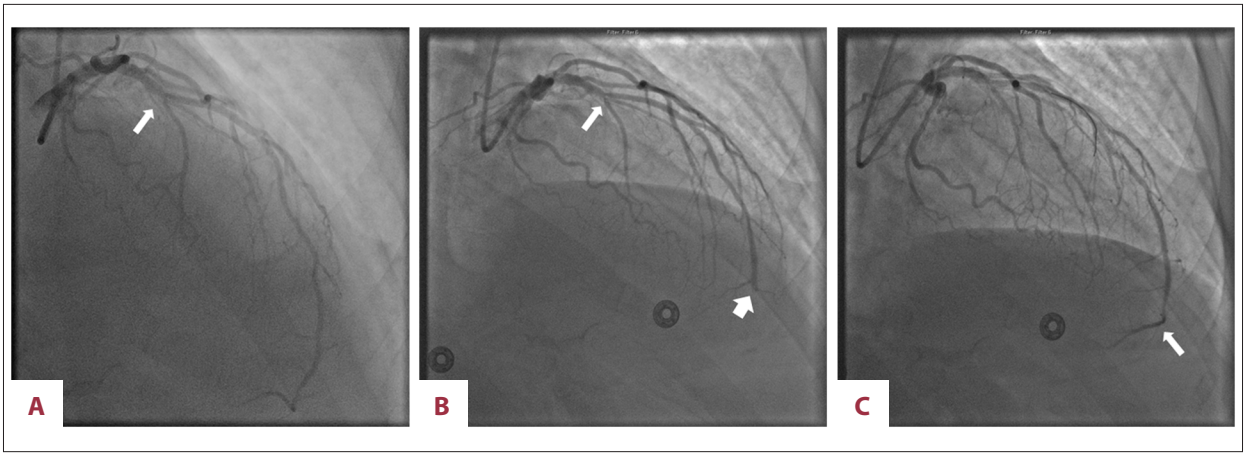
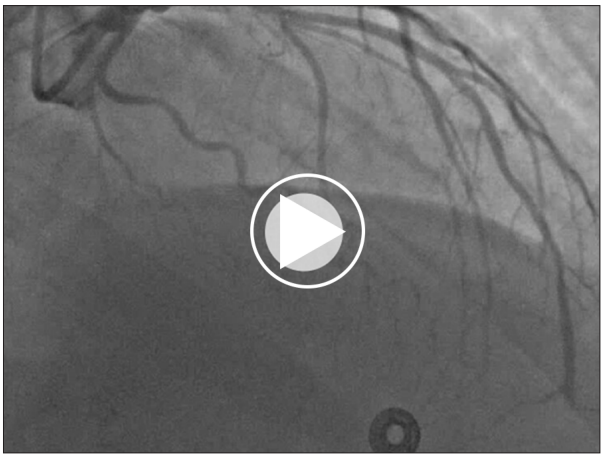
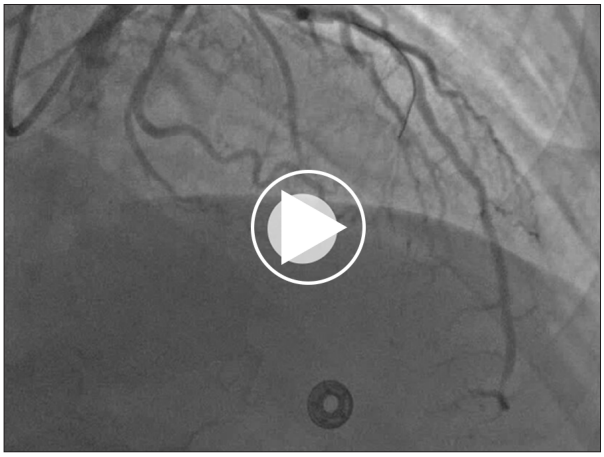


Figure 2. Coronary angiogram shows (**A**) moderate (60%) stenosis in the middle segment of the left anterior descending (LAD) artery (thin arrow), (**B**) stable stenosis in the middle segment of the (LAD) artery (thin arrow) and acute thrombotic occlusion of the distal LAD (thick arrow), and (**C**) complete restoration of the flow in the distal LAD (thin arrow).

Video 1. Coronary angiogram shows moderate (60%) stenosis in the middle segment of the left anterior descending (LAD) artery.



Video 2. Coronary angiogram shows stable stenosis in the middle segment of the (LAD) artery and acute thrombotic occlusion of the distal LAD.



Video 3. Coronary angiogram shows complete restoration of the flow in the distal LAD.

Conclusions

In conclusion, subtherapeutic anticoagulation therapy soon after direct-current cardioversion of atrial fibrillation can lead to potentially fatal coronary artery embolism and acute myocardial infarction. As demonstrated by our case, we suggest emergent thrombus aspiration and long-term treatment with triple antithrombotic therapy, including aspirin, P2Y12 receptor antagonist (e.g., clopidogrel), and factor Xa inhibitor or warfarin.

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