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Acute Myocardial Infarction Due to Coronary Artery Embolism in a 22-Year-Old Woman with Mitral Stenosis with Atrial Fibrillation Under Warfarinization: Successful Management with Anticoagulation

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Female, 22
Final Diagnosis: Acute myocardial infarction due to coronary artery embolism in a 22-year-old woman with mitral stenosis with atrial fibrillation under warfarinization: successful management with anticoagulation
Symptoms: Chest pain
Medication: —
Clinical Procedure: Coronary angiography
Specialty: Cardiology

Objective: Rare co-existance of disease or pathology

Background: Coronary artery embolization is an exceedingly rare cause of myocardial infarction, but a few cases in association with prosthetic mechanical valves have been reported. We report a case of embolic myocardial infarction caused by a thrombus in the left atrium with deranged coagulation profile in a patient with critical mitral stenosis under warfarinization.

Case Report: A 22-year-old woman was taken to the catheterization lab for early coronary intervention in lieu of non-ST elevation myocardial infarction. Electrocardiography showed T↓ in V_1 to V_4, and atrial fibrillation with controlled ventricular rate. Coronary angiography showed total occlusion of the mid-left anterior descending artery with thrombus. After upstream treatment with tirofiban, the apparent thrombus was dislodged distally while passing a BMW wire. No abnormalities were seen by intravascular ultrasound study. Echocardiography revealed critical mitral stenosis, and left atrial clot with mild left ventricular dysfunction. Coagulation profile revealed sub-therapeutic international normalized ratio levels. The sequential angiographic images, normal intravascular ultrasound study, and presence of atrial fibrillation are confirmatory of coronary embolism as the cause of myocardial infarction. Anticoagulation and treatment of acute coronary syndrome were initiated and she was referred for closed mitral valvulotomy.

Conclusions: Coronary artery thromboembolism as a nonatherosclerotic cause of acute coronary syndrome is rare. The treatment consists of aggressive anticoagulation, antiplatelet therapy, and interventional options, including simple wiring when possible. In this context, primary prevention in the form of patient education on optimal anticoagulation with oral vitamin K antagonist and medical advice about imminent thromboembolic risks are of extreme importance.

MeSH Keywords: Acute Coronary Syndrome • Angioplasty • Atrial Fibrillation • Embolism • Ultrasonography, Interventional

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/902250

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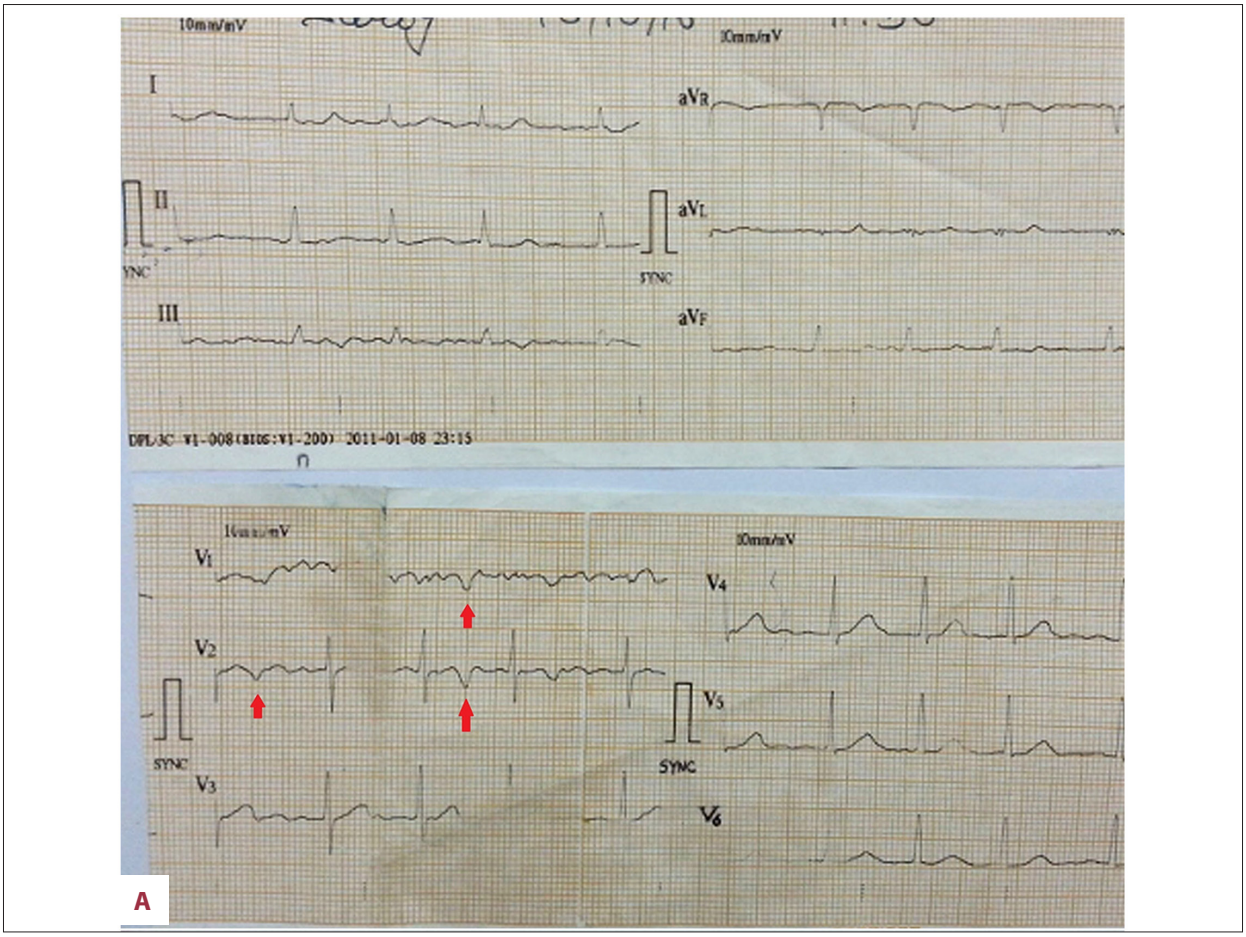
Background

Hypertension, smoking, dyslipidemia, and diabetes are the major causes of atherosclerotic coronary heart disease. Coronary embolism is rare among non-atherosclerotic causes of acute coronary syndrome. It has been reported in the background of cases of bacterial endocarditis, prosthetic valves, and intracardiac thrombus. It is even rarer in patients with mitral stenosis with atrial fibrillation. Furthermore, the effective treatment and management of coronary emboli in this setting remains unknown.

Case Report

A 22-year-old woman was admitted to our hospital with sudden onset of severe chest pain with perspiration of 6 h duration. She had no history of hypertension, smoking, dyslipidemia, diabetes mellitus, or substance abuse. She had been receiving metoprolol, diuretic, warfarin (2 mg), and prophylaxis for rheumatic fever for the last 6 years when rheumatic mitral stenosis was diagnosed. She had visited outpatient clinic 3 months ago, when her international normalized ratio (INR) was 2.1. An electrocardiogram showed T↓ in leads V_1 to V_2, and atrial fibrillation with controlled ventricular rate (Figure 1). In the laboratory analysis, creatine kinase MB (CKMB) was 47 U/L (upper limit 25 U/L), troponin I was 2.89 ng/mL (upper limit 0.01 ng/mL), and INR was 1.6. Blood pressure was 104/78 mm Hg in the right arm in supine position. Pulse rate was 74/min, irregularly irregular, with variable volume with no special character, with all peripheral pulses palpable. Jugular venous pulse pressure was elevated 6 cm above sternal angle with absent a wave. Apex beat was located in the right 5th intercostals space, tapping in character. There was a grade II right parasternal heave. P2 was palpable. S_1 was variable, S_2 was loud; loud P_2 component with narrow split. There was long grade IV mid diastolic rumbling murmur. The lungs were clear to auscultation. From clinical presentation, acute coronary syndrome (ACS) with non-ST elevation myocardial infarction was diagnosed. She was pre-treated with loading dose of clopidogrel (300 mg) and aspirin (325 mg). Urgent coronary angiography was performed through transfemoral route after proper consent. 6F sheath was put and 3000 U of Unfractionated heparin (UFH) was administered. Coronary angiography was normal except total occlusion of mid left anterior descending artery (LAD) with little trickling of contrast distally (Figure 2A).



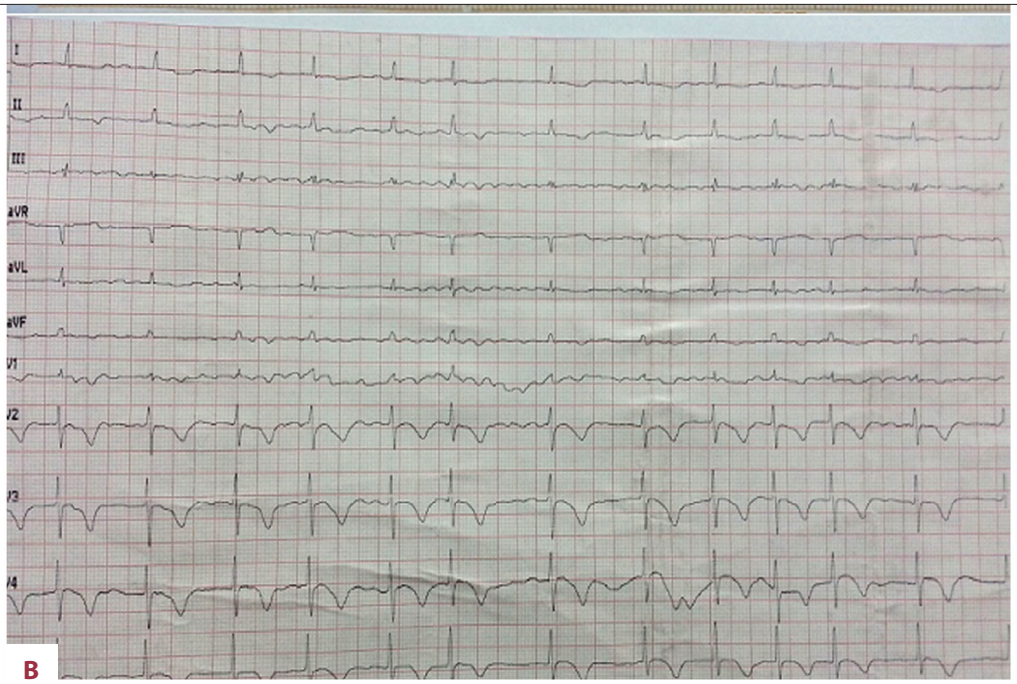


Figure 1. Electrocardiogram showing T↓ in V_1–V_2 at presentation (**A**) (red arrow); predischarge (**B**).

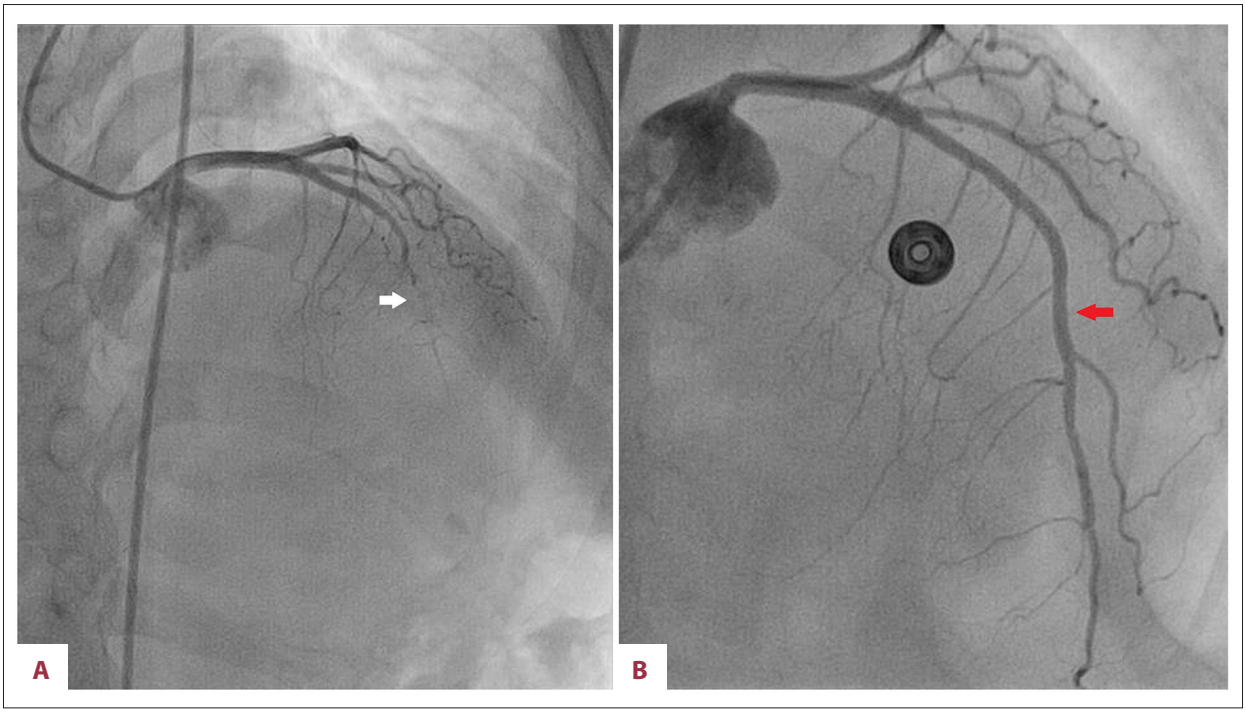


Figure 2. Coronary angiogram showing total occlusion of mid-LAD (white arrow) (**A**); recanalized LAD predischarge (**B**).

Left main artery was hooked with 6F EBU guiding catheter (Medtronic, USA) after giving further dose of 5000 U UFH. She also received 2 bolus doses of tirofiban (10µg/Kg/3 mint) upstream. LAD was wired with 0.014" BMW wire (Abbott, USA). Further injection revealed normal LAD artery with TIMI III flow as thrombus was dislodged distally (Figure 2B). Absence of underlying atherosclerosis was ruled out by using IVUS study (Volcano Corp, USA). Intracoronary nitroglycerin 200 µg was administered before putting IVUS transducer (30-MHz, 3.5-F electronic scanning; Volcano, USA) to avoid coronary spasm.

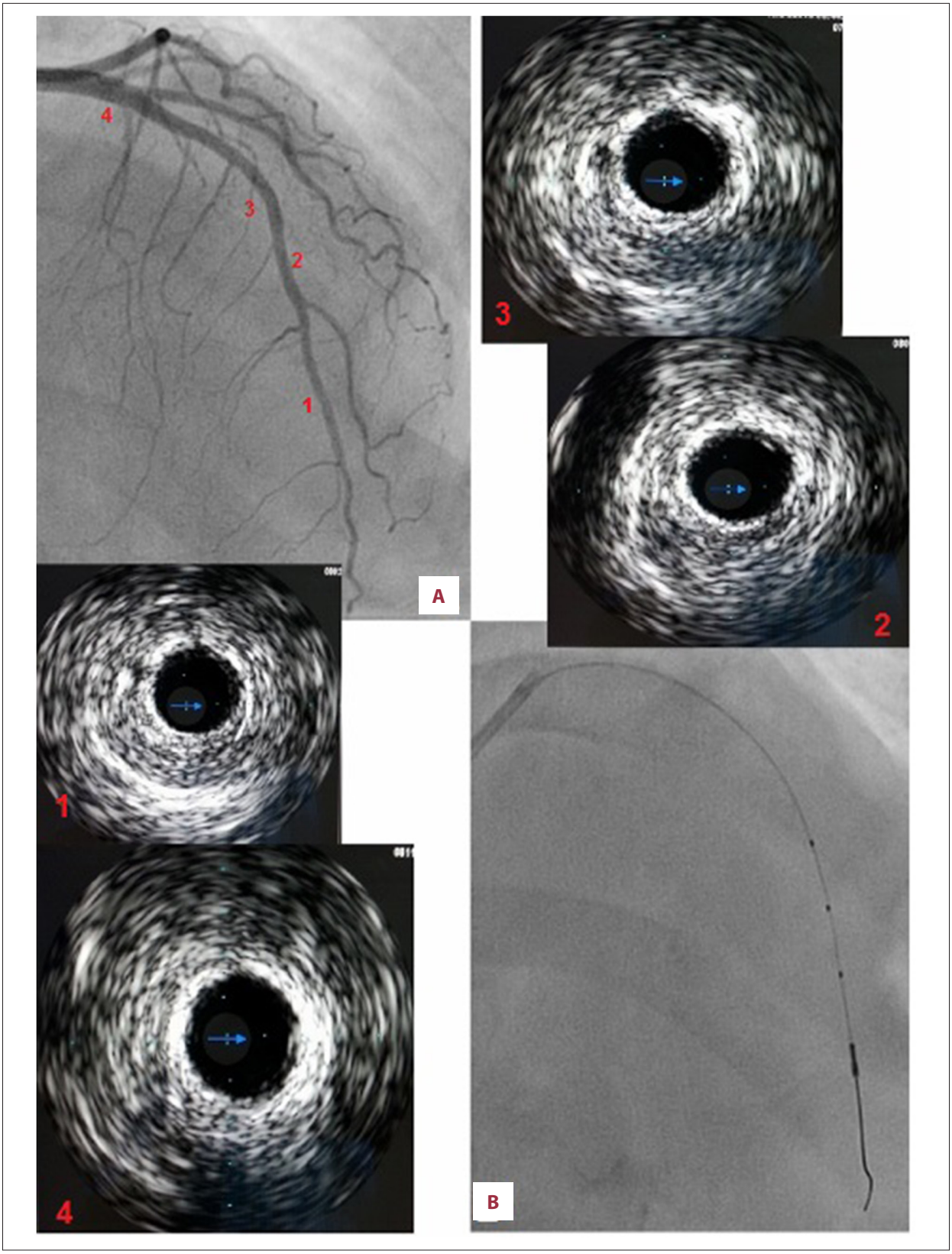


Figure 3. Recanalized LAD (**A**); IVUS probe *in situ* over the BMW wire (**B**); IVUS images at different points after gradual pull-back showing a normal artery.

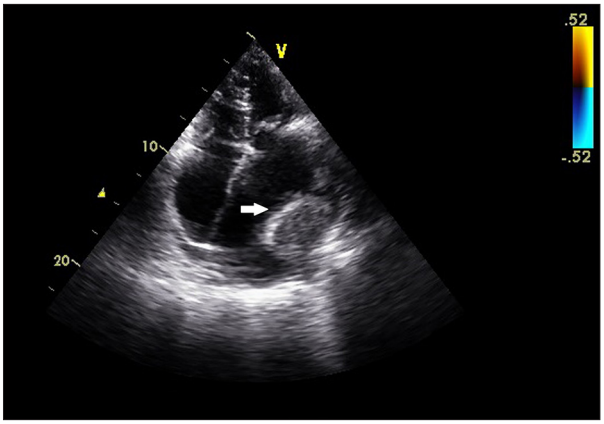


Figure 4. 2D transthoracic echo showing large left atrial clot (white arrow).

The transducer was parked to distal LAD over BMW wire and using manual pullback, images were recorded. It showed normal LAD without any atherosclerotic plaque (Figure 3A–3D). No further intervention was performed because of resolution of the angina and achievement of TIMI III flow in LAD. The patient was shifted to the coronary care unit where transthoracic echocardiography with Doppler interrogation revealed critical mitral stenosis, left atrial clot with mild left ventricular dysfunction (Figure 4). She was treated with dual antiplatelet (DAPT), beta-blocker, and diuretic. Enoxaparin was administered until the warfarin was in the therapeutic range of the international normalized ratio (INR). During further observation, the patient didn't have not any sign of systemic embolism and no complications occurred. The patient was discharged on DAPT, warfarin, beta-blocker, diuretic and advice of closed mitral valvulotomy. She was also counselled about importance of anticoagulation, prophylaxis measures against infective endocarditis and rheumatic fever.

Discussion

Coronary embolism should always be suspected especially in the context of sudden chest pain in patients with valvular prosthesis, chronic atrial fibrillation, dilated cardiomyopathy, infective endocarditis, intracardiac shunts, cardiac myxoma, mural thrombi, and hypercoagulable states with an incidence of 10 to 13% in autopsy series [1–3]. Coronary embolism occurs in the left coronary artery in 75% of cases and most of them present with ST elevation myocardial infarction which could be due to the preferential flow into the artery related to aortic valve morphology [4,5]. Our case was similar, except the presentation was non-ST elevation myocardial infarction. There is no consensus regarding treatment of acute coronary syndrome because of coronary embolism. Treatment options include conservative management including heparin, glycoprotein IIb/IIIa inhibitors, thrombolytic agents and invasive strategy in catheterization lab including thrombus aspiration and angioplasty [6–9]. We treated our patient with unfractionated heparin and tirofiban (glycoprotein IIb/IIIa inhibitor). Simple wiring helped dislodging the thrombus and underlying atherosclerotic lesion was ruled out by IVUS. As there is distal embolization of thrombus, possibility of no- reflow or slow flow is there and all measures should be exercised if it encountered. As far as duration of antiplatelets is concerned, there is no recommendation. It should be individualized [9], but anticoagulation is the cornerstone and close follow-up is needed because it predisposes the patient to higher risk of bleeding. Effective anticoagulation is very important for patients with mitral stenosis with atrial fibrillation; it alleviates the risk of embolism but does not completely eliminate it, as patients may encounter such episodes despite INR falling into therapeutic range. Our patient may have suffered some food or drug reaction causing the fall of her INR below the therapeutic range as her previous value was adequate. These patients should be educated about warfarin and possible adverse reactions with other drugs or food and need to be followed regularly. In individuals presenting with acute coronary syndrome, coronary embolism should be kept in mind in those with atrial fibrillation with rheumatic heart disease even in the absence of classical coronary risk factors, as was the case with our patient. To date, in all the case reports mentioning ACS in the embolic setting, diagnosis was inferential because conclusive evidence of absence of atherosclerosis was lacking [3,6–8]. Coronary embolism was confirmed by the specific angiographic images, the response of the thrombus to the wire manipulations, IVUS finding, and the clinical presentation.

Conclusions

Coronary artery thromboembolism as a nonatherosclerotic cause of acute coronary syndrome is a rare finding with unknown prevalence. The treatment consists of aggressive anticoagulation, antiplatelet therapy, and interventional options, including simple wiring when possible. In this context, primary prevention in the form of patient education on optimal anticoagulation with oral vitamin K antagonist and medical advice about imminent thromboembolic risks are of extreme importance.

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