80. Coronary Artery Embolism as the Culprit of ST-Elevation Myocardial Infarction in Rheumatic Heart Disease Patient: A Case Report

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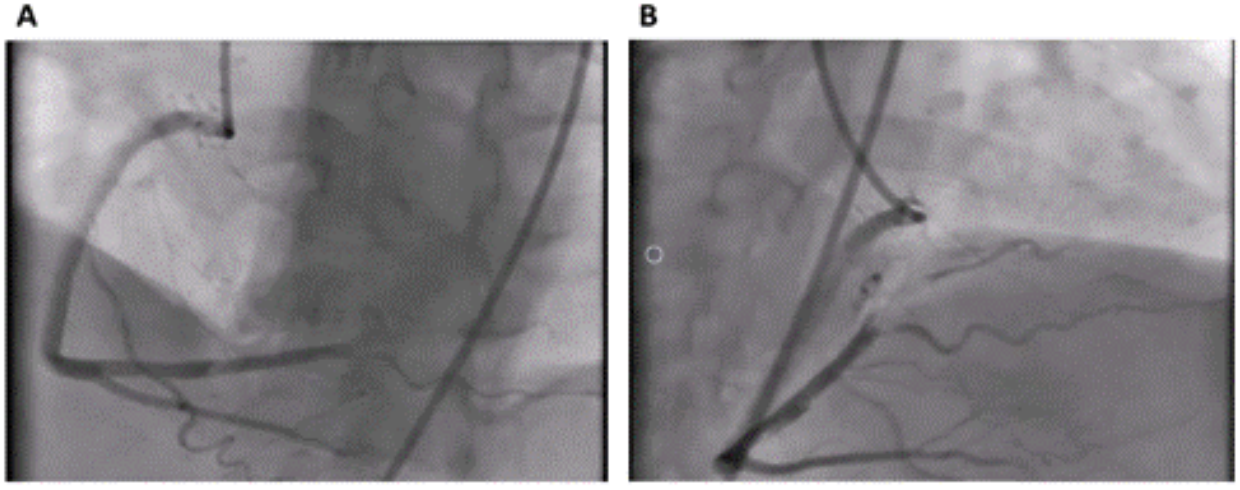
Body

Background: 90% the etiology of ST-Elevation Myocardial infarction (STEMI) is caused by coronary event such as plaque rupture indicating the occurrence of myocardial infarction (MI). In some cases, coronary artery embolism (CAE) caused by thrombi in atrial fibrillation subject with rheumatic heart disease (RHD) is hard to differentiate in acute setting and may had different treatment approach.

Case: 55 years old woman presented with chest pain 11 hours before admission. She was referred from regional hospital and was given dual antiplatelet, low molecule weight heparin, nitrat and morphine injection. She was ex-smoker as risk factor and known to have history of RHD 3 years before admission and did not take medication routinely. She had stable hemodynamic with irregular rate. ECG showed normoventricular atrial fibrillation, STEMI infero-posterior with evolution. Laboratory showed increased Troponin I value >10 ng/ml. Coronary angiography revealed sub total occlusion in RPLA suggestive due to distal thromboembolism. The treatment was changed to heparin adjusted dose based on APTT value overlap with warfarin 2 mg orally per 24 hours without antiplatelet. Echocardiography showed dilated left atrium with reduce ejection fraction 38%, global hypokinetic, severe mitral stenosis, no sec or thrombus found. After 2 weeks of anticoagulant treatment (warfarin 3 mg with latest INR 2,67) she underwent coronary angiography evaluation and revealed no visible thromboembolic at distal RCA.

Discussion: Coronary artery embolism is a rare cause of acute MI and the precise diagnosis remains challenging. The diagnosis of CAE made based on conventional angiographic features specific for coronary occlusion as globular filling defects, horse-riding thrombi, or multiple filling defects. Other characteristic were no atherosclerotic findings in the coronary trees, presence other predisposing factors or absence of significant stenosis at the culprit lesion after thrombus aspiration. No specific guidelines in management of CAE. In our case, we preferred medical management and proved successful in angiography evaluation.



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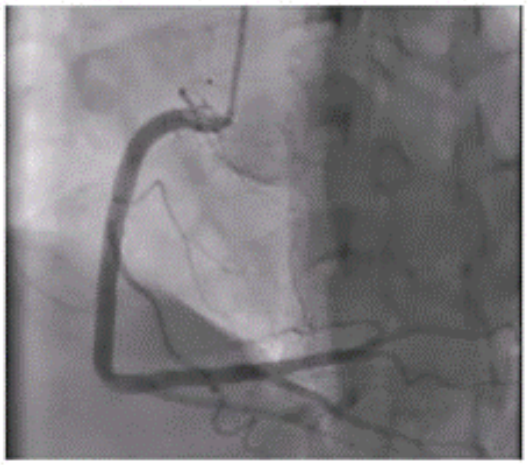

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