Rheumatic Heart Disease Predisposing to Embolic Myocardial Infarction: A Multimodality Imaging Approach

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Abstract

We report a clinical case of a 45-year-old male with a diagnosis of inferior myocardial infarction and previous history of rheumatic fever during his childhood. Coronary angiography demonstrated normal coronary arteries. Transthoracic echocardiogram showed hypokinetic left ventricular inferolateral wall and mitral stenosis; furthermore, speckle tracking analysis revealed reduction of global longitudinal strain involving the inferior wall. A three-dimensional transesophaegeal echocardiography, performed to better characterize the anatomy of the valve and to find possible source of embolic infarct in an enlarged left atrium, showed rheumatic valvular involvement. Cardiac magnetic resonance confirmed the ischemic damage and also provided prognostic information. A multimodality imaging approach should be mandatory in patients with acute myocardial infarction and normal coronary angiography, to define possible sources of embolic infarction and to quantify myocardial damage.

Keywords

- mitral valve stenosis
- rheumatic heart disease
- embolic myocardial infarction
- speckle tracking analysis
- cardiac magnetic resonance

Case Report

A 45-year-old male, with a previous history of rheumatic fever during his childhood, was transferred to our Cardiac Intensive Care Unit after a diagnosis of ST Elevation Myocardial Infarction (STEMI) in a non-percutaneous coronary intervention (PCI)-capable hospital. At admission in the emergency department, the patient presented a typical chest pain radiating to the left arm, lasting more than 30 minutes. The electrocardiogram showed a ST segment elevation in inferior leads with a sinus rhythm (►**Fig. 1**). Fibrinolytic therapy with tenecteplase was administered according to drug scheme. Due to the persistence of ST segment elevation in inferior leads and mild chest pain, he was transferred to our Cardiology Department for a rescue PCI. At admission in our catheterization laboratory, the patient reported a relief in chest pain and the ST segment was near to isoelectric line. The selective coronary angiography showed normal coronary arteries (►**Fig. 2**).

On auscultation, the first heart sound was loud, S2 was normally split with normal intensity of P2 and there was an audible opening snap. A short 3/6 murmur with presystolic accentuation was also heard over the apex. The blood exams revealed increased myocardial damage markers (peak troponin isoform T $>$ 50 ng/mL; creatine kinase isoform MB 60 ng/mL).

The following transthoracic echocardiogram showed a hypokinesis of the left ventricular inferolateral wall, with dilated left atrium and moderate-to-severe mitral valve stenosis (MS) (diastolic maximum gradient 25 mm Hg, medium 10 mm Hg). The left ventricle had normal dimensions with mild systolic disfunction (ejection fraction [EF] 48%). Furthermore, speckle tracking imaging (STI) analysis was also performed and demonstrated a significant reduction of global longitudinal strain involving the inferior wall (►**Fig. 3**).

Patient underwent a full three-dimensional volumetric transesophageal echocardiogram (►**Fig. 4**) to better define the anatomy of the valve (Wilkins score 10): the planimetric

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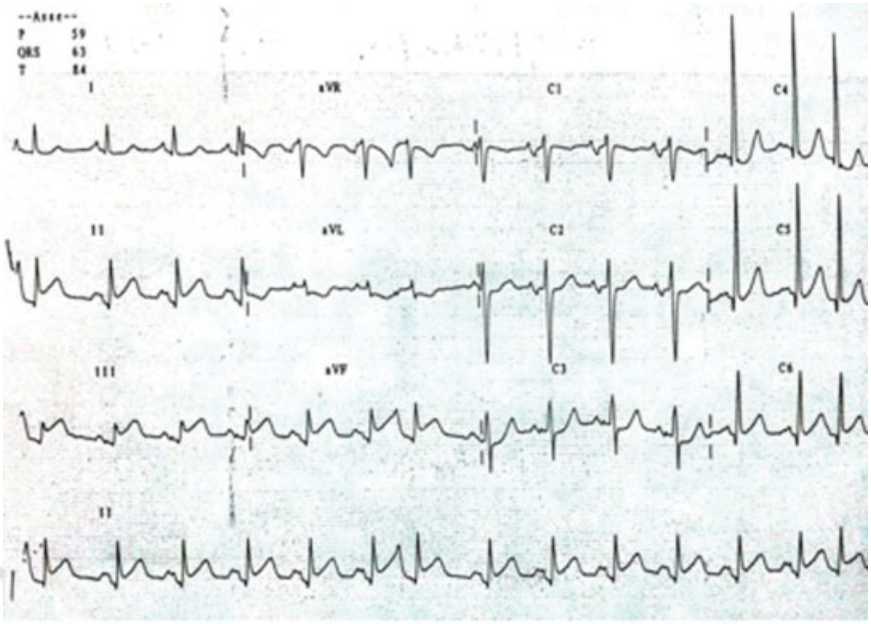

Fig. 1 Electrocardiogram shows ST elevation in inferior leads (DII, DIII, aVF), with sinus rhythm on admission.

valve area was 1 cm^2, and a spontaneous echo-contrast in left atrium and its appendage were revealed, without evidence of intracardiac thrombi. The typical features of rheumatic mitral valve involvement were also noted, with small calcifications, fusion of the leaflet commissures, thickening of the leaflet cusps, and shortening of the chordae tendineae.

Finally, patient underwent cardiac magnetic resonance (CMR) (►**Fig. 5**) that revealed a hypointense core within the edematous area on T2-weighted images related to intramyocardial hemorrhage and transmural extent of delayed enhancement in the mid-apical inferolateral segments associated with no-reflow phenomena, compatible with reperfused STEMI. On SSFP cine sequences, moderate-to-severe MS with thickening of the leaflets commissures was confirmed, together with mild aortic insufficiency and left atrial enlargement.

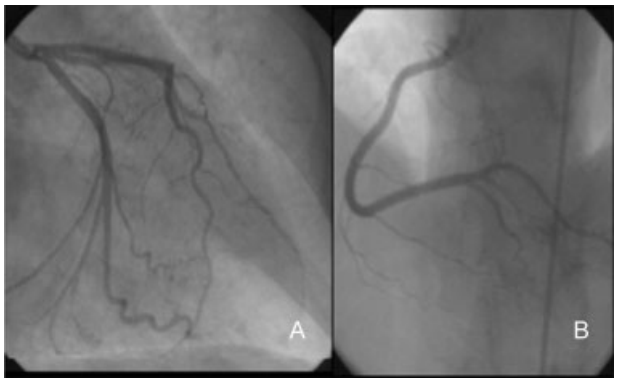

Fig. 2 Selective coronary angiography. (A) Left coronary arteries and (B) right coronary artery were normal.

The in-hospital clinical course was uneventful and the patient was discharged on bisoprolol, atorvastatin, aspirin, and warfarin, and will return for balloon mitral valvuloplasty.

Discussion

We described a case of a patient with previous history of rheumatic fever, presenting with an inferolateral acute STEMI, normal coronary arteries, MS, and left atrial enlargement.

Correlating these findings to the absence of cardiovascular risk factors, the most likely explanation could be an embolic STEMI probably from a left atrial thrombus, associated with a moderate-to-severe MS and rheumatic heart disease.

Embolic acute myocardial infarction (E-AMI) is an infrequent cause of acute coronary syndrome described since 1980s,[1] but the association between valvular heart disease and coronary involvement is well known.[2–5] The thrombus could not always be visible at coronary angiography, as in our case.[6] In most cases with AMI and MS, embolic events involve the left anterior descending coronary artery,[7] but also the right coronary was occasionally found as culprit lesion.[5]

It is also interesting to notice that E-AMI associated with MS often occurs in sinus rhythm,[7,8] as we observed, and not only in atrial fibrillation. In fact, MS may be associated with increased left atrial coagulation activity and therefore constitutes a significant risk for intracardiac thrombi even among patients in sinus rhythm[9]; therefore, a prospective study found an incidence of systemic embolism of 9.1% among patients with MS in sinus rhythm over a 3-year follow-up.[10] These findings suggest that when E-AMI is diagnosed in patients with valvular heart disease, an oral anticoagulant therapy should be considered independently by heart rhythm.[7]

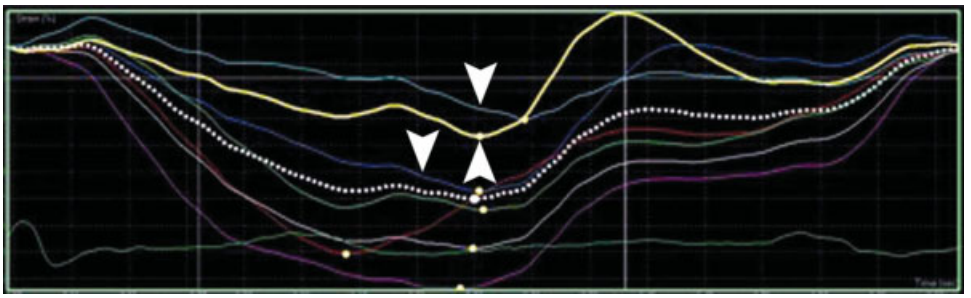

Fig. 3 Two-chamber speckle tracking imaging analysis demonstrated a reduction of global longitudinal strain (GLS) involving the inferior wall: the strain curves corresponding to the inferior wall (basal: teal; mid: yellow; apical: light blue, indicated by arrowheads) were reduced rather than the anterior strain curves (basal: green; mid: red; apical: purple; violet color indicates the apex), with a medium GLS value of − 16.

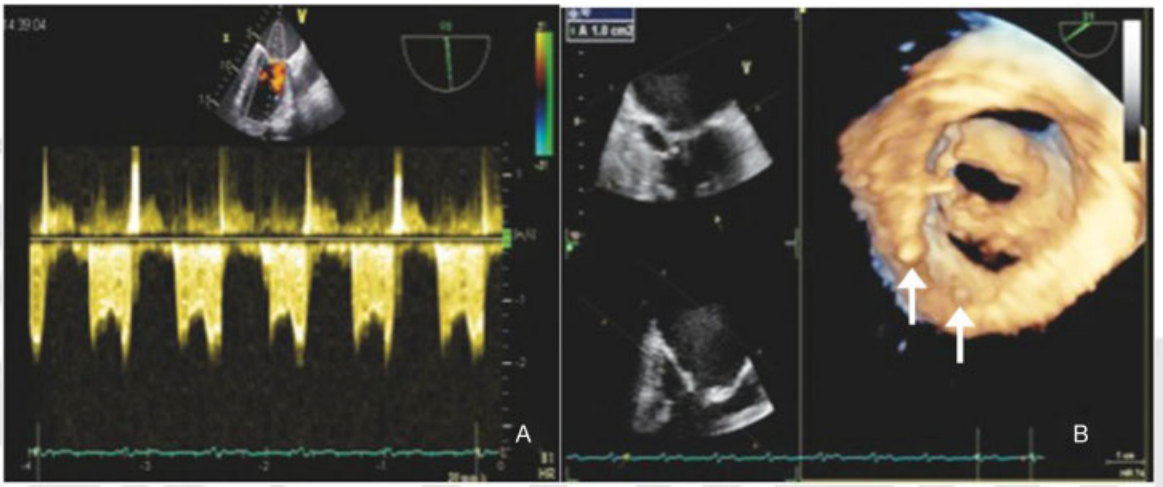

Fig. 4 (A) Three-dimensional (3D) transesophageal echocardiography showed diastolic gradients of moderate-to-severe mitral stenosis and (B) 3D valve area with small calcifications (arrows) and thickening of the leaflet cusps. A spontaneous echo-contrast in left atrium and its appendage could also be noted.

However, this case demonstrates that in patients presenting with AMI and normal coronary arteries, other imaging techniques could be used, such as ultrasound with STI and CMR to better define valvular anatomy, provide prognostic information, and confirm the diagnosis of AMI.

STI is based on tracing of pixel groups in grayness scale for the quantitative measurement of myocardial strain and myocardial strain rate. A close correlation between strain analysis and both wall motion score index and left ventricular EF in ischemic patients was demonstrated[11]: in fact strain analysis

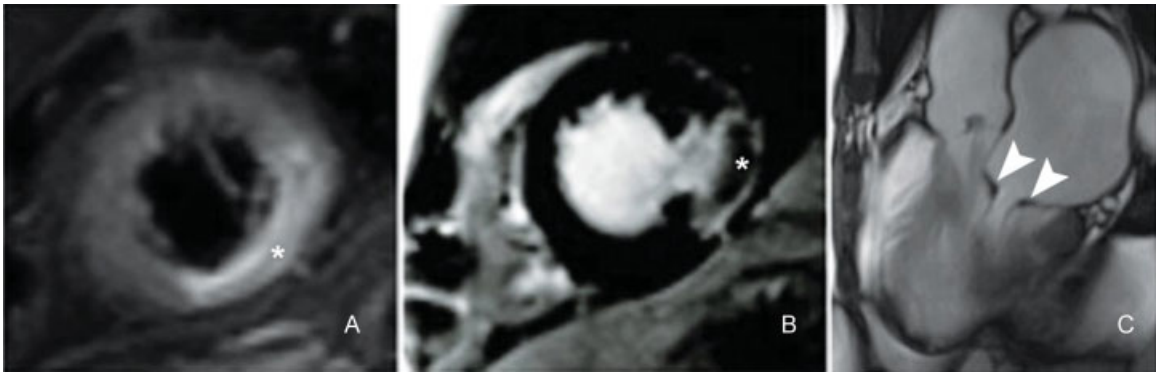

Fig. 5 (A) Cardiac magnetic resonance short axis T2-weighted short tau inversion recovery image revealed a hypointense core rim (*) related to intramyocardial hemorrhage within the hyperintense edematous area in mid-apical inferolateral segments. (B) Short axis T1-weighted inversion recovery image after 15 minutes from gadolinium injection showed transmural extent of delayed enhancement in the corresponding segments due to myocardial infarction associated with large no-reflow phenomena (*). (C) SSFP cine sequences at the left ventricular inflow–outflow tract in diastole illustrate moderate-to-severe mitral valve stenosis with thickening of the leaflets commissures (arrowheads) and left atrium enlargement. Note the concomitant mild aortic valve insufficiency.

assessed by STI can be considered a very sensitive method in assessing even small changes of left ventricular function. Furthermore, the additional value of strain analysis is related to the unique opportunity to identify transmural extent of necrosis, giving information beyond traditional echocardiographic indexes.[11] In particular, left ventricular global strain by STI demonstrated a strong correlation with infarct size investigated by CMR in the acute phase of patients with STEMI treated with thrombolysis.[12] Moreover, a recent study by Kansal et al,[13] performed on a heterogeneous population of patients with different extent of myocardial scar at CMR, showed the reduction of longitudinal strain independently by delayed enhancement distribution when compared with healthy volunteers.

Finally, the evidence of a hypointense core in the edematous area on T2-weighted sequences at CMR, recognized as a noninvasive marker for intramyocardial hemorrhage, is associated with more adverse clinical outcomes in patients with reperfused STEMI,[14] with a significant impact on patient's prognosis.

Conclusion

E-AMI understanding implies a careful stratification of patient's prognosis and an exhaustive characterization of myocardial necrosis, especially in case of rheumatic heart disease and normal coronary arteries. In our case, STI was useful to identify signs of myocardial ischemic injury and correlated with transmural extent of the AMI. Transesophageal echocardiography allowed an accurate depiction of valvular anatomy revealing rheumatic heart disease features. CMR permitted to demonstrate the presence of myocardial infarction associated with intramyocardial hemorrhage, also providing prognostic information.

In conclusion, a multimodality imaging approach should be mandatory in patients presenting with normal coronary arteries and E-AMI, to define its source, quantify myocardial damage, and better define prognosis.

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