Acute Myocardial Infarction Following Blunt Chest Trauma

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Introduction

Acute myocardial infarction following blunt chest trauma is a rare, potentially fatal and often undiagnosed complication, due to the fact that chest pain can be attributed to the trauma itself.

This is a case of acute myocardial infarction with evidence of significant transmural infarction on echocardiography and cardiac magnetic resonance imaging in a 39-year-old patient with no risk factor for acute coronary failure following blunt chest trauma.

Case Report

M.S.L., a 39-year-old male retailer from Rio de Janeiro, with no history of illicit drug use, with a history of trauma in the thoracic dorsal region after falling from the roof of his house, from a height of about 3 meters. He sought medical care due to intense precordial pain triggered after physical exertion one day after the trauma.

A 12-lead electrocardiography (ECG) was conducted and material was collected to look for markers of myocardial necrosis. ECG identified BRD and elevation of the markers (total creatine phosphokinase, fraction and troponin).

One month after the event, he had an echocardiography done, which revealed an akinetic area with thinning and increased echogenicity, which suggested the presence of fibrosis in the mid anteroseptal segment (Figures 1-3). Cardiac magnetic resonance imaging showed late contrast uptake with transmural distribution in this segment (Figures 4-6).

Coronary angiography was performed one week after echocardiography and revealed distal septal branch stenosis of the anterior descending artery (Figure 7).

Although coronary artery lesions occur in less than 2% of the cases of blunt chest trauma, acute myocardial infarction occurs in less than 1% of the patients, and the anterior descending artery is the most frequently involved (72% of the cases). Blunt chest trauma can cause damage to various structures of the heart (in the myocardium, coronary arteries, pericardium and heart valves) and can lead to severe complications – not only myocardial infarction but also arrhythmia and sudden death.

Keywords

Myocardial Infarction; Echcocardiography/methods; Electrocardiography/methods; Accidental Falls; Thoracic Injuries.

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By promoting direct chest compression, acceleration or deceleration, it generates a shear force at the arterial endothelium level, leading to coronary dissection and acute myocardial infarction by the following proposed mechanisms: intracoronary thrombosis, coronary artery dissection, vasospasm or atherosclerotic plaque rupture. We cannot rule out the hypothesis of a mere temporal association between the event and the infarction, since we cannot rule out the presence of coronary lesions prior to the trauma, but there is a great cause and effect relationship. The patient is under clinical follow-up.

Conclusion

Although there is no clear consensus in the literature to guide the identification of post-trauma acute myocardial infarction, it should always be part of differential diagnosis in patients who are victims of chest trauma. Precordial pain should neither be neglected nor just attributed to the event. It is of great importance to perform electrocardiography scans and collect myocardial necrosis markers in the suspicion of acute coronary syndrome. The association of post-traumatic chest pain and arterial ST-segment elevation suggests a coronary angiography scan to rule out this hypothesis, making diagnostic efficiency and immediate treatment to be essential. Myocardial contusion may produce electrocardiographic abnormalities similar to ST-segment elevation, ST-segment depression and new Q waves, being the main differential diagnosis. As a general rule, an individualized approach should be taken.[1-6]

Authors' contributions

Data acquisition: Silva RC, Gottlieb I, Sá GA, Felix AS, Rawet DB, Paulino MR, Adriano EF, Poffo MR, Feijó ALF; Data analysis and interpretation: Silva RC, Gottlieb I, Sá GA, Felix AS, Rawet DB, Paulino MR, Adriano EF, Poffo MR, Feijó ALF; Manuscript writing: Silva RC, Gottlieb I, Sá GA, Felix AS, Rawet DB, Paulino MR, Adriano EF, Poffo MR, Feijó ALF; Critical revision of the manuscript as for important intellectual content: Silva RC, Gottlieb I, Sá GA, Felix AS, Rawet DB, Paulino MR, Adriano EF, Poffo MR, Feijó ALF.

Potential Conflicts of Interest

There are no relevant conflicts of interest.

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Academic Association

This study is not associated with any graduate program.

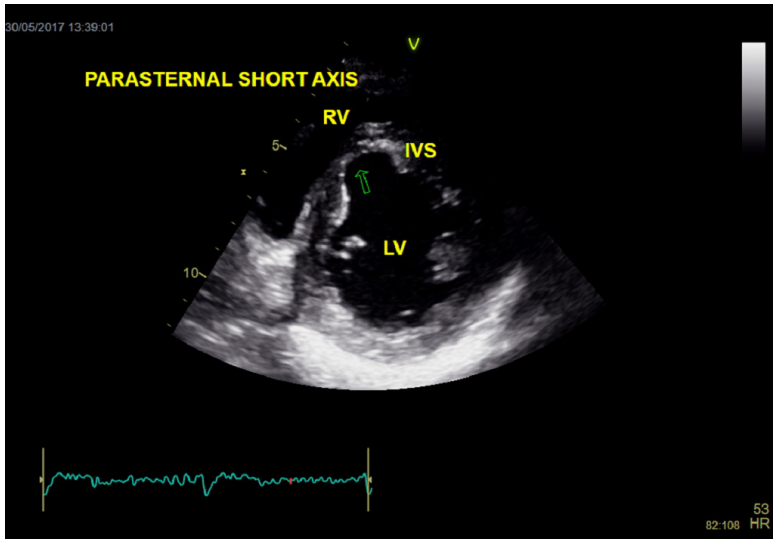

Figure 1 – *Transthoracic echocardiography. Parasternal short axis showing aneurysm and mid anteroseptal wall thinning.*

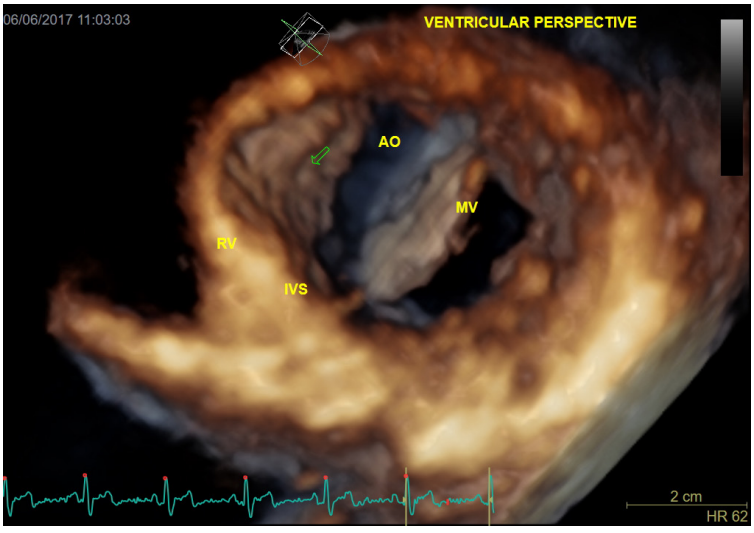

Figure 2 – *Three-dimensional transthoracic echocardiography. Ventricular view of the aneurysm. RV: right ventricular; IVS; interventricular septum; AO: aorta; MV: mitral valve.*

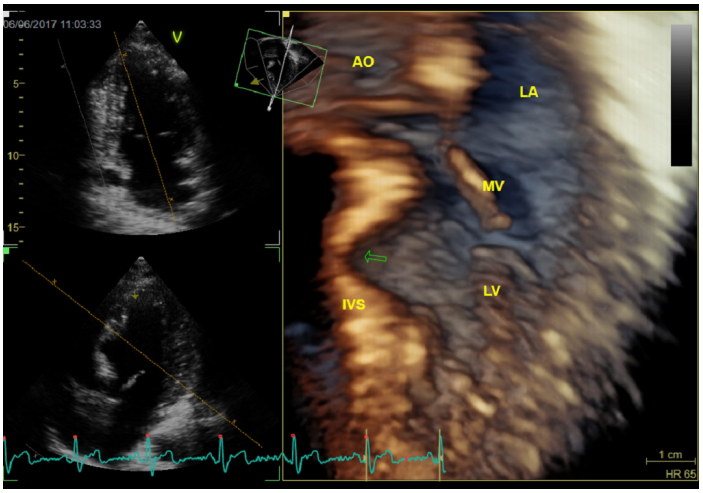

Figure 3 – *Three-dimensional transthoracic echocardiography showing mid anteroseptal aneurysm. AO: aorta; LA: left atrium; MV: mitral valve; IVS: interventricular septum; LV: left ventricle.*

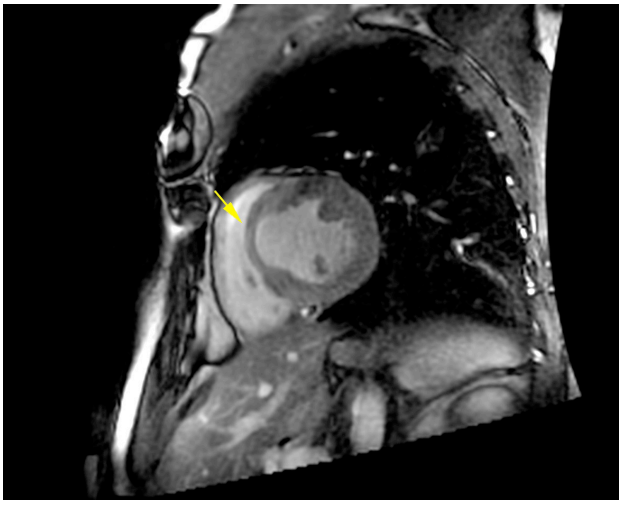

Figure 4 – *Cardiac magnetic resonance imaging. Steady-state free precession (SSFP) coronary angiography sequence on a short axis in systole, showing mid anteroseptal aneurysm.*

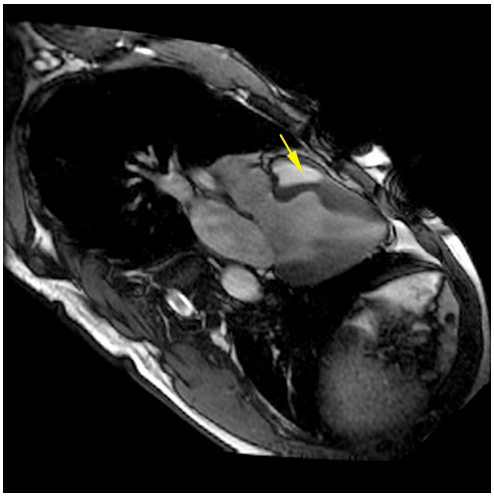

Figure 5 – *Cardiac magnetic resonance imaging. Steady-state free precession (SSFP) coronary angiography sequence of the left ventricular outflow tract in systole showing the mid anteroseptal aneurysm.*

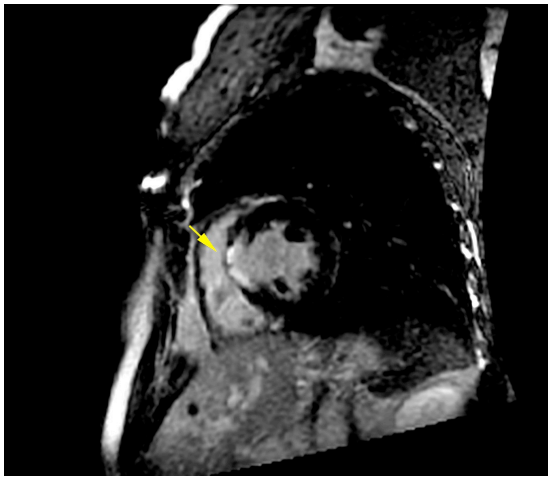

Figure 6 – *Cardiac magnetic resonance imaging. Short axis view showing late gadolinium uptake with transmural distribution in the mid anteroseptal segment.*

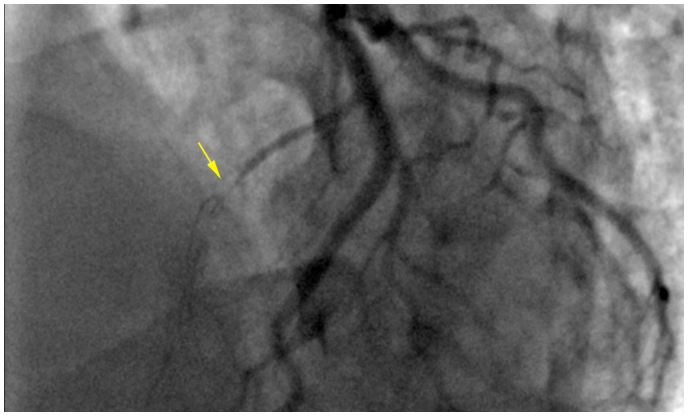

Figure 7 – *Coronary angiography. Left anterior oblique view of the head. First septal branch of the left anterior descending artery with severe stenosis.*

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