



## Case Report

## Acute myocardial infarction due to left main embolization of calcified tissue from mitral valve subapparatus



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## ABSTRACT

An 81-year-old woman was referred for primary angioplasty due to a myocardial infarction. Upon her arrival, the patient was in cardiogenic shock. Coronarography revealed a large filling defect within the left main coronary artery. Thromboaspiration was performed, obtaining thrombotic material and tissue of different consistencies. Balloon angioplasty in the left anterior descending and left main arteries was performed, resulting in incomplete reperfusion, leading to irreversible electromechanical dissociation. Analysis of the aspirated material was consistent with thrombus, atheroma, and calcified tissue. Autopsy revealed a heavily calcified mitral valve, and distal embolization of amorphous material in the microvasculature identical to that found in the mitral valve subapparatus.

<Learning objective: Acute myocardial infarction due to left main embolization of tissue from mitral valve subapparatus is a rare condition but lethal. Early recognition of this condition is important for establishing the best option of treatment, between a percutaneous or surgical approach.>

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## Introduction

Mitral annular calcification is a chronic degeneration of the mitral valve fibrous ring, is common in elderly women, and in some cases this pathology may be complicated by systemic embolization such as retinal artery occlusion, cerebrovascular accidents, and acute coronary syndrome (ACS) [1–4]. The postulated mechanisms include embolization of small calcified tissue, ulceration, and thrombus formation and embolization [1]. Anticoagulation and surgery could be considered in patients with mitral annular calcification and who present with embolic manifestations [1–4].

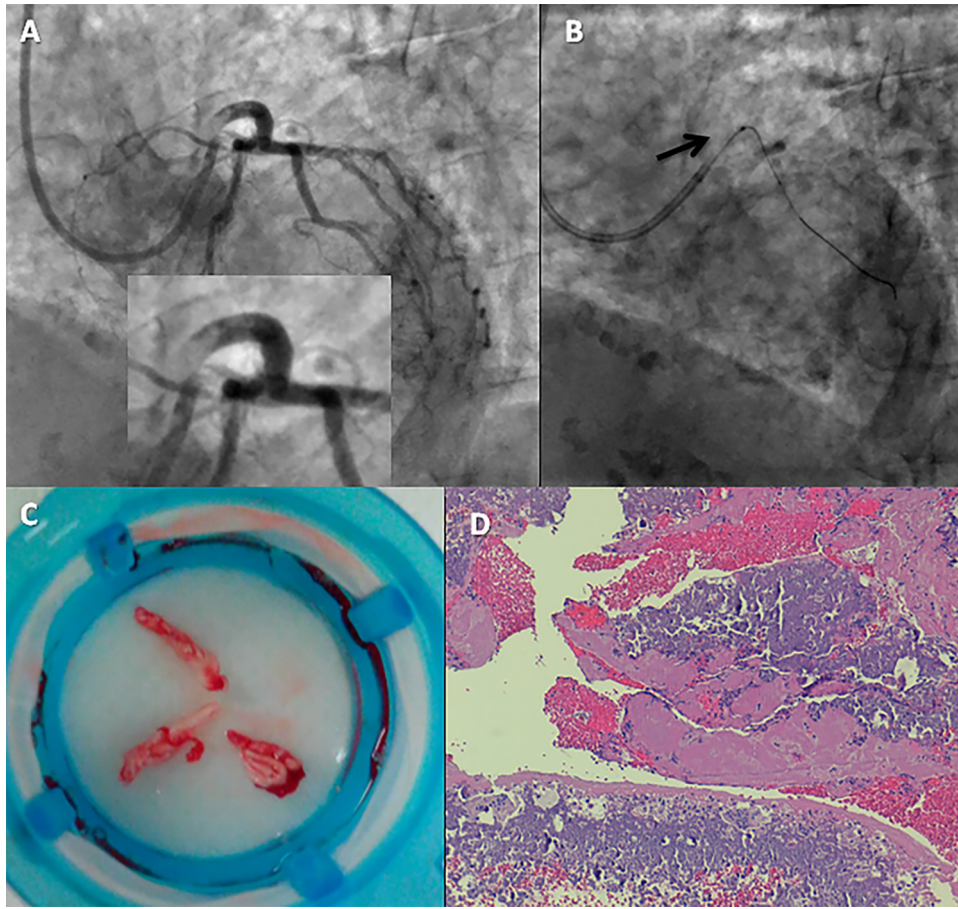
## Case report

An 81-year-old woman was referred to our hospital for syncope in the street. The basal electrocardiogram showed ST-segment

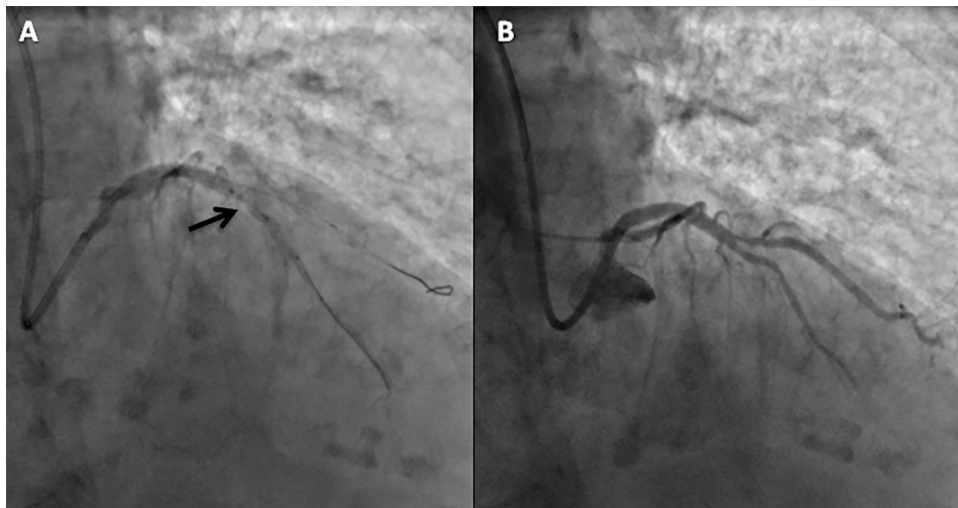
elevation in the anterior precordial leads. At her arrival, the patient was in cardiogenic shock. Coronary angiography revealed a large filling defect within the left main coronary artery (LMCA) with possible thrombus image in the first projections (Fig. 1A and Supplementary Video S1 in the online version at DOI:10.1016/j.jccase.2017.02.002). Thromboaspiration with two different thromboaspiration catheters (Pronto™, Vascular Solutions, Minneapolis, MN, USA and Hunter™, Innovative Health Technologies, Barcelona, Spain) was performed (Fig. 1B and Supplementary Video S2 in the online version at DOI:10.1016/j.jccase.2017.02.002), obtaining thrombotic material and tissue of different consistencies (Fig. 1C, D). Balloon angioplasty in the LMCA and left anterior descending (LAD) coronary artery was performed (Supplementary Video S3 in the online version at DOI:10.1016/j.jccase.2017.02.002) resulting in distal embolization and incomplete coronary reperfusion (Supplementary Video S4 in the online version at DOI:10.1016/j.jccase.2017.02.002). Intracoronary adenosine perfusion through a microcatheter (Amicath™, Innovative Health Technologies) into the distal LAD (Supplementary Video S5 in the online version at DOI:10.1016/j.jccase.2017.02.002) and first diagonal branch was performed (Fig. 2A), without achieving microvascular reperfusion (Fig. 2B). Rapidly, the patient progressed to irreversible electromechanical dissociation. Histopathology analysis of the aspirated

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**Fig. 1.** (A) Coronary angiography image showing possible thrombus in the left main coronary artery (and magnified view). (B) Thromboaspiration catheter (arrow). (C) Material obtained from thromboaspiration catheter, and its (D) histopathology cohort compound of amorphous and calcified tissue.



**Fig. 2.** (A) Intracoronary adenosine perfusion through a microcatheter (arrow). (B) Coronary angiography image showed poor final distal coronary flow.

material was consistent with thrombus, atheroma, and calcified tissue. Autopsy revealed heavily calcified mitral valve leaflets and subapparatus (Fig. 3A), previously visualized by transthoracic echocardiogram (Fig. 3B), and distal embolization of amorphous

calcified material in the myocardium microvasculature identical to that found in the mitral valve subapparatus (Fig. 3C, D).

Autopsy revealed a mitral valve annulus, leaflets, and subapparatus with severe and homogenous calcification. Distal

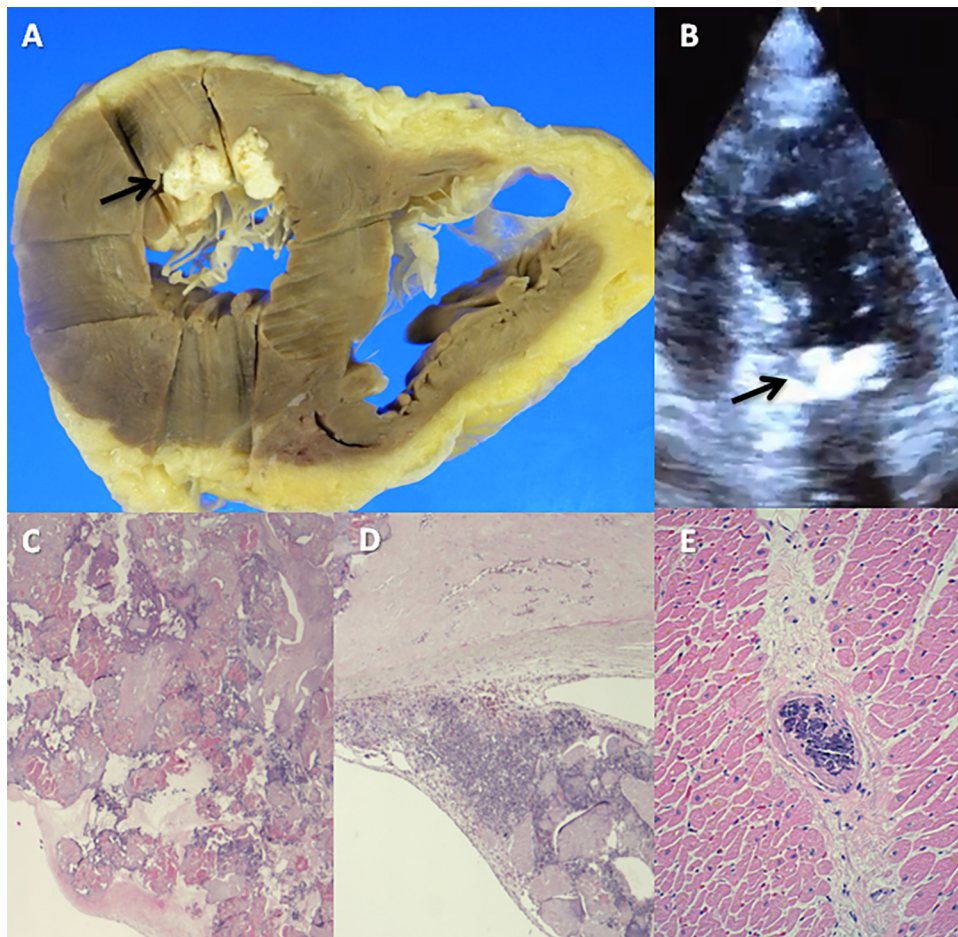


Fig. 3.

(A) Heart cross-section showing severe mitral annular calcification with infiltration areas in the posterior wall (arrow). (B) Transthoracic echocardiogram showed severe calcification of the mitral annulus and valvular subapparatus (arrow). (C) Histopathology cohort of material obtained from mitral valve subapparatus, showing the presence of acellular and amorphous material, similar to atheroma, with calcification and osteoclast-type giant cells, (D) comparable to that found in left main coronary artery. (E) Distal coronary microvasculature with the same embolized material.

embolization in coronary microvasculature (Fig. 3E) of amorphous calcified material with osteoclast-like giant cells in the myocardium microvasculature identical to that found in the mitral valve subapparatus was detected, the distal embolization corresponding with poor final angiographic distal flow (Supplementary Video S6 in the online version at DOI:10.1016/j.jccase.2017.02.002).

## Discussion

Most cases of ACS and LMCA occlusion involve atherothrombotic disease, but few of them are related to left main embolization of foreign material or tissue from other cardiac structures [1–3]. In our case, the patient had an acute myocardial infarction with left main occlusion due to embolization of calcified material from the mitral valve subapparatus. This was corroborated by comparing tissue obtained from the aspiration catheter during primary angioplasty with the histopathology analysis of tissue from the mitral valve by autopsy. Furthermore, the image of a calcium spicule in the coronary angiogram was similar to that obtained in the autopsy from the LMCA.

Another differential diagnosis would be the presence of a calcified nodule in the LMCA that caused subsequent thrombosis. However, this diagnosis seems unlikely due to several reasons: the calcified nodule could hardly be extracted by a thromboaspiration catheter such as the one used; the angiographic image after thromboaspiration showed the LMCA without significant stenosis

or calcification, and the histopathological analysis of the extracted material of the LMCA would be different from that of the mitral valve.

We believe that our case exemplifies an unusual angiography image occluding the LMCA in the setting of an emergency coronary intervention along with macroscopic and histopathology findings. This angiographic image reminds us of the possibility of non-thrombotic left main embolization, which might involve different treatment decisions such as a surgical approach [1–3].

## Conflict of interest

All authors reported no conflict of interest related to the contents of this paper.

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