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Unusual Myocardial Calcification

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Abstract

We report an infrequent case of focal myocardial calcification in an asymptomatic 56-year-old male, with controlled arterial hypertension and hypercholesterolemia, there was no history of impaired calcium metabolism, or impaired renal function. Although the different study methods were negative for myocardial infarction, it could be classified as idiopathic, however, we thought it would be a diastrophic calcification mechanism, as explained in the description.

Introduction

The presence of cardiac calcifications is rare and in most cases they are pathological; its etiology is varied, but usually they are divided according to the mechanisms of production as dystrophic or metastatic. Therefore, it is important to inform and characterize myocardial calcifications when they are visible in diagnostic imaging tests and rely on the medical history to determine the etiology and its clinical impact on the patient.

Case report

A 56-year-old male patient with a diagnosis of controlled systemic arterial hypertension (AH) and hypercholesterolemia. He presented an episode of chest pain of moderate intensity of 8 hours duration and progressive remission that did not receive medical attention, after which he was asymptomatic, functional class I. Six months later, in his routine evaluation, an echocardiogram was performed that reported a normal diameter of cardiac chambers, without alteration in global and segmental contractility, left ventricular ejection fraction was 62% and suggestive image of a calcificied thrombus of anteroseptal location in the middle third. Computed tomography (Figure 1) and cardiac magnetic resonance imaging (MRI) (Figure 2) were performed, both concluded that it was a septal transmural focal calcification in the middle third of the interventricular septum. The patient refused to perform coronary angiography. The MRI did not show data of recent or old myocardial infarction, the diameters of the cardiac cavities, without evidence of alterations in segmental mobility, the ejection fraction of both ventricles were normal. Renal function was normal, calcium, paratohormone and vitamin D values were normal. There was no history of neoplasms alteration in calcium homeostasis, generalized infectious or inflammatory processes, or congenital heart disease.

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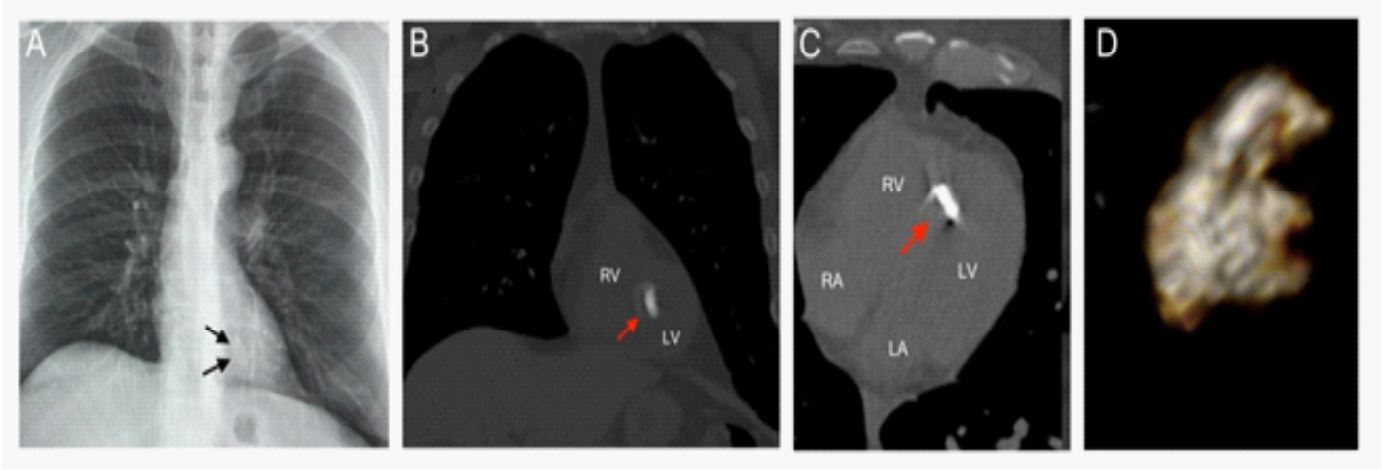

Figure 1: (A) Frontal chest radiograph, shows radiolucent image inside cardiac silhouette (black dates). (B) Simple CT cardiac, coronal section shows image compatible with calcification (red arrow). (C) View of 4 chambers, axial section was observed at the level of the interventricular septum in the middle third: hyperdense image (1300 UH) of 13 x4 mm, irregularly shaped (D) as clearly seen in volumetric reconstruction.
RV: right ventricle, LV: left ventricle, RA: right atrium, LA: left atrium.

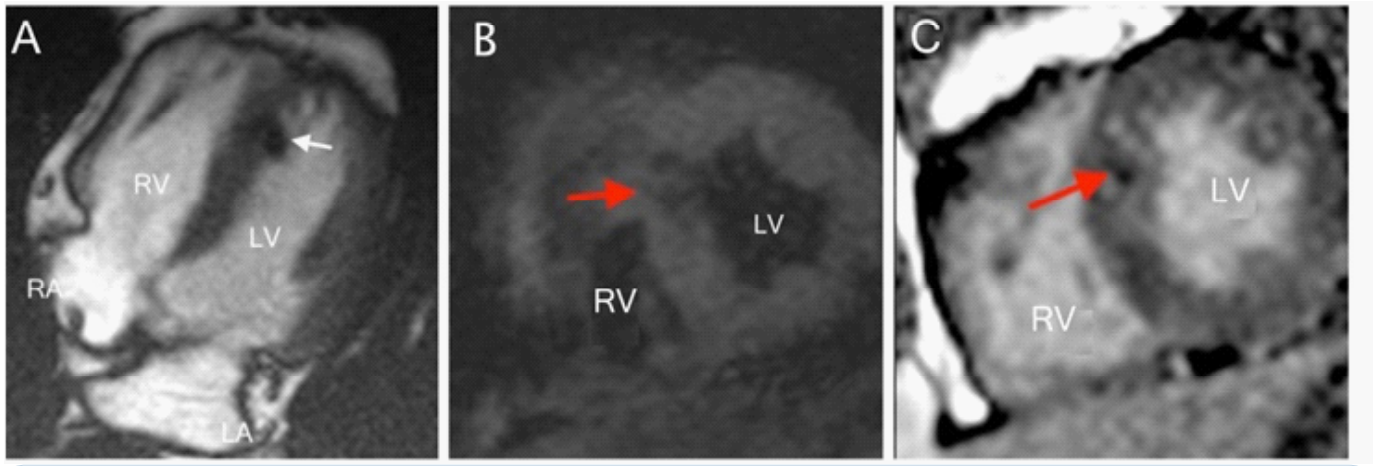

Figure 2: MRI. (A) on white blood sequence, seen from 4 chambers, focal hypointensity (calcification) is observed in the interventricular septum (white arrow) that does not limit its mobility. (B) T2 star sequence shows anteroseptal hypointensity (red arrow) was observed in the middle third (in T2 * from the shortest echo time). (C) Contrast-Enhanced Inversion Recovery GRE, shows hypointense image in the same location as B (red arrow).
RV: right ventricle, LV: left ventricle, RA: right atrium, LA: left atrium.

Discussion

Myocardial calcifications can be divided by their mechanism of production into dystrophic and metastatic; the dystrophic are the most frequent; The most common etiology is myocardial infarction, there are others such as traumatic, infectious or inflammatory and neoplastic. Calcium deposition is accentuated by the local microenvironment induced by ischemia, it has been reported that 8% of patients who suffered from myocardial infarction after 6 years, have dystrophic calcifications [1,2]; It has been proposed that its formation would be initiated by damage to the myocyte membrane, leading to the concentration of calcium ions, which with phosphatases would form microcrystals of calcium phosphates, which would progressively produce intracellular, extracellular deposits or both. On the other hand, metastatic calcifications have as their etiology chronic renal failure and calcium metabolism defects, presenting a more widespread and diffuse distribution, unlike the dystrophic ones that are more localized and linear, as in this case [2-4]. More diffuse forms of intramyocardial calcification have been reported in patients with sepsis and aggressive myopericarditis (ie, secondary to H1N1 virus), tuberculosis rare cardiac tumors (ie, rhabdomyomas and endothelialomas), endomyocardial fibrosis, and in patients with myocardial abscesses [4,5]. Where advanced calcification exists the commonest clinical manifestation is that of heart failure secondary to restrictive physiology [4,6].

Our patient has risk factors (AH and hypercholesterolemia) for ischemic heart disease and presented an episode of chest pain that could be of ischemic etiology, due to the clinical history and morphological characteristics of his injury we propose that it is a dystrophic calcification, secondary to possible old focal myocardial damage, due to obstruction of a small distal branch of the anterior descending coronary artery, which is very rare. If so, the location of this lesion in the septum is unusual, because the apex of the left ventricle is the most frequent site [6-9]. Furthermore, it has been described that calcification can appear six years after an episode of acute myocardial infarction. [6-9]. This case according to the classification [1] would belong to the group of “idiopathic”, in which the prevalence, etiology and mechanisms of calcification are unknown. The published data is largely limited to case reports that contain variable clinical, historical and histopathological information, probably these actually represent dystrophic or metastatic calcifications secondary to a clinically hidden or remote pathological process [1,10].

The development of CT significantly added to sensitivity to the presence of cardiac calcification. In addition, it readily allows etiologic differentiation based on location and character of the calcium deposits. Although limited by long acquisition time, conventional CT became the gold standard for detection of coronary arterial and myocardial, pericardial, valvular, and intracavitary calcium [2]. Calcified tissue generates no or a weak CMR signal and appears dark on all sequences, including LGE MRI. However, with acute myocardial calcification, the calcified myocardium is composed of necrotic myocardium with calcium deposits both within the myocardium and in the interstitial space, leading to hyperenhancement that mimics fibrosis on LGE [11].

Conclusion

Recognize and distinguish the different types of cardiac calcifications and related structures, they will guide the attention of each patient since each one has different etiology, treatment and result, as in this case the calcification does not affect the patient clinically.

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