#### CASE IMAGE

## Echocardiography WII

# Extensive intra-myocardial calcifications: Value of multimodality imaging

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#### Abstract

**Background:** Massive myocardial calcification is a very rare finding.

**Introduction:** Accurate identification and characteriation may help the clinicians to determine the etiology and clinical significance.

**Results:** In this case, the diagnostic pathway excluded previous myocardial infarction, myocarditis, and calcium-phosphate disorders. A possible dystrophic etiology was considered.

**Discussion:** There are no standardized imaging features available to classify specific subtypes of intra-myocardial calcifications. The relative merits of computed tomography and cardiac magnetic resonance (CMR) in providing complimentary diagnostic information in the evaluation of calcific myocardial lesions are shown.

**Conclusion:** Knowledge of the potential etiology and their imging patterns are important to provide a concise and accurate differential diagnosis.

KEYWORDS cardiac CT, cardiac MRI, intra-myocardial calcifications

### 1 | INTRODUCTION

An 81-year-old woman, affected by Horton disease and with previous history of rheumatic fever, was hospitalized for acute chest pain. Physical examination and EKG were not significant. Chest X-ray showed a diffuse hypodense lobulated area in the left ventricle (Figure 1A). The echocardiogram revealed diffuse aortic and mitral calcifications with mild stenosis, marked septum, and antero-lateral asymmetric hypertrophy, with extensive antero-lateral calcifications (Figure 1B, Video 1). Non-contrast computed-tomography (CT) highlighted widespread amorphous confluent calcifications in the left ventricular wall, with septal sparing, extended to the mitral-aortic annulus and

both coronary arteries (Figure 1C-C1 showing VRT reconstruction and C2 MPR axial-view, Video 2). Cardiac magnetic resonance (CMR) 3,0 T presented the following: septal hypertrophy, antero-lateral wall thickening with areas of intra-myocardial signal-alteration, surrounded by normal myocardium matching the CT calcifications. Areas of lowsignal were shown in multiple sequences: SSFP-cine in 4-chambers (4C) (Figure 2D, Video 3) and in short-axis (SAX) (Figure 2D1); T1weighted-spin-echo in 4C (Figure 2E) and SAX (Figure 2E1); STIR in 4C (Figure 2F). T1-native-mapping in 4C focused on diffuse septum and lateral fibrosis with low-signal (550 ms, normal range: 1150–1250 ms) compatible with calcifications (Figure 2G). After contrast-gadolinium injection, PSIR-sequence showed intra-myocardial hypersignal in

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# **Echocardiography**



**FIGURE 1** (A) Chest X-ray showing a diffuse hypodense lobulated area in the left ventricle. (B) Video 1. 2D-Echocardiogram revealing diffuse aortic and mitral calcifications with mild stenosis, marked septum and antero-lateral asymmetric hypertrophy, with extensive antero-lateral calcifications. (C) C1: CT-VRT reconstruction of the left ventricle, respectively axial view and focus on the coronal view. C2: Video 2. Non-contrast CT MPR-axial view presents widespread calcifications in the left ventricular wall extended to the mitral-aortic annulus and both coronary arteries



**VIDEO 1** CMR SSFP-cine 4-chambers and short-axis views respectively showing lateral and antero-lateral wall thickening with areas of intra-myocardial low signal, surrounded by normal myocardium

the lateral wall both in 4C and SAX, Figure 2H-H1, respectively). Coronary angiography documented significant disease that required revascularization. Extensive intra-myocardial calcifications are



**VIDEO 2** Non-contrast CT MPR-axial view presents widespread calcifications in the left ventricular wall extended to the mitral-aortic annulus and both coronary arteries

extremely rare.<sup>1</sup> In our case the diagnostic pathway excluded previous myocardial infarction, myocarditis and calcium-phosphate disorders. A dystrophic etiology has been supposed. The pathological mechanism for dystrophic calcifications is related to calcium deposition in any dead and dying myocardial tissues. Methastatic calcification are



**FIGURE 2** (D) D1: Video 3. CMR SSFP-cine 4-chambers and short-axis views respectively showing lateral and antero-lateral wall thickening with areas of intra-myocardial low signal, surrounded by normal myocardium. (E) E1: CMR T1weighted-spin-echo 4-chambers and short-axis view respectively show thickening of the lateral and antero-lateral wall with areas of low-signal. (F) CMR STIR 4-chambers showing lateral thickening with areas of low-signal. (G) CMR T1-native-mapping demonstrate diffuse septum and lateral fibrosis with low-signal in correspondence of the calcifications in the lateral wall (gray-light blue areas). (H) H1: CMR PSIR-sequences in 4-chamber view and short-axis view, respectively. After contrast-gadolinium injection, intra-myocardial hypersignal (gadolinium enhancement) is shown in the antero-lateral wall



**VIDEO 3** 2D-Echocardiogram revealing diffuse aortic and mitral calcifications with mild stenosis, marked septum and antero-lateral asymmetric hypertrophy, with extensive antero-lateral calcifications

otherwise related to any disturbance in calcium metabolism and can occur with abnormal calcium-phosphorous homeostasis. There are no standardized imaging features available to classify specific subtypes of intra-myocardial calcifications.<sup>2</sup> CT scan is the gold standard examination for the noninvasive detection of myocardial calcifications. CMR with late- gadolinium enhancement and native T1/T2 mapping has the unique ability to provide a differential diagnosis in the whole spectrum of myocardial diseases, allowing tissue characterization and hemodynamic assessment. The combination of imaging pattern and potential etiology plays a key role in characterizing calcifications and deriving its clinical impact.

#### CONFLICT OF INTEREST

No conflict of interests exists.

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