

Located ST-Segment Elevation as Atypical Presentation of Acute Perimycocarditis

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ABSTRACT

We report the case of a young patient admitted to the emergency room with precordial pain. Admission electrocardiogram identified ST-segment elevation located on the side wall associated with “mirror” image, with highly elevated cardiac enzymes, suggesting a diagnosis of coronary syndrome with ST-segment elevation. Echocardiogram revealed pericardial effusion with preserved ejection fraction and absence of segmental abnormalities, thus suggesting acute pericarditis with myocardial involvement. Cardiac magnetic resonance was performed, showing non-ischemic late enhancement, confirming the diagnosis of perimycocarditis. This is an uncommon situation in clinical practice and deserves greater understanding and attention by the doctors working in emergency rooms.

Introduction

Acute pericarditis is an inflammatory process of the pericardium, of diverse etiologies, infectious and noninfectious, of which 80 to 90% are idiopathic due to viral cause. In the emergency room, 5% of patients with chest pain, in whom Acute Coronary Syndrome (ACS) had been ruled out, and 1% with ST-segment elevation, had acute pericarditis.^{1,2}

Electrocardiogram (ECG) is the main exam in the diagnosis of acute pericarditis. PR-segment depression, often overlooked, may be the only finding and, although less sensitive, is highly specific in acute pericarditis. However, the classic finding is the presence of ST-segment elevation, diffuse and upward concave, and involving several leads that differentiate from transmural myocardial ischemia.^{3,4}

Pericarditis does not always result in typical ECG abnormalities. ST-segment elevation may involve fewer leads, and the location on specific walls of the heart makes it difficult to distinguish between ACS and ST-segment elevation.

Moreover, impairment of the pericardium can be extended to the cardiac muscle, resulting in elevation of cardiac enzymes and determining perimycocarditis. Echocardiogram with presence of pericardial effusion and absence of segmental alterations, associated with clinical history and ECG, confirms the diagnosis. Cardiac Magnetic Resonance

(MRI) should be requested in the event of diagnostic doubt and prognostic evaluation, when possible, identifying the signs of perimycocardial inflammatory injury using the late enhancement technique.

This is the case of a young patient admitted to the emergency room with sudden precordial pain and ECG highly suggestive of ACS with localized ST-segment elevation.

Clinical case

A 16-year-old male patient was admitted to the emergency room with complaints of precordial pain of sudden onset 20 minutes before while he was at rest (attending a class at school). Tight pain, with retrosternal location, of 7/10 intensity, with no irradiation and no factors of improvement or worsening. He denied previous pathology or use of illicit drugs. He reported fever (38.1 °C) the day before and diffuse myalgia, no diarrhea or flu symptoms, but physical examination was normal, with no murmurs or pericardial friction.

ECG on admission showed sinus rhythm and ST-segment elevation in the DI, AvL, V4, V5 and V6 leads, with depression, and “mirror” imaging in leads V1, V2 and DIII. The patient received double platelet antiaggregation and enoxaparin, in addition to sublingual nitrate. After nitrate use, the patient presented decreased ST-segment elevation (Figure 1) and reduced pain, with persistent lower intensity residual, continuous pain irradiating to the back.

Myocardial necrosis markers were very high with TnI-Ultra troponin above 50 ng/dL and Creatine Kinase MB (CK-MB) isoenzyme 90.3 U/L (reference values <0.04 and <4.0, respectively). Echo Doppler echocardiogram revealed pericardial thickening and Left Ventricular Ejection Fraction (LVEF) of 50%, with no segmental abnormalities.

In the absence of segmental abnormalities on echocardiogram associated with pericardial effusion, the hypothesis of acute pericarditis impairing the cardiac muscle - perimycocarditis - was suggested.

Anti-inflammatory and colchicine were administered to relieve the symptoms of pain and to reduce recurrences, respectively; as no left ventricular dysfunction was present, no antiremodeling (Angiotensin Converting Enzyme Inhibitors - ACEI or beta blockers) were prescribed.

On the fourth day of admission, CMRI showed a 60% LVEF, minimal pericardial effusion with edema and late enhancement of a non-ischemic pattern, not respecting the coronary, multifocal, middle anterolateral mesoepicardial and apical lateral territory compatible with inflammatory cardiac muscle involvement - myocarditis (Figure 2). It evolved favorably with pain reduction, and the patient was discharged 48 hours, with outpatient follow-up.

Keywords

Myocarditis; Myocardial Infarction; Electrocardiography.

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Case Report

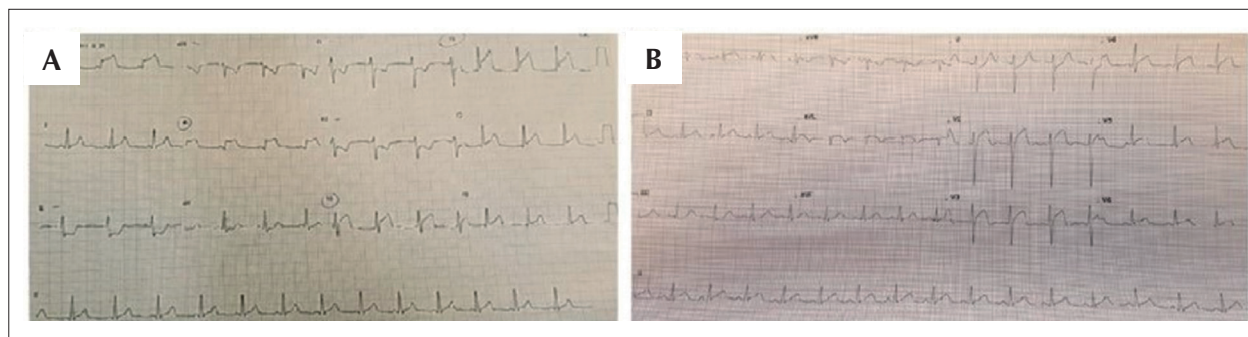


Figure 1 – Electrocardiogram on admission (A) and post-nitrate (B).



Figure 2 – Cardiovascular magnetic resonance imaging with late enhancement of non-ischemic mesoepicardial pattern: (A) 4 chambers; (B) short axis.

Discussion

We report the case of a young patient, with no risk factors for CAD, with perimyocarditis, which is defined as acute inflammation of the pericardium associated with cardiac muscle involvement. Acute pericarditis is an inflammation of the pericardial sac with or without associated pleural effusion, accounting for 5% of the cases of chest pain in the emergency department.⁵ The main etiology is idiopathic/viral in 80 to 90% of the cases in developed countries; the others are bacterial causes (tuberculosis being the most common), autoimmune, neoplastic (mainly breast cancer and lymphoma) or metabolic diseases (uremia and myxedema).⁶

Diagnosis is based on four criteria, two of which are mandatory: (1) chest pain, typically pleuritic, worsening in dorsal decubitus and improving when the trunk is tilted forward; (2) auscultation of pericardial friction (<33% of cases); (3) ECG findings, such as diffuse ST-segment elevation and PR-segment depression; (4) pericardial effusion finding better viewed on echocardiogram.⁷

Perimyocarditis in the acute form, especially in emergency rooms, may involve similar findings to those of ACS such as chest pain similar to anginal pain, abnormalities found on ECG and elevation of myocardial necrosis markers.⁸

The electrocardiographic abnormalities observed in pericarditis are of fundamental importance in diagnosis and depend on the evolutionary phase of myocardial inflammatory aggression: in the early phases (hours and days). The onset of concave and diffuse ST-segment elevation is common (except in AvR and V1, in which there is depression), without respecting the territory of the coronary anatomy, with apical T wave and slight increase in amplitude.^{9,10} Another characteristic strongly associated with pericarditis is PR-segment depression, better viewed in leads V5 and V6.¹¹

Perimyocarditis is defined in the presence of elevation of myocardial necrosis markers, without ventricular dysfunction (pericardial and myocardial involvement).⁷ CMRI is a useful methodology for the study of pericardial diseases, allowing the identification of effusion and pericardial thickening with higher accuracy than echocardiography.³ Sometimes, when differential diagnosis of ACS cannot be ruled out or confirmed, CMRI becomes essential in the evaluation of the late enhancement/fibrosis site, which allows the differentiation between areas of myocardial damage by coronary thrombosis or vasospasms and signs suggestive of inflammatory myocardial injury of perimyocarditis.

Coronary tomography angiography can also be used in the differential diagnosis of ACS with high negative predictive value in ruling out coronary disease.³

Patients diagnosed and with no risk factors (fever, pleural effusion > 20 mm, cardiac tamponade, subacute onset, specific etiology or no response to initial treatment in the first 7 days) do not require hospital admission.⁷

Initial treatment is with non-steroidal anti-inflammatory drugs (NSAIDs), preferably ibuprofen 600 mg every 8 hours or indomethacin 25 to 50 mg every 8 hours or acetylsalicylic acid 750 to 1,000 mg every 8 hours for 1 to 2 weeks. In these cases, always associate proton-pump inhibitor. Colchicine should be added and maintained for 3 months at a dose of 0.5 mg twice daily (if weighed >70 kg) or once daily (if weighing <70 kg), with lower disease recurrence rates.^{3,12}

The use of corticosteroids should be considered as second-line treatment in patients with contraindications or who have failed with NSAIDs or acetylsalicylic acid. The dose of prednisone is 0.2 mg/kg for 6 months and gradual weaning of the medication.³

Prognosis of the disease is good, and cardiac tamponade rarely occurs, being more common in cases with well-established etiology.¹²

Authors' contributions

Research creation and design: Silva PVP, Antunes MO; Data acquisition: Silva PVP, Pereira MP; Data analysis and interpretation: Silva PVP, Antunes MO, Torres ALN; Statistical analysis: Silva PVP, Antunes MO; Fund-raising: Silva PVP; Manuscript writing: Silva PVP, Torres ALN; Critical revision of the manuscript as for important intellectual content: Antunes MO, Soeiro AM, Oliveira MT.

Potential Conflicts of Interest:

There are no relevant conflicts of interest.

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