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CASE IMAGE

Sudden death due to fulminant lymphocytic myocarditis with atypical prodromal symptoms

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Abstract

Diagnosis of myocarditis is complex because it is not always preceded by clear symptoms. We report the case of a woman who died suddenly. Autopsy and histopathological investigations revealed a lymphocytic myocarditis. Clinical history showed the patient went to the hospital some days before for joint pain and low-grade fever.

KEYWORDS

autopsy, myocarditis, sudden death

1 | CASE REPORT

We report the case of a 70-year-old woman suffering from chronic liver disease (eradicated HCV), previous pneumonia with pleural effusions, and chronic cerebral ischemia. The past cardiological history was negative and the previous ECG showed sinus rhythm while the echocardiogram proved only low E/A (0.62) with minimal tricuspid regurgitation. The patient went to the hospital for intense inflammatory pain in hips, not associated with shoulder pain, and a slight low-grade fever (37°C), without evident cardiovascular symptoms or signs. Since septic arthritis of the hip was suspected, X-ray was performed showing only coxarthrosis, while procalcitonin levels and white blood cell count (9.66×10^9/L) resulted within normal limits. The molecular swab for SARS-CoV-2 was negative. For this reason, the patient was discharged with a diagnosis of coxarthrosis. About 10 days after this episode, she was transferred to hospital for an episode of dyspnea with severe desaturation (78% in ambient air) and wide QRS monomorphic ventricular tachycardia (170 beats/min) that was treated with amiodarone and a single 200 J direct current shock (Figure 1).[1] A cardiologist was involved, and echocardiograms were repeatedly performed showing a very severe reduction of the ejection fraction of the left ventricle (from 5% to 20%) with pericardial effusion; no right ventricular dysfunction or valvulopathy were found. Laboratory tests showed increase of markers of myocardial damage including troponin T hs (9800 ng/ml), myoglobin (467 ng/ml), CPK-MB (62.50 ng/ml), LDH (718 U/L), CK-MB (124 U/L). Electrolytes showed only low sodium levels (133 mEq/L), while calcium and potassium were

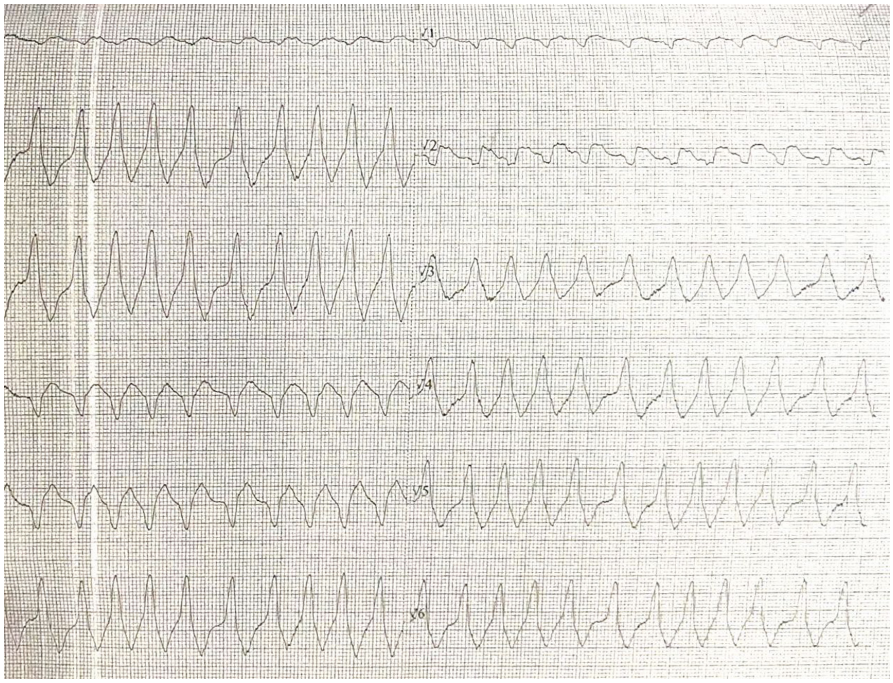

FIGURE 1 Electrocardiographic trace of the arrhythmia (on the left from the top to the bottom leads I-II-III- aVR-aVL-aVF; on the right from the top to the bottom leads from V1 to V6)

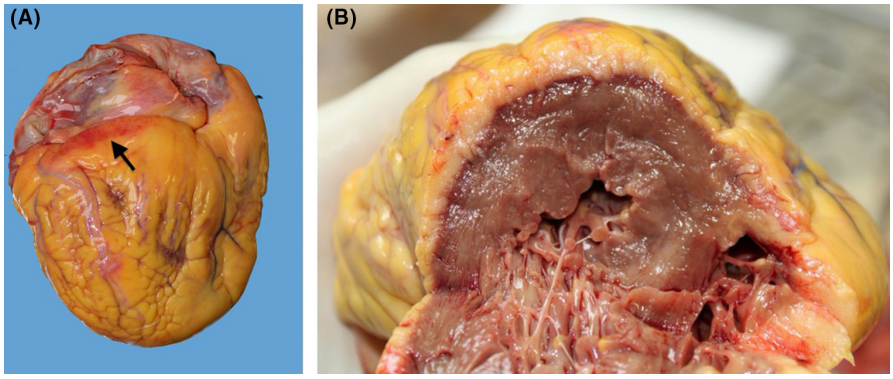

FIGURE 2 Panel (A) Areas of suffusion on the atrioventricular line; Panel (B) Myocardial wall paleening with whitish dyschromia at the left ventricular section and areas of adiposities

in the reference range. Within an hour from the onset of symptoms, the patient developed mottled skin, cyanosis, hemodynamic instability with episodes of ventricular tachycardia needing repeated defibrillation. Total body CT was also performed, in the suspect of a pulmonary embolism, and it revealed cardiomegaly with stagnation of contrast medium in the right heart chambers, bilateral pulmonary areas of consolidation, pericardial effusion; no hilar or interstitial congestion were found. After cardioversion, the patient had cardiac asystole, for which she died despite ACLS maneuvers. Autopsy showed cardiomegaly with massive myocardial fat, most likely related to the patient's overweight, pale color, whitish discoloration of the ventricular wall and adiposities spread over the papillary muscles (Figure 2). Microscopic histological examination revealed widespread biventricular lymphocytic inflammatory infiltrate with necrotic areas in the right ventricle, compatible with viral myocarditis (Figures 3–4). Even if no further analysis were performed on biopsy, differential diagnosis was performed. An acute myocardial infarction was excluded, both clinically and histopathologically, due to the integrity of coronary circulation. Also, an isolated right ventricle failure was excluded due to the cardiac biventricular involvement.

2 | DISCUSSION

Fulminant myocarditis is an acute form of severe cardiac inflammation, most often of viral etiology, characterized by hemodynamic instability requiring immediate support.[2] Symptoms include fatal ventricular arrhythmias needing defibrillation and assisted respiratory ventilation.[3,4] Diagnosis includes echocardiography, magnetic resonance (MRI), cardiac catheterization and, in rare cases, endomyocardial biopsy which remains the *gold standard* for diagnosis.[5,6] Myocarditis remains a pathology burdened by high mortality, above all because it may be anticipated by different and often vague symptoms, such as to make its onset unsuspected.[4,5]

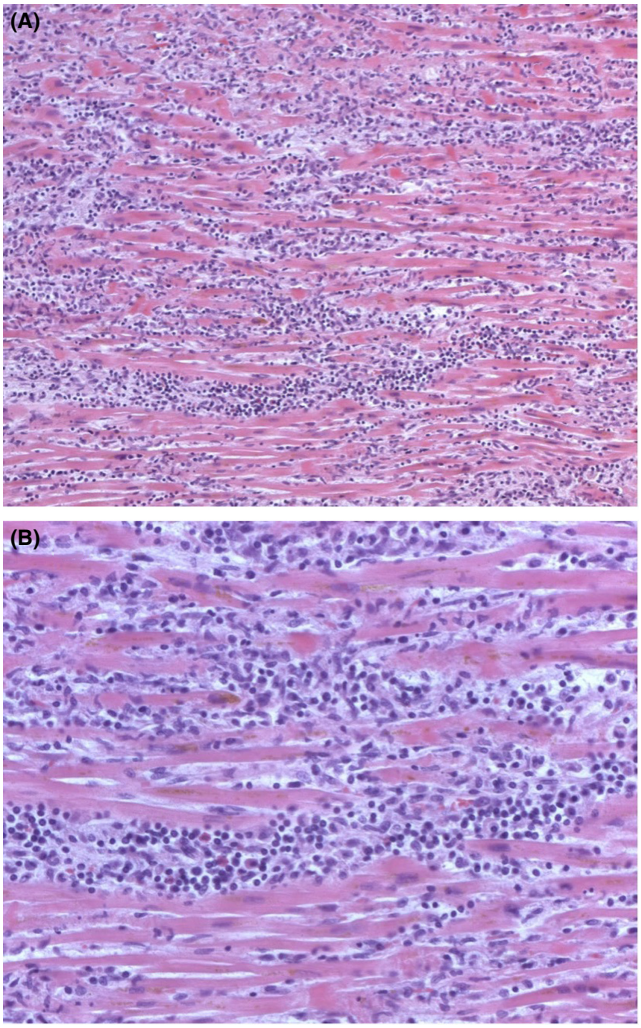

FIGURE 3 Panel (A and B) Widespread inflammatory lymphocytic infiltrate in myocardial tissue

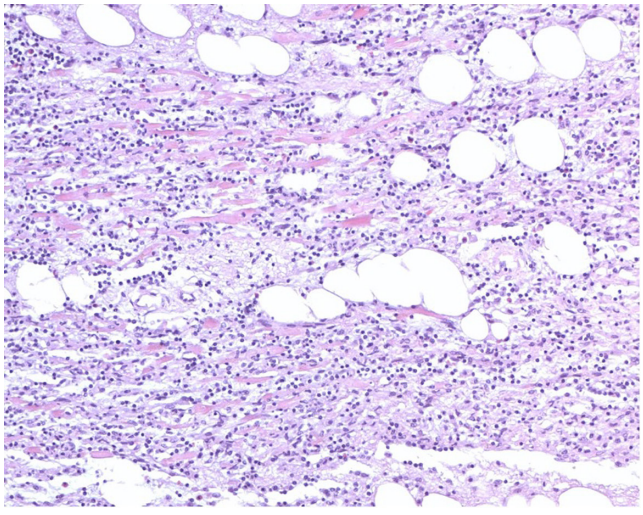

FIGURE 4 Evidence of necrotic areas in the right ventricle

The patient of reported case presented a vague previous symptomatology characterized by accentuated joint pain with low-grade fever so we can hypothesize that these symptoms could be prodromal with respect to the acute event. The case demonstrates the difficulty in formulating the diagnostic suspicion of myocarditis, especially when it is preceded by vague and flu-like prodromal symptoms. For this reason, it is essential to pay close attention also to symptoms that may apparently seem minor and of non-cardiac origin. Further research on atypical prodromal symptoms and potential clinical predictors is needed to improve the prompt recognition of myocarditis.

AUTHOR CONTRIBUTIONS

IA conceived the idea of the research. IA and MAS managed the organization and drafting of the paper. LA, FC, FMG, EV and PR have contributed to analyze the data and write the paper.

ACKNOWLEDGEMENT

None.

CONFLICT OF INTEREST

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

DATA AVAILABILITY STATEMNENT

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

CONSENT

Written informed consent was obtained from the next of kin to use the data and publish this report.

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REFERENCES

1. Caforio AL, Pankuweit S, Arbustini E, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on myocardial and pericardial diseases. *Eur Heart J*. 2013;34(33):2636-2648, 2648a–2648d. doi:10.1093/eurheartj/eht210
2. Ammirati E, Veronese G, Cipriani M, et al. Acute and fulminant myocarditis: a pragmatic clinical approach to diagnosis and treatment. *Curr Cardiol Rep*. 2018;20(11):114. doi:10.1007/s11886-018-1054-z
3. Kociol RD, Cooper LT, Fang JC, et al. Recognition and initial management of fulminant myocarditis: a scientific

statement from the American Heart Association. *Circulation*. 2020;141(6):e69-e92. doi:10.1161/CIR.0000000000000745

4. Olejniczak M, Schwartz M, Webber E, Shaffer A, Perry TE. Viral myocarditis-incidence, diagnosis and management. *J Cardiothorac Vasc Anesth*. 2020;34(6):1591-1601. doi:10.1053/j.jvca.2019.12.052
5. Irabien-Ortiz Á, Carreras-Mora J, Sionis A, Pàmies J, Montiel J, Tauron M. Fulminant myocarditis due to COVID-19. *Rev Esp Cardiol (Engl ed)*. 2020;73(6):503-504. doi:10.1016/j.rec.2020.04.005
6. Aquila I, Ricci P, Galassi A, Sacco MA, Malfa O, Gratteri S. Sudden death and Takayasu's arteritis: the role of histopathological investigation. *Med Leg J*. 2019;87(3):139-143. doi:10.1177/0025817219862484

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