Fulminant lymphocytic myocarditis due to Parvovirus B19: a case report

Miocarditis linfocitaria fulminante por parvovirus B19: a propósito de un caso

We present the case of a 34-year-old man, with celiac disease, a smoker, regular alcohol user, and occasional consumer of cocaine, ecstasy, and hashish. He had not been vaccinated against COVID-19. He presented to the emergency department with a 5-day history of chest pain radiating to the epigastrium, vomiting, and fever of 38 °C. Additional tests revealed a newly developed complete left bundle branch block (Figure 1), marked elevation of troponins, and hypertransaminasemia. COVID-19 testing was negative.

Within the first hours of hospitalization, he developed hypotension, hypothermia, arrhythmic heart sounds, and complete atrioventricular block. Bedside echocardiography showed global contractility deficit and a left ventricular ejection fraction of 10%. Suspecting acute coronary syndrome, a coronary angiography was performed, which showed no coronary lesions but severe left ventricular dysfunction. Treatment with norepinephrine and dobutamine was initiated. He was admitted to the intensive care unit (ICU) in cardiogenic shock, with accelerated idioventricular rhythm, weak pulse, and hypothermia. No bite marks, erythema migrans, or other findings suggesting a possible zoonosis were observed. A marked hyperlactatemic metabolic acidosis was noted.

Thirty minutes after ICU admission, the patient developed sudden dyspnea and went into cardiac arrest due to ventricular fibrillation. Cardiopulmonary resuscitation was initiated, and after 20 minutes, transcatheter cannulation and connection to an extracorporeal membrane oxygenation (ECMO) system were performed. Milrinone was administered for associated right ventricular failure, and a massive transfusion protocol was initiated due to hemorrhagic complications due to liver failure and coagulopathy. The patient subsequently developed multiple organ failure with progressive lactate increase and renal failure. He entered refractory ventricular fibrillation despite amiodarone, lidocaine, and continuous defibrillation attempts, resulting in inevitable death.

An autopsy was requested due to the absence of a definitive diagnosis, which revealed genetic material from Parvovirus B19, confirming the diagnosis of fulminant myocarditis caused by this virus (Figure 2).

Myocarditis is an inflammatory disease of the myocardium with multiple etiologies. Its differential diagnosis can be challenging due to the heterogeneity of its clinical presentations.[1]

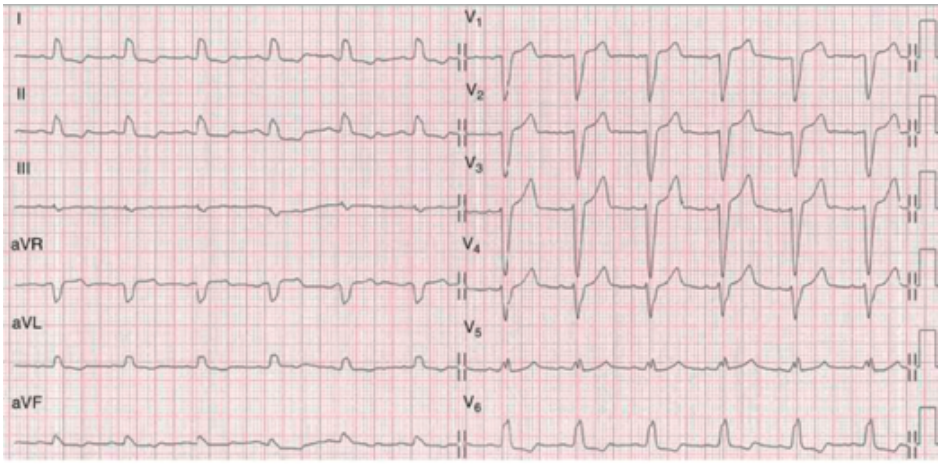

Figure 1. Patient's ECG showing complete infra-Hisian left bundle branch block.

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Parvovirus B19 is a single-stranded DNA virus belonging to the *Parvoviridae* family, consisting of a promoter sequence (p6) and open reading frames encoding the main nonstructural protein (NS1) and the viral capsid proteins (VP1/2). The latter is responsible for the production of eicosanoids, prostaglandins, and leukotrienes, which play an important role in inflammation and host cell dysfunction.[2] Parvovirus B19 has been associated with myocarditis, allograft rejection in children, and post-transplant myocarditis. However, this virus has also been found in the hearts of patients without evidence of cardiomyopathy and shows high variability in prevalence and viral load. Therefore, the clinical significance of Parvovirus B19 in the myocardium remains unclear and is still under discussion.[3]

The pathogenesis of viral myocarditis follows a 3-phase process, leading to cellular dysfunction and inflammation induced by the production of leukotrienes and prostaglandins, followed by intravascular accumulation, adhesion, and infiltration of inflammatory cells, resulting in endothelial dysfunction, impaired myocardial microcirculation, and ultimately, myocyte necrosis and dilated cardiomyopathy.[4]

Patients with myocarditis often present with mild, nonspecific systemic symptoms.[5,6] In our patient, clinical worsening manifested as a flu-like syndrome during the early inflammatory phase, with high levels of acute-phase reactants, rapid deterioration of cardiac function, elevated myocardial injury markers, and the electrocardiographic abnormalities observed upon admission.

The sudden appearance of a left bundle branch block not previously present led us to suspect an acute coronary syndrome; however, urgent coronary angiography ruled this out.

Bedside echocardiography was highly useful in our case for the early diagnosis of severe biventricular dysfunction, which allowed us to expedite ICU admission and the initiation of inotropic and vasopressor agents. Nonetheless, these measures were insufficient for hemodynamic support, and progressive deterioration ensued, culminating in cardiac arrest following degeneration of the accelerated idioventricular rhythm into ventricular fibrillation.

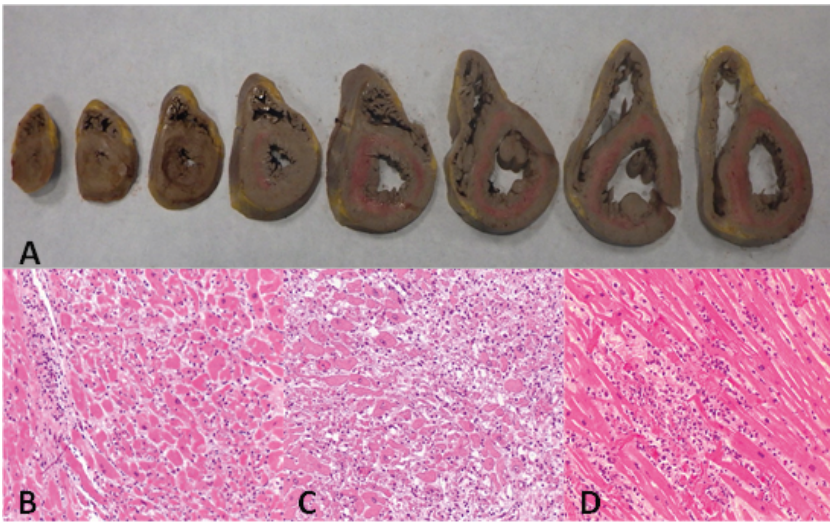

Figure 2. A. Macroscopic image: serial sections. B. Lymphocytic inflammatory infiltrate. C. Areas of myocardial necrosis with foci of hemorrhagic extravasation and cytopathic changes. D. Longitudinal section showing myocardial fiber necrosis.

The main objectives of hemodynamic support are to reduce biventricular afterload, improve systemic and coronary perfusion, and decrease preload and venous congestion.[7]

In our case, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) was initiated at the bedside, as refractory cardiopulmonary resuscitation precluded patient transfer to the operating room.

The most frequently used temporary mechanical support devices reported in registries include the intra-aortic balloon pump, Impella®, and VA-ECMO with peripheral cannulation. The latter provides rapid and complete cardiorespiratory support but may increase left ventricular afterload, sometimes requiring additional unloading strategies to prevent distension and pulmonary edema, such as the balloon pump or Impella®, as well as low-dose inotropes and vasodilators.[8,9]

Regarding etiologic treatment, several randomized clinical trials have evaluated corticosteroids and azathioprine, showing improvements in left ventricular ejection fraction, ventricular diameter, functional class, and survival[10]. Immunoglobulins have both proinflammatory effects (activation of immune cells and complement) and anti-inflammatory effects (toxin neutralization).

In our patient, corticosteroids and IV immunoglobulins were not considered due to the rapid progression. Management focused on hemodynamic support with vasoactive agents, cardiopulmonary resuscitation, and extracorporeal support.

Although rare in clinical practice, this case should be considered in the differential diagnosis of acute myocarditis, especially in young patients.[1,5,9]

Note of the editors: This is a BOWMAN-generated English translation of the officially indexed Spanish-language article, which should be cited as Rev Esp Urg Emerg. 2023;2:110-112. In this translated version, the editors have supervised the process; however, it cannot be ruled out that some errors resulting from the artificial intelligence translation process may have gone unnoticed.

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