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Case Report

Respiratory syncytial virus myopericarditis in an immunocompetent adolescent

Miopericardite por vírus sincicial respiratório em adolescente imunocompetente

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ABSTRACT

Respiratory syncytial virus infection is responsible for seasonal outbreaks, usually manifested with acute respiratory disease in childhood. Acute myocarditis is a rare and underdiagnosed complication of this infection. Myocarditis has a heterogeneous clinical presentation and is related to an increased risk of ventricular arrhythmias, sudden death, and chronic dilated cardiomyopathy. This paper is a case report of confirmed myocarditis associated to respiratory syncytial virus in an immunocompetent adolescent, not classified in typical risk groups. A review of the management of myocarditis is presented, with an emphasis on diagnosis, risk stratification, etiological assessment, and treatment options.

Keywords: Respiratory syncytial virus infections; Myocarditis; Pericarditis; Electrocardiography; Emergency service, hospital

RESUMO

A infecção pelo vírus sincicial respiratório é responsável por surtos sazonais, geralmente manifestados como doença respiratória aguda na infância. A miocardite aguda é uma complicação rara e subdiagnosticada dessa infecção. A apresentação clínica da miocardite é heterogênea e está relacionada a um aumento do risco de arritmias ventriculares, morte súbita e miocardiopatia dilatada crônica. Este artigo apresenta um relato de caso de miocardite confirmada associada ao vírus sincicial respiratório em um adolescente imunocompetente, que não pertence aos grupos de risco típicos. Adicionalmente, apresenta revisão completa sobre o manejo da miocardite, abordando aspectos como diagnóstico, estratificação de risco, avaliação etiológica e opções de tratamento.

Descritores: Infecções por vírus respiratório sincicial; Miocardite; Pericardite; Eletrocardiografia; Serviço hospitalar de emergência

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INTRODUCTION

Respiratory syncytial virus (RSV) infection is responsible for seasonal outbreaks two to three times a year worldwide. Humans are the only hosts, and the near disappearance of the virus between outbreaks is not well understood. The main manifestation of RSV is acute respiratory infection in individuals of all ages, most commonly in childhood. Almost all children will have contact with this virus by the age of 2 years old. There is no effective vaccine thus far, and reinfection is common. Approximately 1 to 2% of RSV infections require hospitalization, mainly due to lower respiratory tract involvement, such as bronchiolitis and pneumonia. Cardiovascular complications are rare but potentially life-threatening.[1]

There are few RSV-induced myocarditis case reports, especially in immunocompromised children.[2]

This report aimed presenting an uncommon case of RSV cardiovascular manifestation in an immunocompetent adolescent outside of the usual risk group for complications. The study and consent waiver were approved by the local Review Board (CAAE: 54477821.9.0000.0071).

CASE REPORT

A 16-year-old male presented to the Emergency Department with a few hours of pleuritic chest pain associated with sweating and severe malaise. The pain worsened while lying down and improved in orthostasis. Five days earlier, he had 3 straight days of fever and myalgia. The physical examination revealed a blood pressure of 92/60 mmHg, heart rate of 85 bpm, temperature of 38°C, regular general condition, diaphoresis, normal peripheral perfusion, a cardiac rub while in a sitting position with the chest flexed forward and no signs of pulmonary or systemic congestion. The admission electrocardiogram showed diffuse ST segment elevation and PR segment depression in DI and V6 (Figure 1). Laboratory tests confirmed myopericarditis with a high-sensitivity troponin I value of 1,023 pg/mL (normal range < 52 pg/mL), which remained on a plateau for the following days. The admission echocardiogram showed increased pericardial echogenicity, mildly decreased left ventricular function (ejection fraction of 50%), apical hypokinesia, and no signs of pericardial effusion. The patient underwent a reverse transcriptase polymerase chain reaction (RT-PCR) swab that was negative for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and a respiratory viral panel that identified RSV. He was admitted to a Cardiac Care Unit and was treated with ibuprofen and colchicine. The patient was discharged after 4 days with controlled symptoms, decreased troponin, and improved ventricular function.

DISCUSSION

Acute myocarditis is an inflammatory disease of the cardiac muscle. Viral myocarditis has an annual incidence estimated at 10 to 22 per 100,000 individuals. However, the heterogeneous clinical presentation and difficulty in establishing diagnosis

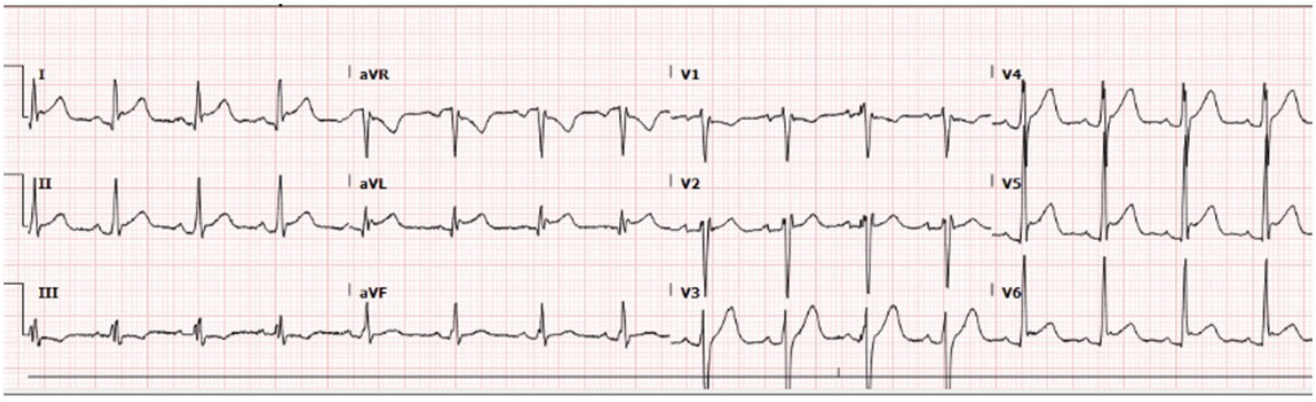

Figure 1. Admission electrocardiogram. Diffuse ST segment elevation and PR segment depression.

possibly lead to underestimated rates.[3] Over the past 30 years, the prevalence of myocarditis and its related mortality have increased; however, the age-standardized rate of death has decreased.[4]

The causes of myocarditis can be divided into two groups: noninfectious and infectious. Noninfectious myocarditis causes include hypersensitivity reactions (following recent exposure to vaccines, medications, and insect bites) and muscle aggression secondary to toxins (i.e., recreational drugs, catecholamines, chemotherapy agents, and radiation). Among infectious causes, viruses are the most common, followed by bacteria and fungi.[5] Several RNA and DNA viruses have been considered etiologies of acute myocarditis, including coxsackie B virus, adenovirus, parvovirus B-19, human herpesvirus 6, human immunodeficiency virus, cytomegalovirus, varicella, SARS-CoV-2 and, rarely, paramyxoviruses such as RSV.[5-7] Only 4% of patients with myocarditis are diagnosed using serology assays. Most patients do not show warning signs and have a good prognosis, which makes this investigation unnecessary.[5]

In this report, the diagnosis of RSV-induced myopericarditis was the most likely. Over the past decades, RSV infection has progressively increased in older patients because of nonstandardized surveillance and a lack of prophylaxis strategies, antiviral treatment, and vaccines.[8]

Clinical manifestations of myocarditis may range from asymptomatic or subclinical disease to heart failure, arrhythmia, and sudden death. In viral myocarditis, patients may present with fever and myalgia, but usually no prodromal symptoms are present.[5]

The patient described above had the most typical epidemiological profile related to myopericarditis: a young male with fever and chest pain. The electrocardiogram was also typical, with diffuse ST-segment elevation and PR depression. There was a plateau elevation of troponin and abnormal echocardiogram findings. Fortunately, the patient showed no signs of a poor prognosis, such as severe left ventricular dilatation or dysfunction, severe dyspnea, or cardiogenic shock.[9] In this case, the treatment was based only on supportive measures.

Endomyocardial biopsy is the gold standard for the diagnosis of myocarditis; it is potentially useful in patients presenting suspected fulminant myocarditis or acute myocarditis with acute heart failure, left ventricular dysfunction, and/or rhythm disorders. Biopsy can also be performed in selected cases of suspected myocarditis in hemodynamically stable patients. The results may guide specific therapy with immunosuppression and immunomodulation.[10]

Cardiac magnetic resonance imaging is a very useful noninvasive strategy for diagnosing acute myocarditis. Cardiac magnetic resonance improves the possibility of finding a differential diagnosis, allows follow-up of treatment, and can be used to guide biopsies and stratify the prognosis. Limitations of the method include the low possibility of identifying the cause of myocarditis and the absence of pathognomonic findings compatible with the disease, although the accuracy of cardiac magnetic resonance can be greater than 90%. A decreased left ventricular ejection fraction and late gadolinium enhancement in myocarditis are predictors of major cardiovascular events.[6]

In this case, after the confirmation of low-risk myopericarditis together with the patient's clinical improvement, cardiac magnetic resonance, endomyocardial biopsy and specific immunotherapy became unnecessary. In conclusion, the use of complementary tests for the diagnosis of myocarditis and the investigation of etiology to direct specific treatments should be based on the clinical symptomatology and the severity of the patient.

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