Cytomegalovirus (CMV) and Acute Myocarditis in an Immunocompetent Patient

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Abstract

Acute viral infections can lead to heart inflammation, including acute myocarditis. We report a rare case of myopericarditis in a young immunocompetent adult, in a context of recent Cytomegalovirus (CMV) infection. The clinical presentation was an influenza-like syndrome, classical for a CMV infection, without any chest pain or dyspnea, but a systematic exploration showed multiple inflammation-compatible myopericardial images on MNR-scan. The diagnosis of asymptomatic myopericarditis was established. We present the MNR-scan findings and discuss the CMV cardiac effects and systematic cardiac MRI interest in viral infection.

Key words: CMV, acute myocarditis, myopericarditis, MRI

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Introduction

Acute viral infections can lead to heart inflammation, resulting in acute myocarditis. Cytomegalovirus (CMV) has been rarely reported in this context.

Here, we describe for the first time asymptomatic acute myopericarditis in a young immunocompetent patient, and present the MRI findings.

Case Report

A 35-year-old man had been admitted for acute sudden and severe headaches, preventing him from sleeping normally for seven days. He had been suffering at the same time from a viral syndrome including odynophagia, polymyalgia, and asthenia, without fever. He had no chest pain, nor dyspnea. He had no previous medical history, and took no medications.

On admission, the patient was hemodynamically stable (blood pressure 120/60 mmHg, pulse 88 bpm, temperature 38.2°C). Physical examination did not reveal evidence of meningial syndrome, and the cardiac examination was normal (particularly no pericardial rub). There was neither hepatomegaly nor splenomegaly, and no lymph nodes were palpable.

A chest X-ray and ECG were normal.

Hemoglobin level was normal (12.7 g/dL), as well as platelets (279,000/mm³, normal between 150,000 to 500,000/mm³). Inflammation syndrome was evident: polynucleosis (9,200 cells/mm³) and elevated CRP (initial CRP=157.8 mg/L, n<5). White cell count was 11,600/mm³, without any monocytosis (684/mm³, normal between 100 to 1,000/mm³) or lymphocytosis (1,531/mm³, normal 1,000 to 4,000/mm³). Liver and cardiac enzymes were not increased (troponin I 0.02 μg/L, myoglobin 19.8 μg/L, CPK 56 UI/L). BNP was normal (48 ng/L, n<100). Blood cultures were all negative.

Results

Cerebral MR-scan and lumbar puncture to explore the reason for headaches with inflammatory syndrome were normal. CRP remained very high for several days (around 260 mg/L) leading us to perform total-body CT-scan, which was normal, and trans-thoracic echocardiography, to search for infectious endocarditis.

Trans-thoracic echocardiography evaluation suggested myocarditis, based on the non-specific left ventricle segmen-
Figure 1. MR imaging findings. Horizontal long axis True-FISP performed before (A) and after (B) gadolinium infusion demonstrated a diffuse ill-defined subepicardial enhancement in the lateral free-wall of the left ventricle (arrows). This pattern was also demonstrated on short-axis (C) and horizontal long-axis (D) delayed-enhancement imaging. These findings were concordant with the diagnosis of myocarditis.

tal dysfunction, encompassing coronary artery territories. Left ventricular ejection fraction was 50% (Simpson), telediastole left ventricular diameter was enlarged (measured 59 mm, normal<55 mm). No valvulopathy was noticed. No vegetation was found.

Cardiac MR-scan was performed using a 1.5T imager (Avanto, Siemens, Erlangen, Germany). The horizontal long axis, vertical long axis and short axis planes were obtained. The MR protocol included T2 STIR, cine True-FISP and delayed-enhancement imaging sequences. The latter sequences were obtained after infusion of 0.2 mM/kg of gadolinium (Prohance, gadoteridol, Bracco Diagnostics). Cardiac function was abnormal, with lateral hypokinesia and a left ventricular ejection fraction estimated to 35%. Delayed-enhancement sequences demonstrated a diffuse ill-defined subepicardial enhancement in the lateral free-wall of the left ventricle (Fig. 1). These findings were concordant with the diagnosis of myocarditis.

Most of the etiologic analyses remained negative. In particular the following biological tests were normal: serologies for EBV, parvovirus B19, HBV, HCV, HIV₁,₂, HTLV₁,₂; nasopharyngeal secretions examinations (influenzae, parainfluenzae, adenovirus), bacterial serologies (bartonella, borrelia, chlamidia coxiella, mycoplasma, rickettsia). TSH was normal. Serological evaluations remained normal, except for CMV; immunofluorescence tests demonstrated a recent infection (IgG negative, IgM positive).

The patient was discharged a few days later, under the following treatment: colchicin 0.5 mg, bisoprolol 2.5 mg, ramipril 5 mg per day. Two weeks later, he felt very well, without any symptoms: biological inflammatory syndrome was resolved.

Discussion

Acute cytomegalovirus (CMV) infection in immunocompetent patients is common worldwide, with seroprevalence rates of 40%-100%, depending on the country, socioeconomic conditions, and the patient’s age.

In immunocompetent patients, CMV infections are most often asymptomatic or inducing a mononucleosis syndrome, but rarely can lead to severe organ complications. Both
EBV (1) and CMV (2) induce exceptional cardiac symptoms, especially in immunocompetent patients. Zubiaurre et al (2) reported a case of simultaneous hepatic and myopericardic CMV infection in a 36-year old immunocompetent man. The presentation of pericarditis and myocarditis associated with acute CMV infections is primarily chest pain with ECG abnormalities, and cardiac enzymes elevation (2). CMV myopericarditis can lead to dilated cardiomyopathies (4): systematic intramyocardial viral genome quantification (4) in “idiopathic” dilated cardiomyopathy (246 patients) have shown multiple viral infections in the myocardium of adults with “idiopathic” left ventricular dysfunction: viruses such as parvovirus B19 (51.4%), or HHV-6 (21.6%). By contrast, CMV genomic expression was found in only 2/246 (0.8%). Another viral study in acute myocarditis revealed about 7.5% CMV prevalence (5).

In immunodepressed patients, CMV infection can lead to myocarditis, especially in transplant and HIV patients (6). Cytomegalovirus (CMV)-associated carditis in the immunosuppressed patient carries a 60% mortality.

As far as we know, the present case may be the first reported acute asymptomatic myocarditis linked to a recent CMV infection, suspected by performing systematic trans-thoracic echocardiography leading to cardiac MRI, with consistent MRI images. Diagnosis of CMV acute infection relied on positive serology for CMV IgM antibodies and negative serologies for other viruses. This case underlines the interest of MR-scan to diagnose myocarditis (11), and specify local involvement and heart dysfunction. This diagnosis is very important due to its prognostic significance. Here, no specific treatment was performed, but a specific long-term follow-up was adopted. Cardiac MRI could provide additional information in order to predict CMV myocarditis complications, such as arrhythmias and cardiac dilatation leading to heart failure.

Conclusion

Clinically significant heart injury may be a rare, but life-threatening, manifestation of CMV infection, even without alarming symptoms as chest pain or dyspnea. Myocarditis linked to CMV infection may be asymptomatic, but as dangerous as symptomatic forms. This is the reason that it would likely be interesting to perform cardiac MR-scan in CMV acute infections with heart involvement suspicion especially in patients with cardiac disease. Trans-thoracic echocardiography and cardiac MR-scan are non-invasive exams allowing precise tissue lesion evaluation.

References