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Unusual etiology of acute pericarditis with pericardial effusion in immunocompetent adult: Acute cytomegalovirus infection.

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ABSTRACT
We report the case of a 70 years old patient who has an acute pericarditis accompanied by a pericardial effusion of moderate abundance, secondary to an unusual etiology in immunocompetent patients: acute cytomegalovirus (CMV) infection. Will be discussed clinical aspects, the difficulties of serodiagnosis and principles of treatment.

INTRODUCTION

Acute pericarditis has many possible etiologies and can develop due to diverse conditions. Viral and idiopathic nonspecific causes are the most prevalent etiologies in developed countries and generally have a benign course. In immunocompetent individuals, most CMV infections are clinically silent or manifest by a banal influenza-like syndrome. Typical forms, minority, are manifested by prolonged fever, lasting for several weeks with mononucleosis-like syndrome, pain syndrome (headache, myalgia), asthenia and moderate elevation of transaminases. Forms with acute pericarditis accompanied by moderate pericardial effusion are rare (1).

MATERIALS AND METHOD

We report the case of an immunocompetent patient of 70 years. He had hypertension followed for 10 years and febrile syndrome with myalgia occurred 20 days earlier. On admission the patient was hemodynamically stable, subfebrile, with dyspnea stage II of NYHA. Physical examination was normal without hepatomegaly, peripheral edema, elevated jugular venous pressure, or enlarged tonsils. No lymph nodes were palpable. The electrocardiogram objectified microvoltage and left ventricular hypertrophy. The chest X-ray demonstrated an enlarged cardiac silhouette with normal lung fields. Blood tests revealed a markedly elevated erythrocyte sedimentation rate, above 85 mm in the first hour, mild leukocytosis without atypical or relative lymphocytosis, normal liver function tests, normal troponin and creatine phosphokinase and normal thyroid stimulating hormone levels. Transthoracic echocardiography was then performed and revealed moderate circumferential pericardial effusion (figures 1, 2, 3), normal left ventricular (LV) function, without signs of tamponade. Thoracic Computed Tomography Scan showed moderate pericardial effusion (figure 4). Situated in a tuberculosis endemic country, Mantoux test and the research of Acid-fast bacillus (AFB) in sputum and gastric aspirat fluid are performed. This research came back negative. Broad

serological tests of infectious and collagen disease were taken, including hepatitis viruses, Brucella, Mycoplasma, Rickettsia, Q-fever, Legionella, VDRL, cytomegalovirus, Epstein-Barr virus (EBV), human immunodeficiency virus, coxsackie viruses, and antinuclear and rheumatic factors. Blood and urine cultures were sterile. All serological tests came back normal, except positive CMV IgM and IgG antibodies performed by enzyme-linked immunosorbent assay ELISA. Measurement of the CMV specific IgG avidity index was low which allowed establishing the diagnosis of recent CMV primary infection. With a diagnosis of acute pericarditis accompanied with pericardial effusion, secondary to cytomegalovirus infection in an immunocompetent patient, therapy with aspirine (3g/d) was initiated. The third day of treatment, dyspnea decreased very significant way; a pericardial friction rub was first recognized on auscultation at the left sternal border. The tenth day of treatment, the inflammatory syndrome had completely disappeared and the echocardiography showed a significant reduction in the quantity of pericardial effusion. The patient was discharged on the same therapy and guidance for further follow-up. Echocardiography repeated at three weeks (figure 5A) showed the persistence of a blade (5mm) pericardial effusion in lateral LV. Echocardiography performed at two months (figure 5B) showed a complete disappearance of the effusion.

RESULTS AND DISCUSSION

Acute pericarditis may have many causes including infections, malignancy, collagen vascular and autoimmune conditions, uremia, myocardial infarction, trauma, surgery, hypothyroidism, and drugs such as hydralazine and procainamide. Viral infections and idiopathic causes, which are probably viral or autoimmune in most cases, are the prevalent etiologies in developed countries (2).

Viral pericarditis is notably associated with coxsackie viruses and other enteroviruses but has also been associated with numerous other viruses, such as adenovirus, hepatitis viruses, HIV, Epstein-Barr virus (EBV), and parvo B19 virus (3).

CMV pericarditis has an increased incidence in immunocompromised and HIV infected hosts (4). In immunocompetent individuals, CMV Pericarditis is rare, usually asymptomatic, and objectified by ECG abnormalities during acute infection (5,6) . Severe forms (tamponade, constrictive pericarditis) have occurred in patients with predisposing factors (rheumatoid arthritis treated with steroids, chronic renal failure, congenital heart disease) (7,8,9).

Inflammatory abnormalities are due to direct viral attack, the immune response (antiviral or anticardiac), or both. Deposits of IgM, IgG, and occasionally IgA, can be found in the pericardium and myocardium for years (10).

Serodiagnosis of CMV is usually based on the detection of specific IgM and IgG (11) by enzyme-linked immunosorbent assay (ELISA) or equivalent techniques. The positivity of IgM anti-CMV is not in itself a sufficient argument to confirm the diagnosis of a primary CMV infection. In immunocompetent patient, IgM positivity in a specific clinical context is just one additional presumption element. In the presence of IgG, the determination of the avidity index is an important element to affirm (low index) or disprove (high index) diagnosis of primary CMV infection. The interpretation remains difficult to intermediate avidity index. The evolution of the avidity index over time (> 1 month) can help in this case to distinguish an old infection of recent infection: a stable index is in favor of an old infection, and that an increasing index is in favor of a relatively recent infection. In the absence of IgG, the diagnosis can be confirmed by the detection of IgG seroconversion on a second sample or, if possible, for the detection of CMV viremia (antigenemia, DNAemia) (12). The techniques of PCR amplification of the genome have greatly facilitated the diagnosis of these infections, particularly the quantitative measurement of the viral load in the blood.

Etiologic evaluation of pericarditis and pericardial effusion is frequently unsuccessful, especially when noninvasive methods are used. Zayas and al evaluated 100 consecutive patients who were hospitalized for acute primary pericarditis using a standardized diagnostic protocol that included pericardial drainage and biopsy, when indicated, and identified a specific cause in only 20% of the patients (13).

Echocardiography remains the investigation of choice in acute pericarditis. It allows assessing the amount of effusion and tolerance by looking for signs of tamponade. But it does not allow an accurate assessment of the thickness of the pericardium. The scanner and especially MRI allow to visualize the entire pericardium and its thickness can be determined easily. It is of great interest for suspected associated myocarditis (14,15). In Matter of pericarditis, pericardial hyperintensity on both sheets on T2-weighted images enhances the diagnosis of acute or subacute inflammatory process. Cine-MRI allows assessing cardiac function and especially appreciating the absence of progression to constrictive pericarditis. MRI is superior to echocardiography to specify the nature of the pericardial effusion (16).

Treatment of viral pericarditis is directed to resolve symptoms, prevent complications, and eradicate the virus. The vast majority of CMV infections are spontaneously favorable evolution in the absence of immunosuppression. Nonsteroidal anti-inflammatory drugs (NSAIDs) and aspirin, under cover of a gastrointestinal protection, are the first-line treatment in CMV pericarditis. In the current case of recent cytomegalovirus infection with pericarditis moderate effusion, without signs of tamponade, not causing any hemodynamic compromise, and the rapid, significant response to a therapeutic trial with aspirin, initial drainage is not indicated (17). In patients with chronic or recurrent symptomatic pericardial effusion and confirmed viral infection, the authors mention, in general, a positive response, often very fast under antiviral therapy. Several anti-viral are used. We will cite Ganciclovir (1988) Foscarnet (1991) and Valganciclovir (2001) (18).

CONCLUSION

The possibility of primary CMV implication should be considered in cases of acute pericarditis accompanied with pericardial effusion in immunocompetent patient. The difficulties in interpreting the serodiagnosis of CMV must be mastered. Although evolution is usually spontaneously favorable under NSAIDs drugs, we must think that severe forms of CMV infection in immunocompetent subject are possible.

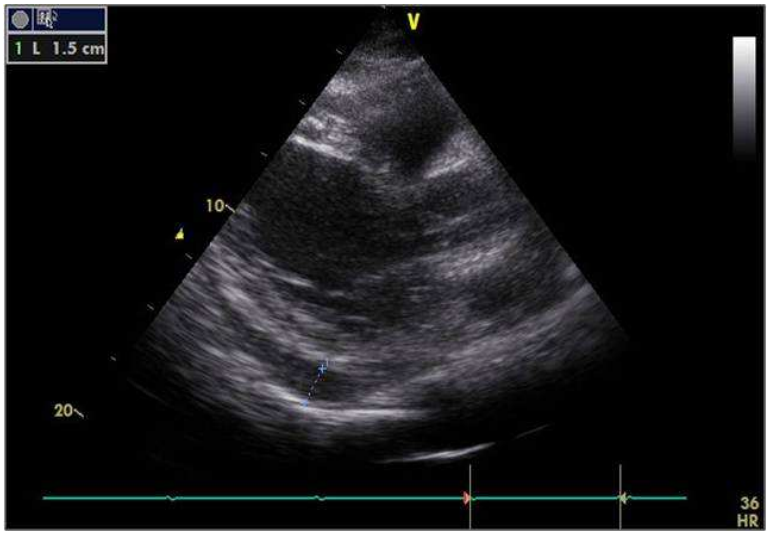

Figure 1: Para sternal long axis section showing pericardial effusion moderate abundance.

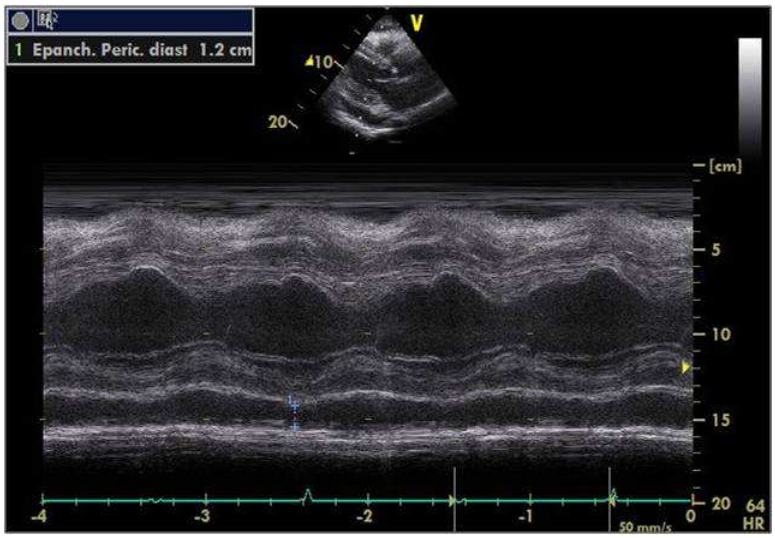

Figure 2: Clear space, empty of echoes between the layers of the pericardium.

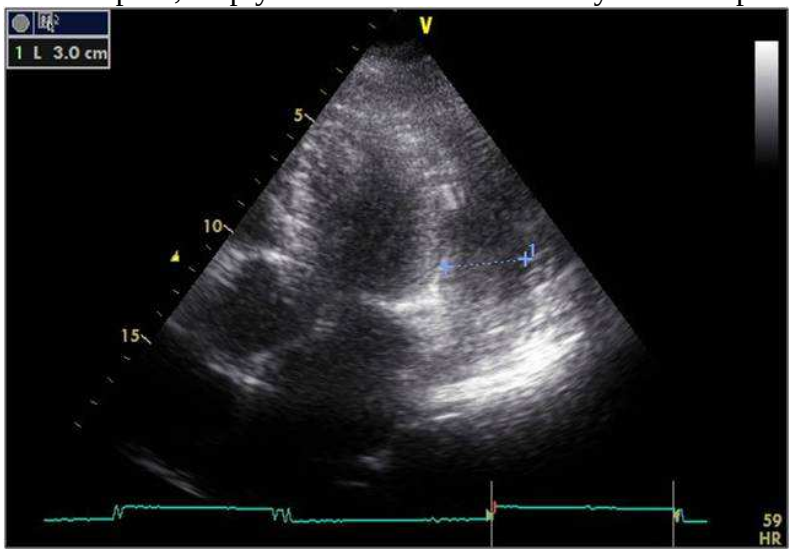

Figure 3: 4 cavities section objectifying a large pericardial effusion in lateral LV.

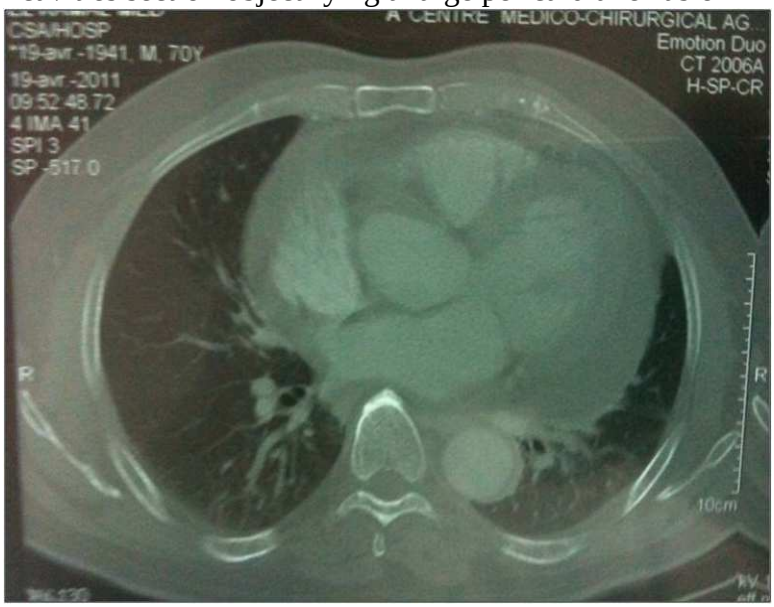

Figure 4: Thoracic Computed Tomography Scan showing pericardial effusion.

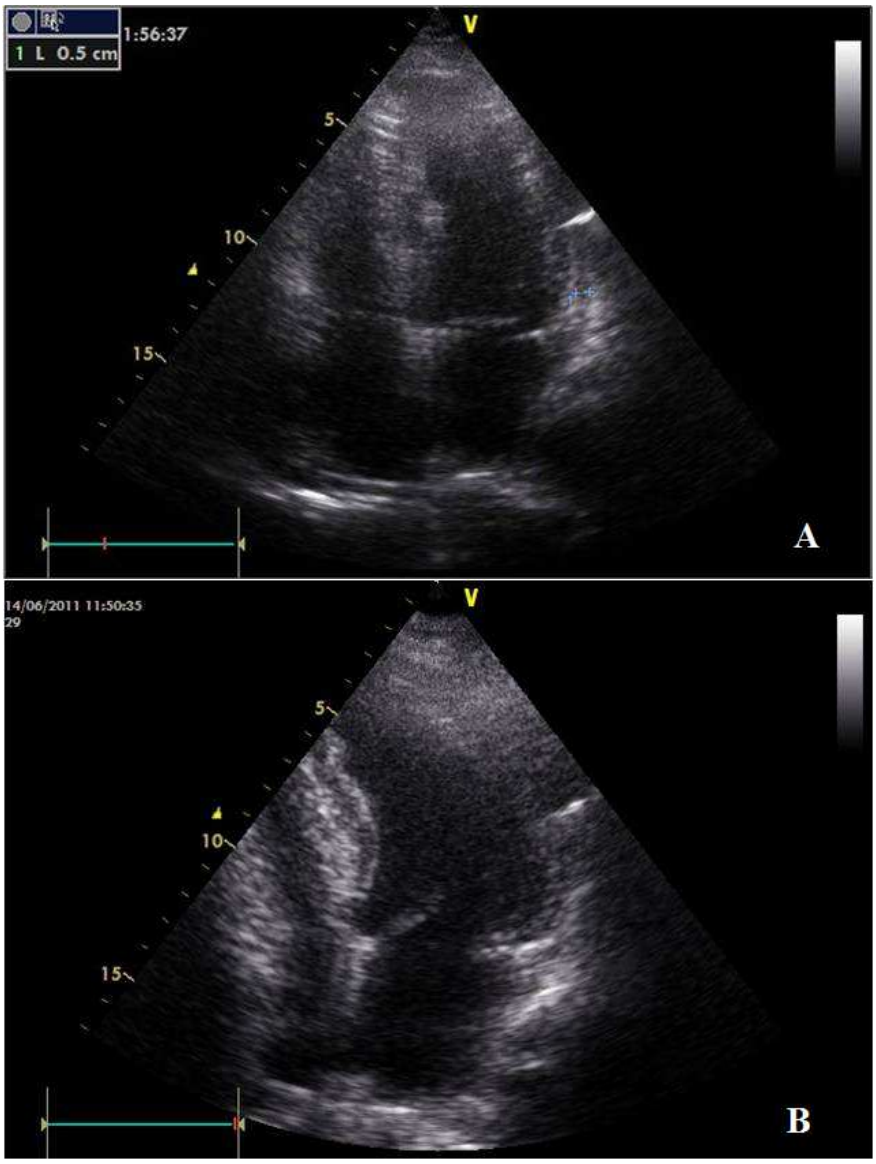

Figure 5: Evolution of the pericardial effusion after 3 weeks (A) and after 2 months (B) of treatment.

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