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Scientific letter

Myocarditis by *Toxoplasma gondii* in an immunocompetent young man

Miopericarditis subaguda por Toxoplasma gondii *en paciente inmunocompetente.*

We present the case of a 15-year-old male patient, with no relevant previous medical history, who came to Accident & Emergency reporting oppressive, non-radiating chest pain associated with dyspnoea lasting approximately one hour, within the previous 24 h. He reported no associated sweating or dizziness, or palpitations, but he had had a fever in the previous few days. On examination: BP 100/54 mmHg; HR 100 bpm; afebrile; $SatO_2$ 98%; heart sounds were of tachycardia without murmurs; and the rest of the examination was unremarkable.

A series of electrocardiograms (ECG) showed diffuse concave ST-segment elevation, more marked in the lateral and inferior leads. Laboratory tests showed elevated troponin I (8.66 mg/dl). The rest of the laboratory tests, chest X-ray and transthoracic echocardiogram were normal. Cardiac magnetic resonance imaging (cardiac MRI) was performed; the study met two diagnostic criteria (Lake Louise) for myocarditis by cardiac MRI: hyperintensity on the T2-STIR sequence associated with oedema, with late enhancement with a lateral subepicardial pattern consistent with hyperintense segments on T2-STIR. Both ventricles had normal segmental and overall contractility, with a left ventricular ejection fraction of 56% (Fig. 1A). The patient was diagnosed with myopericarditis and admitted to cardiology for seven days. He made good progress on treatment with non-steroidal anti-inflammatory drugs (NSAIDs) and cardiac MRI in six months was recommended.

Ten days after being discharged, the patient consulted with painless occipital and right retroauricular lymphadenopathy, 1–1.5 cm in size and adhered to deep planes. He had not had any fever or other symptoms of infection. He only reported having had contact with cats and a canary. Multiple serology was performed for viruses (CMV, EBV, HIV, HBV, HAV, ECV, rubella), syphilis and toxoplasma, as well as an immunological study. He was diagnosed with acute toxoplasmosis (IgG 1,100 IU/ml and IgM 28.65 IU/ml) and no treatment was prescribed, as he was asymptomatic.

Six months later, the patient attended the cardiology clinic for review after having had a follow-up cardiac MRI. The new cardiac MRI once again showed findings consistent with myocarditis with oedema and late enhancement, with a similar distribution to the previous scan (Fig. 1B, C). The Infectious Diseases Unit was consulted about the case and it was decided to request repeat serology for toxoplasma. This came back positive: IgG 2.16 IU/ml and IgM 3.33 IU/ml. PCR for toxoplasma was also requested from the reference laboratory, with negative results.

It was decided to treat with pyrimethamine 50 mg/12 h loading doses followed by 25 mg/12 h, sulfadiazine 500 mg, two/6 h for four weeks and calcium folinate 15 mg/24 h. From a cardiological standpoint, the patient was asymptomatic at all times, except for self-limiting episodes of palpitations, for which he received treatment with carvedilol 6.25 mg/12 h. At the next follow-up cardiac MRI four months later, the patient showed no signs of myopericarditis.

Acute *Toxoplasma gondii* infection in immunocompetent patients is asymptomatic in most cases, but they may occasionally develop fever, lymphadenopathy and asthenia.[1] However, there are very few cases published in the literature of *T. gondii* myocarditis in non-immunocompromised patients.[2] The clinical manifestations associated with myocarditis are fever, dyspnoea, chest pain, lymphadenopathy and myalgia.[3,4] In our case, the patient had a primary infection due to *T. gondii*, which we decided not to treat as he was immunocompetent and practically asymptomatic, despite the fact that the onset was with acute myopericarditis. At subsequent check-ups, he was diagnosed with subacute myocarditis, so due to

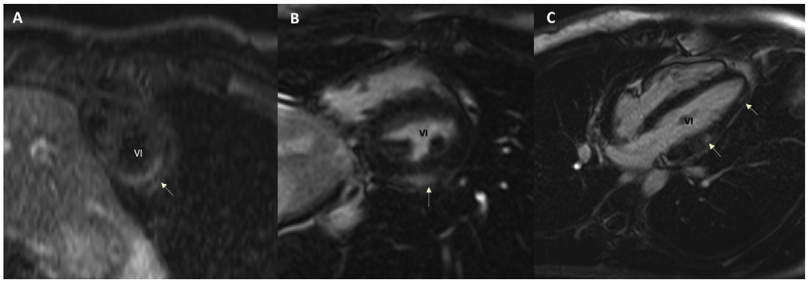

Figure 1. Cardiac magnetic resonance imaging study. A. Enhanced sequences (short-tau-inversion recovery [STIR]) revealed myocardial oedema in the lateral-apical aspect (arrowheads). B,C. In the later study after the intravenous administration of contrast, gadolinium uptake was observed with a subepicardial and lateral intramyocardial distribution (arrows).

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the persistence of disease, we decided he required treatment, and this then resolved the condition.

In the published cases of myopericarditis in immunocompetent patients, the treatment is not clear, and although most authors decide to give specific treatment, as in our case,[4–8] there are reports of cure with no treatment.[9,10] Although it been described, the persistence of myocarditis six months after diagnosis of the acute infection is also unusual.[5]

Routine serology testing is not usually performed in myopericarditis, as it has low diagnostic yield and rarely changes patient management. However, repeat cardiac MRI is recommended in the follow-up of these patients, as the persistence of inflammatory activity is not a common finding and should prompt us to seek an aetiological diagnosis and specific treatment.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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Severe acute hepatitis in a person with HIV and simultaneous infection with hepatitis C virus and SARS-CoV-2

Hepatitis aguda grave en persona que vive con VIH: infección simultánea por el virus de la hepatitis C y el SARS-CoV-2

Dear Editor,

Acute hepatitis due to the hepatitis C virus (HCV) is frequently seen in men who have sex with men (MSM), coinfected or not with HIV, but the presentation as severe hepatitis is extremely uncommon.[1] SARS-CoV-2 infection can have different effects on the liver, including direct parenchymal infection, cytokine-mediated tissue injury, etc.[2] However, it is not clear whether SARS-CoV-2 could change the clinical expression and/or natural history of viral liver diseases.

Here, we report the case of an HIV-infected man with concomitant SARS-CoV-2 and HCV infection, who developed severe acute hepatitis.

A 44-year-old male infected with HIV, who had been taking antiretroviral treatment with tenofovir disoproxil alafenamide, emtricitabine, and darunavir-cobicistat for 5 years, attended the Emergency Department of our hospital. He was a MSM with an open relationship partner, without regular condom use. He reported not attending chem-sex parties or using intravenous drugs. He showed plasma HIV load below the detection level and 891 CD4 cell/μL three months earlier. He had not been vaccinated against SARS-CoV-2. He complained of a fever and cough of 48 h. He reported narrow contact with a relative with COVID-19. Then a SARS-CoV-2 nasopharynx PCR test was performed, with a negative result. An X-ray chest film showed no evidence of pneumonia. He was discharged without treatment. Fifteen days after initial presentation, he returned to the Emergency Department complaining of nausea, fever reemergence, general malaise, diffuse abdominal pain, jaundice, and dark urine for 2 days. Physical examination revealed conjunctival jaundice, without evidence of encephalopathy or ascites. A new nasopharynx SARS-CoV-2 PCR test yielded positive results. Blood analysis showed plasma gamma glutamyl transferase (GGT) 171 U/L, alanine aminotransferase (ALT) 10,339 U/L, aspartate aminotransferase (AST) 6470 U/L, alkaline phosphatase (FA) 201 U/L, total bilirubin 6.2 mg/dL, INR 1.58 and ferritin 38,009 ng/mL. Serum antibodies against HCV, which had been negative 6 months earlier, were then positive. Plasma HCV-RNA level was 219,000 IU/mL (genotype 1a). He received intramuscular vitamin K and no other therapy. He did not develop signs of encephalopathy but, on day 3 of admission, total bilirubin rose to 9.7 mg/dl, whereas aminotransferases had declined (ALT 7566 U/L and GOT 2971 U/L). HCV-RNA became undetectable and plasma bilirubin and aminotransferase levels returned to normal values 4 weeks after symptom onset. The evolution of the liver function test in the acute hepatitis episode is shown in Fig. 1.

The vast majority of cases of acute HCV infection are asymptomatic and fulminant hepatitis is an exception.[1] The patient reported here developed acute hepatitis C with severity signs, such as increased INR and transaminase levels consistent with