Fever and acute cytomegalovirus hepatitis in Crohn’s disease

Key words: Cytomegalovirus. Hepatitis. Crohn’s disease. Ulcerative colitis.

Dear Editor,

The control of inflammatory bowel disease (IBD) is complex. Immunomodulatory therapies cause situations of immunosuppression and increases susceptibility to opportunistic infections. We report a case of acute hepatitis and Crohn’s disease with prolonged fever in the context of a cytomegalovirus (CMV) primo-infection.

Case report

We present a 33-year-old male patient with ileal Crohn’s disease on maintenance treatment with 6-mercaptopurine. The patient went to the emergency department because of fever, fatigue and increased serum transaminase levels. The physical examination was unremarkable. An abdominal CT scan with intravenous contrast showed inflammation of the ileum however, all other tests ruled out other sources of infection.

Funduscopia was normal and lumbar puncture was not possible due to patient’s scoliosis. The patient received empirical treatment with ceftriaxone without improvement.

Serology for herpes, Epstein Barr virus, Coxiella, Legioella, HIV, Lues, Brucella, ag galactomanann, QuantiFERON, stool cultures and Clostridium toxine were negative. The CMV IgM was positive as the blood polymerase chain reaction (PCR) tests.

Before starting treatment with ganciclovir, liver biopsy was performed observing a lymphocyte-predominant infiltrate and necrosis of the limiting plate with some granuloma necrotizing and virus isolation by immunohistochemical techniques (Fig. 1).

The patient was discharged after 21 days of treatment with a diagnosis of acute CMV hepatitis receiving additional valacyclovir for a week without relapse.

Fig. 1. A. Granuloma necrotizing with a lymphocyte predominant infiltrate. B. Presence of lymphocytes and some eosinophils in a duct.
Discussion

CMV incidence in patients with IBD ranges from 0.53 % to 3.4 % increase if receiving immunosuppressive agents (1-3).

The prevalence in Crohn’s disease is low, whereas in ulcerative colitis (UC) it can rise up to 70 % (4, 5).

The affinity of the virus in tissues and the high seroprevalence suggest that the virus plays only an incidental role. However, it has proven to be a pathogen in 20-30 % rate of severe steroid-refractory IBD.

The first case of UC and CMV was described by Powell et al. in 1961 (6). Most are due to reactivation of a latent infection. The presentation of the disease as an acute mononucleosis as in our case is rare (2, 7).

CMV infection occurs in steroid-refractory UC, but were not the case in our patient who had a Crohn’s disease and had not received corticosteroids although he was an immunocompromised patient due to treatment with mercaptopurine (8).

The diagnosis is based on histological techniques, as other methods do not differentiate between a latent colonization and active infection (9, 10). Another technique is PCR in intestinal tissue or blood. Immunohistochemistry (IHC) is the standard diagnostic technique.

Clinical guidelines stipulate that the diagnosis should include PCR (without defining whether blood or colonic mucosa) and colon IHC.

Ganciclovir is the treatment of choice in severe steroid-refractory UC. However, other studies have reported improvement without antiviral treatment.

In conclusion, we present an atypical case of acute CMV hepatitis in a patient with a corticosteroid-free treated Crohn’s disease, without intestinal involvement and favorable outcome after antiviral therapy.

References