Severe Crohn’s Disease in a HIV-HCV Co-Infected Man

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Case Report

We report the case of a man 47-year-old, with Human Immunodeficiency Virus (HIV) and Hepatitis C Virus (HCV) infection diagnosis since 1998. When he was twenty, he had been intravenous drug user. In November 2006 he had 608 lymphocytes CD4/μl and a viral load of 12,600 cop/ml of HIV, without antiretroviral treatment (ART), but a HCV viral load of 5,000,000 UI/ml, genotype 1 and portal hypertension in echography. He began treatment with pegylated interferon (p-IFN) and ribavirin at a dose of 180 μg/week and 1500 mg/day respectively, for his first time, in January 2007. At week 12, the HCV viral load was 185 UI/ml.

In December 2007, with 936 lymphocytes CD4/μl, he showed an intense cutaneous eruption, with fever (38°C) a dry cough, and was admitted in the hospital. At this point, viral load for HIV was undetectable. Treatment for HCV infection was stopped and eruption improved, but after a week, he began with diarrhoea with culture, detection of parasites and Clostridium difficile toxin in stools all negatives. Colonoscopy was made, and it showed the presence of deep ulcers, with a biopsy congruent with inflammatory bowel disease, beginning treatment with intravenous steroids. Lymphocytes CD4 rose down to 245 cells/μl and HIV-1 viral load rose up to 43,424,020 cop/ml. So, ART with tenofovir, emtricitabine, efavirenz and enfuvirtide was instaured. After this, cutaneous lesions got worse and a biopsy of skin was made, with diagnosis of seborreic dermatitis. Four weeks after admittance, the patient had lost 30 kg of weight and fever continued. PCR test in blood for cytomegalovirus was negative, scan of thorax and abdomen did not showed collections, fundoscopy, transthoracic echocardiography, blood and sputum cultures, blood cultures for mycobacteria and bacilloscopies in sputum and urine were normal too. So, capsule endoscopy was made, revealing terminal ileitis with ulcers deep and fibrinous, with a biopsy of Crohn’s disease. The patient began treatment with mesalazine, oral corticosteroids, metronidazole and metronidazole, despite ART, and general status improved and fever and diarrhoea disappeared. He was discharged with treatment with mesalazine, oral corticosteroids, emtricitabine, efavirenz and tenofovir.

Two weeks after, he was admitted again because fever and worsening of general status. Parenteral nutrition was instaured and Staphylococcus epidermidis was isolated in blood cultures, beginning treatment with vancomycin (1 gr BID, iv for two weeks). Fourteen days after admission, the patient began to have bloody and intense diarrhoea, with hypotension, and ileostomy was made on day 17 after admission. The patient improved and walked without help, he gained weight and fever disappeared, but bloody and intense diarrhoea appeared again, beginning adalimumab 40 mg weekly was instaured, with an excellent response of the clinical status, fever and diarrhoea. Ten days after, HIV viral load was 83 cop/ml and lymphocytes CD4 were 50 cells/μl. Four weeks after instauration of adalimumab, the patient presents a sepsis with infiltrate in right lung, and a positive culture in bronquial samples for E coli, with extended spectrum beta lactamases production, that was treated with etrapenem, but the patient died.

Discussion

There are few cases published about Crohn’s disease in the context of treatment with peg-IFN and ribavirin in people with HIV infection [1]. Although anti-TNF-α such etanercept and infliximab have been used safely in a short number of cases in patients with HIV infection [2,3], ours died in the context of sepsis, probably facilitated by the immune depression induced by adalimumab. Peg-IFN has a very high antiviral activity, but has complex immune effects that can lead to exacerbate previous immune disorders. We cannot exclude that Crohn’s disease, diagnosed previously to the beginning of ART, was exacerbated in the context of immune reconstitution syndrome, because the very important decrease of viral load in this patient, since the cutaneous lesions worsened after beginning of ART. By the other hand, despite the dramatic decreasing of viral load, lymphocytes CD4 rose down. It has been described in relationship with apoptosis of these cells induced by infliximab [4]. Certainly, the coincidence of severe immune disease with HIV infection leads us to an infrequent scenario, in which each decision must be individualized.

Management of severe autoimmune diseases in people with HIV infection can be very difficult, looking for the balance between immune suppression and infectious complications.

References