LETTER TO THE EDITOR

Pulmonary and nodal tuberculosis in a patient with inflammatory bowel disease and HIV infection treated with infliximab

KEYWORDS
Infliximab; TNFα; Inflammatory bowel disease; HIV; Tuberculosis

Dear Sir,

Tumour necrosis factor (TNF) plays an important role in HIV infection, allowing an intracellular reservoir of the virus.1 For this reason, TNF was considered an appealing therapeutic target in HIV-infected patients; however, the use of anti-TNF agents only achieves a transient decrease in TNF levels, with no changes in CD4 count and viral load.2 TNF is also a key cytokine in the pathophysiology of inflammatory bowel disease (IBD), and anti-TNF agents have become a cornerstone in its management. A major drawback in the use of anti-TNF in IBD is the risk of developing severe infectious complications, but no recommendations for those patients with immunosuppressive conditions other than drug-induced immunosuppression such as HIV infection or congenital immunodeficiencies are available. We report the case of an HIV-infected patient with indeterminate colitis who was treated with infliximab.

A 47-year-old male of Pakistani origin with no history of drug abuse was diagnosed of indeterminate colitis in January 2006, while living in Spain. Ten months later, HIV infection was diagnosed when the patient was re-evaluated because of persistent mild-to-moderate disease activity despite oral mesalazine; at that time, CD4 count was within the normal range, viral load was low (50 copies) and no specific treatment was prescribed. In January 2008, azathioprine was started because of steroid-dependent disease; three months later, when he was still on systemic steroids, he presented cryptosporidium enteritis in the setting of an increase in HIV viral load, and a CD4 count of 474 μL. Although intestinal infection was resolved, the patient relapsed of his abdominal symptoms while tapering prednisone. Infliximab therapy was started after latent TB was ruled out (normal chest X-ray, negative tuberculin skin test and "booster"). In addition, antiretroviral therapy with efavirenz (EFV) and tenofovir (TDF) was also prescribed given a progressive increase in viral load (3600 copies) and a fall in CD4 count (368 cells/μL). The patient experienced a rapid decrease in viral load to ~50 copies (Fig. 1). After the third infliximab infusion, he was admitted because of fever and malaise; chest X-ray showed a widened mediastinum and tuberculosis was diagnosed by a lymph node fine-needle puncture, which was complicated by the bronchial dissemination of the infection leading to the pulmonary spread of TB. Tuberculostatic drugs were started, and infliximab and azathioprine were discontinued. The patient slowly improved his clinical status, although several infectious complications such as a bacterial pulmonary abscess and a herpetic stomatitis led him to a 5-month hospital stay. Indeterminate colitis remained clinically and endoscopically inactive throughout the hospital stay only on mesalazine therapy.

In clinical practice, anti-TNF treatment in patients with rheumatologic or dermatologic diseases and concomitant HIV infection several case reports have been reported,3–6 with no infectious complications. To our knowledge, only one case of inflammatory bowel disease and associated HIV infection treated with infliximab has been reported to date, with a similar outcome.7 In the present case infliximab administration was followed by the development of a severe opportunistic infection, despite antiretroviral drugs (which are known to promote immune reconstitution) were concomitantly started. Moreover, an appropriate work-up to rule out latent tuberculosis was not enough to prevent this infection, highlighting that current preventive measures in patients with marked immunosuppression or coming from epidemic areas of tuberculosis need to be optimized.

References


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20 April 2009