LETTER TO THE EDITOR

Crohn’s disease of the large bowel following diagnosis of chronic lymphoid leukaemia: A case report

Dear Sir,

It is well established that haematological malignancies could develop during the clinical course of Inflammatory Bowel Disease (IBD).1,2 Epidemiological data suggest that this risk might be increased1 and be related with the use of either immunosuppressants or biologic agents.4 So far, these malignancies have been observed after the establishment of diagnosis of IBD.

We describe the case of a patient in whom the diagnosis of Crohn’s disease (CD) was made 6 years after the diagnosis of chronic lymphoid leukaemia. To the best of our knowledge, the appearance of CD years after the establishment of chronic lymphoid leukaemia has not yet been described.

A 71-year old man was admitted to our department on November, 2011 because of persistent diarrhoea, fatigue and loss of weight. Chronic lymphoid leukaemia was incidentally diagnosed 8 years earlier. Chemotherapy was started and continued with satisfactory clinical results. The patient was in a good health up to one year before, when he started complaining of watery stools (>8/d), fatigue, low fever, and loss of weight. Five months later he developed a perianal fistula which was surgically confronted.

At that time a colonoscopy showed moderately inflamed mucosa of the descending, transverse and ascending colon. Rectosigmoid mucosa was grossly normal, as it was the terminal ileum. Histology revealed mild non-specific ileitis whilst large bowel histology was compatible with IBD. An upper GI endoscopy revealed mild antral gastritis which was confirmed histologically. The WBC were 30,600 (lymphocytes 19,700 [64.3%), of moderate size 2100 [7.0%), and neutrophils 8800 [28.7%]). He was treated with peros corticosteroids and mesalamine. However, because of the persistenct of diarrhoea, he was referred to us. On admission he was complaining of 3–5 loose stools per day, loss of weight and fatigue. Laboratory investigation revealed WBC 21,130 (neutrophils 39%, lymphocytes 27%, large lymphocytes 12%, monocytes 21%, and eosinophils 1%), reduced levels of serum immunoglobulins, complement, and fibronectin and increased serum levels of a1-acid-glycoprotein. An abdominal computed tomography revealed multiple lymph nodes of normal size and moderate descending colon wall thickness. On colonoscopy the ileocecal valve was edematous not allowing the colonoscope to pass into the ileum. The endoscopical picture was compatible with CD (large ulcers, and pseudopolyps in the right and transverse colon), whilst histology showed dense inflammatory infiltration, solitary granulomas and an area with picture compatible with DALM. Immunohistochemistry for CMV was negative. The patient refused to accept treatment with biologic agents. He started on azathioprine which however produced severe dyspeptic complains and abnormal LFT and was interrupted. He is now on peros prednizolone and Modulen-IBD (an immunomodulating diet rich in TGF-β, 50 g × 3/d). After 4 weeks the patient achieved clinical remission. He continues to be on low doses of corticosteroids and Modulen-IBD 4 months after the establishment of diagnosis.

We have no obvious explanation concerning the possible etiopathogenetic link between these two disorders. However it has been suggested that both disorders could share similar pathologic mechanisms as lymphocytes in both diseases fail to undergo apoptosis and die properly due to increased signalling by TNF-α, and their respective pathologies directly follow from this apoptosis failure.5

In conclusion, this case underlines the fact that CD could develop during the course of chronic leukaemia after the establishment of diagnosis of the haematological malignancy. Clinicians could keep this in mind thus proceeding with the relevant investigation of a patient with chronic lymphoid leukaemia who complains of symptoms from the digestive tract.

Conflict of interest

The author has no conflict of interest to declare.

References


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