LETTER TO THE EDITOR

Rapid onset of ulcerative colitis after treatment with interferon β1a in a patient with multiple sclerosis

Dear sir,

Interferon is frequently used in chronic viral hepatitis, and it is also used in treatment of multiple sclerosis, certain solid tumors and myeloproliferative diseases. Side effects are not rare; it may exacerbate pre-existing autoimmune diseases and may precipitate the development of autoimmune diseases, especially in thyroid and liver.1 With this case report, it’s intended to alert clinicians on a side effect of interferon, active ulcerative colitis, in a patient with multiple sclerosis which had developed in a very short time after initiation of beta interferon treatment.

A 44-year-old woman referred to our hospital with vomiting, abdominal pain and distension, and bloody diarrhea for 3 days. A week ago, interferon beta 1a treatment (30 mcg) was initiated for multiple sclerosis. Bloody diarrhea was up to 9–12 times a day and has started after one dose of newly initiated interferon treatment. Vital signs were normal other than a body temperature of 38 °C. In physical examination she had high bowel sounds and had tenderness in all quadrants of the abdomen. Other systems were normal. Laboratory analysis was as follows: Hg: 12.1 g/dl, leucocytes: 13,190/mm³, platelets: 110,000/mm³. Biochemical parameters including liver enzymes, renal function tests, and electrolytes were in normal limits. Sedimentation rate was 28 mm/h, C-reactive protein: 29.4 mg/dl (N: 0–0.8), and prothrombin time was 13.4 s. Serological tests for hepatitis B, hepatitis C and HIV were all negative. The stool examination for ova, parasites and cultures was also negative. Abdominal X-ray was normal. Abdominal ultrasonography revealed thick edematous intestinal walls and no intraabdominal fluid was observed. BT angiography showed normal vascular structures without obstruction, severe edema and thickening in every region of colon wall. Colonoscopic examination revealed diffuse edema and hyperemia, ulcerations and mucoid exudates on mucosa in all segments of the colon from rectum to cecum. Biopsies obtained from the mucosa of the colon revealed ulcerative colitis. Interferon beta treatment was thought as the triggering factor and was discontinued, and mesalazine 3 g/day was initiated. On day 3, abdominal pain disappeared and bloody diarrhea has returned to normal. On 20th day of treatment, the patient had no complaints and rectosigmoi-

doscopic examination revealed complete healing of the mucosal lesions.

In the literature, there are conflicting reports regarding the effect of interferon on inflammatory bowel diseases. Up to date a few ulcerative colitis cases were reported to be associated with interferon treatment in the literature.2–5 On the other hand, it was reported that observation of no decrease in colitis activity after discontinuation of interferon beta 1a treatment was related with the persistent autoimmune response. In a case–control study conducted by Bargiggia et al., no relapse of inflammatory bowel disease was observed in patients with HCV, treated with interferon, for 12 months.6 On the other hand, it was stated that interferon treatment causes gastrointestinal diseases such as celiac disease and Crohn's disease through potent immune activation.7,8 Interferon may be related with the development or exacerbation of ulcerative colitis. The site of involvement may vary in each interferon induced ulcerative colitis patient from total colitis to rectitis and the time onset of ulcerative colitis also varies from the first day of the initiation of interferon to the week after stopping the drug.4–8 Treatment of interferon induced ulcerative colitis involves discontinuation of interferon treatment, mesalazine monotherapy or short-term steroids combined with mesalazine.

In conclusion, INF treatment can be a predisposing factor for development or exacerbation of UC.

Conflict of Interest Statement

We (all authors, Yaşar Tuna, Ömer Başar, Hilmi Dikici and Seyfettin Köklü) declare that there is no conflict of interests among the authors.

References


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