

Peripheral Neuropathy Under Anti-TNF Therapy in a Patient with Ulcerative Colitis: A Case Report

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Abstract

Case Report

Infliximab is widely used in moderate to severe ulcerative colitis (UC). Neurological side effects of anti-TNF agents are uncommon, and when they occur it can be difficult to separate a drug reaction from a neurological manifestation of the disease itself. We report a 26-year-old woman with UC, revealed by steroid-refractory acute severe colitis, who received infliximab with azathioprine. After a 10-month interruption of treatment due to loss to follow-up, infliximab was restarted for a moderate flare. Eight days after the infusion, she presented with rapidly progressive symmetrical weakness of the four limbs, paresthesia and decreased reflexes. Electroneuromyography showed a mixed polyradiculoneuropathy, mainly in the lower limbs. Viral and syphilis serologies, vitamin B1, B9 and B12 levels, autoimmune tests and cerebrospinal fluid analysis were normal. Infliximab was stopped and the neurological signs disappeared spontaneously within six weeks. After discussion with the neurologists, infliximab was restarted, with no recurrence and good control of the colitis. In this patient, the clinical course suggests that the neuropathy was more likely an extra-intestinal manifestation of UC than a direct effect of infliximab.

Keywords: Ulcerative colitis; Infliximab; Anti-TNF; Peripheral neuropathy; Polyradiculoneuropathy; Extra-intestinal manifestations.

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INTRODUCTION

Ulcerative colitis (UC) is a chronic inflammatory disease of the colon and rectum. It results from an inappropriate immune response to environmental factors in genetically predisposed individuals. Anti-tumor necrosis factor (anti-TNF) agents, and infliximab in particular, now have an important place in the treatment of moderate to severe forms [1]. Their side effects are rare and generally well described.

Neurological complications are a different matter. In a patient with inflammatory bowel disease (IBD) receiving anti-TNF therapy, a new neuropathy may be an extra-intestinal manifestation of the bowel disease or an adverse effect of the drug, and the distinction is not always obvious [2,3]. We report a case of peripheral neuropathy in a young woman treated with infliximab for UC.

CASE REPORT

A 26-year-old woman had been followed in our department for two years for UC, revealed by an episode of acute severe colitis that did not respond to intravenous corticosteroids. Pre-biologic screening was negative, and she was started on infliximab 5 mg/kg (induction at weeks 0, 2 and 6) combined with azathioprine. Clinical and biological response was good.

She was subsequently lost to follow-up for 10 months, and the infliximab infusions were stopped during this period. She came back with a moderate flare according to the Mayo score, a slight deterioration in general condition and an endoscopic Mayo subscore of 3. Infliximab was restarted.

Eight days after the infusion, she developed symmetrical muscle weakness of rapid onset involving all four limbs, with paresthesia. Tendon reflexes were decreased on examination. Electroneuromyography (ENMG) showed a mixed polyradiculoneuropathy predominating in the lower limbs.

The etiological work-up was negative: serologies for EBV, CMV, HIV, hepatitis B and C and syphilis, vitamin B1, B9 and B12 levels, and autoimmune tests. Lumbar puncture was normal, with clear cerebrospinal fluid, 2 white cells/mm³, 1 red cell/mm³, protein 0.20 g/L and glucose 0.52 g/L.

Infliximab was withheld. The neurological signs regressed and then disappeared completely, without specific treatment, within six weeks. The colitis improved clinically and biologically, but not endoscopically. After discussion with the neurologists, we decided to restart infliximab. No neurological symptoms recurred, and the disease has remained well controlled.

DISCUSSION

IBD mainly affects the digestive tract, but extra-intestinal manifestations are seen in 20 to 50% of patients, most often joint, mucocutaneous and hepatobiliary [2,4]. Neurological involvement is rare and probably underestimated. It can concern the central or the peripheral nervous system, with cerebrovascular disease, central demyelination or peripheral neuropathy (PN), which may be axonal or demyelinating, acute or chronic [2,3,5].

The mechanisms are still not clearly understood. Immune-mediated injury, a prothrombotic state, nutritional deficiencies and drug toxicity, in particular from anti-TNF α agents, have all been proposed [2]. Neurological involvement may worsen during flares of the bowel disease, but it may also follow its own course and fail to respond to treatment of the intestinal inflammation [2,3].

When drug-induced cases are excluded, PN is more frequent than central nervous system involvement. In the series of Gondim *et al.*, the mechanism was axonal in more than two thirds of patients, and the mean time to onset of neurological symptoms in UC was 26 years [3].

Since anti-TNF agents came into use in 1996, an increased risk of neurological events has been reported, mainly central demyelination such as optic neuritis and multiple sclerosis [2,6]. PN has been described in small case series. The incidence of neurological events under anti-TNF therapy is estimated at less than 1 per 1000 treated patients [2]. In reports to the Food and Drug Administration (FDA) Adverse Event Reporting System, patients with IBD were the second largest group after those with rheumatoid arthritis [7].

Most reported cases of PN are demyelinating, Guillain-Barré syndrome being the most frequent [8,9]. Chronic inflammatory demyelinating polyradiculoneuropathy and multifocal motor neuropathy with conduction block have also been reported, as well as mononeuritis multiplex and axonal

sensorimotor polyneuropathy, which suggests that axonal damage also plays a part [8]. The outcome of these events is usually favorable. Intravenous immunoglobulins (1 to 2 g/kg) have given good results in demyelinating forms, and gabapentin has also been used [8].

The effect of continuing anti-TNF α treatment on the long-term course of the neuropathy varies between reports, so stopping the drug does not always seem necessary [8]. In our patient, the symptoms resolved without immunoglobulins after infliximab was withheld and did not reappear when it was restarted. With the mixed pattern on ENMG and the negative work-up, this course makes a direct toxic effect of infliximab unlikely and points rather to an extra-intestinal manifestation of UC.

CONCLUSION

Various neurological disorders can be seen in UC, whether or not they are related to anti-TNF α treatment. Given the type of involvement and its timing in our patient, the neuropathy was most probably an extra-intestinal manifestation of UC. Infliximab could be safely restarted after a joint decision with the neurologists.

Patient consent: Written informed consent was obtained from the patient for publication of this case report.

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