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Letter to the Editor

Axonal neuropathy during a severe exacerbation of ulcerative pancolitis

K.H. Katsanos, Ch.D. Zois, V.E. Tsianos, I. Vagias, E.V. Tsianos

Sir, the exact incidence of neurological complications in inflammatory bowel disease (IBD) is unknown, with reports varying from 0.25 to 35.7%.[1] Peripheral neuropathy is included in the spectrum of neurological complications in IBD patients and clinical studies in IBD patients have provided evidence of an association between IBD and axonal or demyelinating neuropathy.[2]

We present herein a patient with acute motor and sensory axonal neuropathy during a severe exacerbation of ulcerative pancolitis diagnosed two years ago. A 48-year-old male presented with a flare of severe ulcerative pancolitis despite treatment with methylpredniozolone. The patient never received azathioprine but did received mesalazine (3g/day) since UC diagnosis with no evidence of neuropathy. During hospitalisation the patient complained of weakness and paresthesias in his left leg. Neurological assessment showed axonal sensorimotor polyneuropathy affecting the left lower limb. Magnetic resonance of the patient's spine was normal. Electrophysiological study showed motor axonal degeneration. Colonoscopy revealed severe active ulcerative pancolitis colitis. Vitamin and folic acid levels were normal. Virological, immunological and other laboratory tests were negative. After treatment with Infliximab the patient had gastrointestinal recovery while neurological symptoms significantly improved after five infliximab doses.

It has been suggested that IBD alters the function of the enteric nervous system and the sensory innervation of the gastrointestinal tract. Less is known about whether IBD also affects the sympathetic nervous system (SNS). Given the importance of the SNS in regulating gastrointestinal function and possibly immune system activation, the present report examines the evidence of sympathetic dysfunction during IBD and its possible consequences. [3]

Pathophysiologically, disorders of the nervous system in association with IBD can be ascribed to at least six different mechanisms, which may be present in isolation or in combination: (i) malabsorption and nutritional, particularly vitamin deficiencies such as B_1, B_{12}, D, E, folic acid and nicotinamide deficiencies (ii) toxic metabolic agents, (iii) infections as a complication of immunosuppression, (iv) side effects of medications (metronidazole, sulfasalazine, steroids, cyclosporine A) or iatrogenic complications of surgery, (v) thromboembolism, (vi) immunological abnormalities.[4-5]

In addition to these clearly defined and distinct etiologies, neurologic signs and symptoms may also be due to a so far speculative and not further specified neuronal influence of enteric disease on the nervous system (and vice versa). Such a hypothesis may be derived from contemporary theories considering the existence of a 'brain-gut axis', and from results of respective functional neuroimaging studies. Finally, the role of anti-TNFa therapies in such neuropathies in IBD patients is still to be defined.

1st Department of Internal Medicine & Hepato-Gastroenterology Unit University Hospital of Ioannina, Greece.

Author for correspondence:

Prof. Epameinondas V. Tsianos, MD, Ph.D, AGAF, Professor of Internal Medicine, Department of Internal Medicine, Medical School, University of Ioannina, Leoforos Panepistimiou, 45 110 Ioannina, Tel:++30-26510-07501, Fax:++30-26510-07016, email: etsianos@uoi.gr

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