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Guillain-Barré Syndrome as an Extra-Intestinal Manifestation of Ulcerative Colitis: A Case Report and Review of the Literature

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Abstract Case Report

Ulcerative colitis is a chronic inflammatory disease that affects the colon and rectum, the pathogenesis is complex. Extra-intestinal manifestation is present in 40% of patients with ulcerative colitis. The neurological involvement is part of the extra-intestinal manifestations. We report the case of a young patient of 29 years, followed for 5 months for ulcerative colitis, placed on azathioprin. Admitted with tetraparesis rapidly ascending, associated with hypoesthesia without any sphincter disorders, evolving. Guillain-Barré syndrome was confirmed based on the results of EMG and the study of cerebrospinal fluid and after elimination of other etiologies. The decision was to treat the patient with intravenous corticosteroid therapy and discuss the infusion of immunoglobulins but the patient died on day 1 of treatment because of cardiopulmonary arrest. Guillain-barré syndrome is an unknown and rare complication of ulcerative colitis even in the case of clinical remission of ulcerative colitis. This is a serious complication that is life-threatening. It is imperative to realize an EMG and the LCR study in front of any sign of appeal.

Keywords: ulcerative colitis, UC, Guillain-Barré syndrome, peripheral neuropathy, IBD.

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Introduction

Ulcerative colitis is a chronic and recurrent inflammatory disease that affects the colon and rectum. The pathogenesis is complex. It is reported that about 40% of patients with ulcerative colitis have extraintestinal manifestations, where as the literature on neurological involvement as extra-intestinal manifestation is rather limited [1]. The exact incidence of neurological complications is unknown, with ratios ranging from 0.2% to 35.7%; variation may be due to selection bias or different definitions of disease [2, 3].

Peripheral neuropathy (PN) is one of the most frequently reported neurological complications [2, 3]. Several NP phenotypes have been described in patients with IBD. Excluding known risk factors for neuropathy, such as vitamin B12 deficiency and metronidazole exposure, the association between IBD and peripheral neuropathy has been described only in cases and in small series [2, 3].

In this study, we describe an observation of a patient with ulcerative colitis who developed an acute polyradiculoneuropathy (PRNA) type Guillain-barré.

OBSERVATIONS

We report the case of a young patient of 29 years. She has been followed for 5 months for ulcerative colitis treated with azathioprine as a background treatment. Admitted in a table of ascending tetraparesis, rapidly progressive installation evolving since 3 weeks associated with hypoesthesia without sphincter disorders. She was in gastrointestinal remission, 1 to 2 stools / day without mucus, no blood, no abdominal pain, no rectal syndrome. On clinical Patient examination, generally impaired, hemodynamically and respiratory stable, flaccid tetraparesis with 4-member areflexia and sensory level D8. An etiological report was made. The drug and vaccine origin was eliminated during the interrogation. Infectious test, vitamin dosage and serology (HVB, HCV, HIV, CMV, EBV, HSV, and VZV) are negative.

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The electromyogram (EMG) was in favor of demyelinating acute sensory-motor polyradiculoneuropathy consistent with blocked Guillain syndrome. The study of cerebrospinal fluid (CSF) revealed cytoproteinorrhagic dissociation.

The decision was to put the patient on intravenous corticosteroid therapy and discuss the infusion of immunoglobulins but the patient died on day

1 of intravenous corticosteroid therapy following cardiopulmonary arrest.

DISCUSSION

In our case, the diagnosis of Guillain-Barré Syndrome was retained in front of the clinical and paraclinical arguments (EMG, CSF), the infectious origin was eliminated in front of negative serologies (HVB, HVC, HIV, CMV, EBV, HSV, VZV) and vaccine and drug origin are eliminated during the interview.

The combination of acute polyradiculoneuropathy and inflammatory bowel disease is a rare occurrence, regardless of factors such as the intestinal absorption deficit of vitamin B12 or the neurotoxicity of drugs used to treat inflammatory bowel disease.

D'après Une étude rétrospective menée par Larrodé P *et al*. [4] sur une série de quatre patients avec cette association, rapportent que dans tous les cas, la polynévrite a suivi une évolution parallèle à la maladie inflammatoire de l'intestin, étant aiguë et réversible dans deux cas.

According to a retrospective study conducted by Larrodé P and *et al.* [4] on a series of four patients with this combination, report that in all cases, polyneuritis followed a parallel evolution to inflammatory bowel disease, being acute and reversible in two cases. Polyneuropathy could be attributed to a vitamin B12 deficiency in one case and to the neurotoxicity of metronidazole in another; in the two remaining cases, the polyneuropathy was chronic and no etiological factor was found, except for its own activity of inflammatory bowel disease

However, few studies have reported the association Guillain-Barré and chronic inflammatory bowel disease in particular ulcerative colitis.

Zimmerman J et al. [5] describe Guillain-barré Syndrome as an extra-intestinal manifestation of ulcerative colitis observed in two patients while the colitis was in remission. Patients were treated with corticosteroids and recovered.

Another case reported by Liu Z [1] has Guillain-Barré syndrome during the period of remission of ulcerative colitis. The clinical manifestations were numbness of the upper limbs, weakness of the limbs and the incapacity of the fingers. The diagnosis was made before the albumino-cytological dissociation of CSF and a neurogenic lesion with EMG. The patient was treated with methylprednisolone IV with good evolution and disappearance of neurological signs.

Other individual cases have been described by Higelmo M [6] and Roca B [7] as Guillain-barré

syndrome is an neurologic manifestation of ulcerative colitis based on the same paraclinical arguments (LCR and EMG) for the identification of neurological involvement.

The association Guillain-Barré and ulcerative colitis is described in the context of autoimmune causality but in other cases it is secondary to the treatments used in IBD in particular Anti TNF. A case of Guillain-Barré was reported in 2009 secondary to Anti TNF- α in a patient with severe spondyloarthropathy secondary to ulcerative colitis, in this case the neurological involvement was druginduced [8].

Conclusion

The association Guillain-Barré and ulcerative colitis has been rarely described and observed but it is possible, it is suggested from our discussion and our case reported that the Guillain-barré is part of the extraintestinal manifestations of ulcerative colitis hence the need for diagnostic vigilance in cases of ulcerative colitis involving peripheral nerves during the period of remission. We recommend cerebrospinal fluid examination and electromyography for cerebral palsy given the rare but serious risk of Guillain-Barré syndrome.

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