

9. Judd S, Miller L, Antaki F. Symptomatic calculi in a remnant gallbladder: a rare cause of post-cholecystectomy syndrome and biliary pancreatitis. *Endoscopy*. 2014;46 Suppl. 1:E67–8.

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2444-3824/

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Foot drop during adalimumab therapy for Crohn disease[☆]



Desarrollo de pie caído durante el tratamiento con adalimumab en enfermedad de Crohn

To the Editor:

Common peroneal nerve (CPN) palsy is the most common lower extremity mononeuropathy, but it is a rare complication of anti-tumour necrosis factor- α (anti-TNF- α) drugs.

We present the case of a 44-year-old man who had been diagnosed with Crohn disease, with extensive small intestine involvement (Montreal classification A2, L1+L4, B1), more than 8 years previously. At the time of consultation he was in remission, and had been taking adalimumab 40 mg every 14 days as monotherapy for the last 4 years. After multiple sclerosis (MS) had been ruled out by neurology specialists using the McDonald criteria, he attended the rehabilitation clinic for left foot drop (loss of dorsiflexion and eversion of the ankle) that had commenced 3 months earlier, with no history of trauma. Directed physical examination revealed a motor deficit in the dorsal flexion and eversion of the left ankle (1/5 on the modified Medical Research Council scale for muscle strength), and he received an ankle-foot orthosis for ambulation. An electromyogram showed severe neurapraxia-axonotmesis of the left CPN with no voluntary activity in the CPN-dependent muscles. In view of these findings, rehabilitation treatment was started, which consisted of 21 sessions of electrostimulation and 9 of kinesitherapy. An improvement in muscle balance was noted in the re-evaluation after this intervention: muscle balance was 3/5, and the patient was able to walk independently with no technical aids, so it was not necessary to modify the biological treatment of his underlying disease.

The question of whether neurological complications are part of the spectrum of extra-intestinal manifestations of inflammatory bowel disease, or simply secondary to nutritional deficiencies or medication use, is widely debated nowadays.¹ These complications are rarely associated with

anti-TNF- α drugs—incidence is estimated to be 0.1% for adalimumab and 0.03% for infliximab.² Of these complications, the most commonly described are development or exacerbation of MS and demyelination of the central nervous system (CNS), with peripheral nerve demyelination being even more uncommon. Furthermore, a clear cause-effect relationship cannot always be established between neuropathies and the use of anti-TNF agents. In a study to evaluate the onset of complications over time in a series of 9 patients who received anti-TNF- α (4 for rheumatologic disorders, 3 for sarcoidosis, 1 for psoriasis and 1 for Crohn disease), most developed neurological deficits within the first 6 months of treatment, although in the case of the Crohn disease, these appeared 38 months after initiation of infliximab.³

Certain predisposing factors may contribute to the onset of CPN palsy, such as weight loss, trauma or continuous compression (habit of crossing one's legs).⁴ Electromyography is the gold standard for diagnosis.

Although it has been established that the use of anti-TNF- α should be avoided in patients with a history of MS or demyelinating diseases of the CNS,^{5,6} there is no consensus on their use when neurological disorders develop during administration of these drugs. The course of the neuropathy is usually unpredictable, so the risk of continuing anti-TNF treatment should be weighed up on an individual basis against the benefit it provides to the underlying disease.⁸ Both maintenance of treatment³ and a reduction in the anti-TNF dose have been described, so discontinuation of this drug should be limited to patients with controlled underlying disease and incapacitating neuropathy.⁷

Other therapeutic options in the case of peripheral demyelinating neuropathies are corticosteroids, cyclophosphamide, intravenous immunoglobulins or plasmapheresis. In the case presented, the improvement in the Crohn disease under immunomodulatory treatment prompted us to choose conservative management. This achieved a good outcome, and therefore could be a therapeutic option in similar individual cases.

References

1. Gondim Fde A, de Oliveira GR, Teles BC, Aquino Pde S, Brasil ÉF, Carvalho AM, et al. Clinical and electrodiagnostic findings in patients with peripheral neuropathy and inflammatory bowel disease. *Inflamm Bowel Dis*. 2015;21:2123–9.

[☆] Please cite this article as: Collazo Diéguez M, González Carrera V. Desarrollo de pie caído durante el tratamiento con adalimumab en enfermedad de Crohn. *Gastroenterol Hepatol*. 2016;39:724–725.

2. Tristano AG. Neurological adverse events associated with anti-tumor necrosis factor α treatment. *J Neurol*. 2010;257: 1421–31.
3. Nozaki K, Silver RM, Stickler DE, Abou-Fayssal NG, Giglio P, Kamen DL, et al. Neurological deficits during treatment with tumor necrosis factor-alpha antagonists. *Am J Med Sci*. 2011;342: 352–5.
4. Malas FU, Ozçakar L, Fırat F, Kuyumcu ME, Ateş O, Sivri B. Drop foot in Crohn's disease: reported nadir incidence of peroneal neuropathy. *Inflamm Bowel Dis*. 2007;13:1586–7.
5. Mohan N, Edwards ET, Cupps TR, Oliverio PJ, Sandberg G, Crayton H, et al. Demyelination occurring during anti-tumor necrosis factor alpha therapy for inflammatory arthritides. *Arthritis Rheum*. 2001;44:2862–9.
6. Gómez-Gallego M, Meca-Lallana J, Fernández-Barreiro A. Multiple sclerosis onset during etanercept treatment. *Eur Neurol*. 2008;59:91–3.
7. Lozeron P, Denier C, Lacroix C, Adams D. Long-term course of demyelinating neuropathies occurring during tumor

necrosis factor-alpha-blocker therapy. *Arch Neurol*. 2009;66: 490–7.

8. Solomon AJ, Spain RI, Kruer MC, Bourdette D. Inflammatory neurological disease in patients treated with tumor necrosis factor alpha inhibitors. *Mult Scler*. 2011;17:1472–87.

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2444-3824/

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