Lewis-Sumner syndrome associated with infliximab therapy for psoriasis *

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Dear Editor,

The introduction of tumor necrosis factor immunobiological blocking agents (anti-TNF) has brought great advances in the treatment of autoimmune diseases. Currently, there are five anti-TNF drugs available (infliximab, adalimumab, etanercept, golimumab, and certolizumab pegol), which can be used in the treatment of rheumatoid arthritis, ankylosing spondylitis, ulcerative colitis, Crohn's disease, or psoriasis. Although they are generally well tolerated, there are some infrequent, but well-documented, cases reported of serious adverse events, such as systemic lupus erythematosus and vasculitis induction. Neurologic complications are rare and include central and peripheral demyelinating disorders, such as multiple sclerosis, optic neuritis, Guillain-Barré syndrome, chronic inflammatory demyelinating polyneuropathy, and multifocal motor neuropathy.[1] We describe the clinical case of a 40-year old male patient who developed Lewis-Sumner syndrome (LSS), a clinical variant of multifocal acquired demyelinating sensory and motor (MADSAM) neuropathy after the use of infliximab in the treatment of psoriasis.

Patient with severe psoriasis for 21 years, using infliximab in the past 13 months, and with no personal or family history of demyelinating disease, he was admitted to the emergency room reporting muscular pain when climbing stairs four days after his last infusion of infliximab. The pain gradually evolved to a loss of foot dorsiflexion, numbness on the left side of the body, decreased strength in the anterior region of the left forearm and left hand, followed by walking difficulty due to lower limb weakness, and worsening of motor deficit of the right side. On neurological examination, Mingazzini test was positive for the left lower limbs. We noted decreased strength of the extremities, especially on the left lower limb. Cranial nerves were preserved. We also noted lower limb areflexia and loss of superficial sensitivity on the left toes. The patient walked with a paraparetic and ataxic gait due to the loss of sensitivity. He denied headache, visual changes, diplopia, dizziness, seizures and recent fever. Laboratory tests — assessment of renal function, potassium and supplement dosage, protein electrophoresis, and serology for syphilis, hepatitis B, hepatitis C, HIV, and cytomegalovirus — showed no changes. Lumbar puncture showed protein concentration with protein-cytologic dissociation, and electromyography revealed multifocal sensory-motor impairment with associated motor conduction block. The findings were consistent with LSS. The patient was initially treated with pulse methylprednisolone therapy with no improvement. We then initiated the administration of intravenous human immunoglobulin 2g/kg for five days (0.4 mg/kg/daily) with no adverse effects and improvement of neurological deficit. The patient was discharged with suspension of infliximab, keeping only topical treatment for psoriasis until the evaluation of the introduction of ustekinumab. He was also referred to physical and occupational therapies, and oriented to attend the peripheral neuropathy clinic for follow-up treatment, with monthly rehospitalization for at least three months for infusion of human immunoglobulin as maintenance treatment.

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LSS was first described by Lewis *et al.*[1] in 1982 and is characterized by a multifocal and asymmetrical acquired immune-mediated sensory and motor neuropathy. Clinically, it is associated with predominantly distal asymmetrical weakness, mainly affecting the upper and/or lower limbs with sensory deficits and persistent multifocal nerve conduction block. Its association with anti-TNFs is still poorly described. To date, only six cases were reported, all of which with the use of infliximab – 3 patients with Crohn's disease,[2,3] 2 patients with rheumatoid arthritis,[1] and 1 patient with ulcerative colitis.[4] The case described here is the first in a patient with psoriasis. The duration of treatment with infliximab in those cases varied between 3-9 months and the appearance of neurological symptoms could be observed between 3-8 weeks after the last infusion.[1-4] In our case, infliximab use was slightly longer (13 months), but the onset of symptoms was much earlier (4 days).

We suggest that this neuropathy may be triggered by auto-antibodies that recognize epitopes on peripheral nerves induced by infliximab.[1] Antiganglioside antibodies (generally IgG) have been described in LSS.[5] However, it is questionable whether these antibodies would be pathogenic or an epiphenomenon secondary to nerve inflammation.[1]

These data show that, although rare, demyelinating diseases may complicate the course of treatment with anti-TNF drugs, and physicians should be aware of their signs and symptoms.❑

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