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LEWIS-SUMNER SYNDROME AS A MANIFESTATION OF NEUROPSYCHIATRIC SYSTEMIC LUPUS ERYTHEMATOSUS

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BACKGROUND

Lewis–Sumner syndrome is a multifocal acquired demyelinating sensory and motor neuropathy (MADSAM) characterized by asymmetric involvement, with weakness of distal predominance, with evolution from months to years, categorized as a variant of chronic inflammatory demyelinating polyneuropathy (CIDP). Among the 19 neuropsychiatric manifestations of systemic lupus erythematosus (SLE) classified by the American College of Rheumatology, 7 are peripheral manifestations; however, CIDP and its variants are not considered.

CASE REPORT

Female patient, 38 years old, diagnosed with SLE, presented with reduction strength in her right foot for 3 months, with difficulty in dorsiflexion, associated with pain and local paresthesia and in left hemiface, with worsening in the period nocturnal and metacarpophalangeal and ankle arthritis. No respiratory complaints, dysphagia, fecal incontinence or urination. On physical examination, decreased sensitivity in branches V2 and V3 of the left trigeminal nerve, reduced muscle strength in the right lower limb using the Medical Research Council (dorsal interossei: grade 3/5, iliopsoas: grade 3/5, quadriceps: grade 3/5, hamstrings: grade 3/5, anterior tibial: grade 2/5, sural triceps: grade 3/5 and long hallucis extensor: grade 2/5), tactile-painful hypoesthesia in L4-L5 topography more pronounced on the right and distal hypoesthesia of the right lower limb. Globally reduced muscle reflexes. Laboratory tests with lymphopenia, anti-DNA reagent (1/160), consumption of complement and increased C-reactive protein. Renal and hepatic function tests, glycaemia, vitamin B12 and folate levels and thyroid function tests were within normal ranges. Viral hepatitis and infection by the acquired immunodeficiency virus were excluded by serological tests. Electroneuromyography with sensory-motor multineuropathy of myelin predominance and with conduction block, suggestive of Lewis–Sumner syndrome. Pulse therapy with methylprednisolone 3 g and intravenous cyclophosphamide was started, with improvement.

CONCLUSION

Chronic inflammatory demyelinating polyneuropathy is the most common demyelinating neuropathy in the general population, but rare in SLE. Treatment depends on the presence of systemic manifestations of SLE. In the case of active SLE, cyclophosphamide is a good option. If it occurs in isolation, immunoglobulin should be the first option. There is little data in the literature on the treatment of CIDP in SLE.