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Recurrent Miller Fisher syndrome: Case report☆

Recurrencia del síndrome de Miller Fisher: descripción de un caso

Dear Editor:

The term Miller Fisher syndrome (MFS) refers to the clinical spectrum described in 1932 by Cullier, characterised by ataxia, ophthalmoparesis, and areflexia; it is considered an infrequent variant of Guillain-Barré syndrome.[1—5] Its aetiology is considered to be autoimmune. MFS is caused by a cross-reaction, after history of a respiratory or gastrointestinal infection or vaccination, to the GQ1b antigens present in the axons of oculomotor nerves, neurons of the posterior spinal root ganglia, and neuromuscular spindles.[2,3] Anti-GQ1b antiganglioside antibodies are detected in 80% of patients.[1—4] Annual incidence is between 1% and 5% of all cases of GBS spectrum.[3,6] Treatment includes intravenous (IV) immunoglobulins (Ig) or plasmapheresis, and outcomes are frequently favourable. In most cases, the clinical course is monophasic, with recurrences being very rare (approximately 5% of cases).[1,4,5]

We present the case of a 21-year-old man with recurrent MFS and history of seborrheic dermatitis and hyperhomocysteinaemia. In 2018, one week after manifesting tonsillitis and diarrhoea, treated with amoxicillin/clavulanic acid, he was admitted due to gait instability and binocular diplopia in the horizontal plane, progressing for less than 24 hours, with no dysautonomic signs. The patient presented right palpebral ptosis, bilateral limited upgaze, paralysis of the right medial and lateral rectus muscles, hypoactive deep tendon reflexes in the upper limbs and abolished deep tendon reflexes in the lower limbs, preserved positional and arthrokinetic sensitivity, and reduced distal vibration sensitivity in the left lower limb, with ataxic gait. Analysis of a cerebrospinal fluid (CSF) sample collected on the day of admission only revealed an increased protein level (34 mg/dL; normal range: 8—32 mg/dL). Serology results for HIV, *Rickettsia*, *Brucella*, *Coxiella*, and hepatitis C virus yielded negative results. We detected IgG antibodies against the hepatitis B virus surface antigen, with a PCR test yielding negative results. A blood sample collected on day 2 after admission was negative for antiganglioside antibodies (anti-GM1, GM2, GM3, GD1a, GD1b, GT1b, and GQ1b). We detected vitamin B_{12} deficiency (83 pg/mL; normal range: 211—911 pg/dL), which was treated with supplementation. Non-contrast magnetic resonance imaging (MRI) yielded normal results. No spinal MRI scan was performed. A motor nerve conduction study revealed generalised motor sensory axonal polyradiculoneuritis. After diagnosis of MFS, the patient started treatment with IV Ig, which led to pronounced worsening, with ophthalmoplegia and truncal

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ataxia, at 3 days. He then started 12 cycles of plasmapheresis, which improved ophthalmoparesis and ataxia. The patient was asymptomatic at 6 months, without recovery of stretch reflexes (with the exception of hypoactive right brachioradialis reflex).

At 2 years, a week after presenting symptoms of cold without fever, he was admitted due to similar symptoms of gait instability and diplopia, generalised tingling sensation, and difficulty speaking. The examination revealed mild dysarthria, complete ophthalmoplegia, universal areflexia, ataxic gait, left-sided dysmetria on the finger-to-nose test, and preserved deep sensitivity, with no dysautonomic signs. Complementary testing yielded normal or negative results, including PCR testing for SARS-CoV-2 in nasopharyngeal aspirate, motor nerve conduction study (performed at 48 hours after symptom onset and repeated after 2 weeks), non-contrast brain MRI scan, and antiganglioside antibody determination, with no changes with respect to previous serology tests. He received treatment with Ig for 5 days, which improved symptoms, and was discharged with mild ophthalmoparesis.

Recurrence of MFS is infrequent.[1,4,5] We present the case of a patient whose symptoms resolved after a first episode, before experiencing a relapse 2 years later, with similar symptoms and an infectious trigger in both episodes. We should highlight the negative results for antiganglioside antibodies during both admissions, as well as the normal findings in the motor nerve conduction study performed during the second admission. This lack of pathological findings has previously been described in other cases of recurrence,[4,7] and may be explained by the early performance of the study.[7,8]

In other reported cases of recurrent GBS spectrum disorders, symptoms are usually more severe in the second episode,[9] which was not the case with our patient. He also presented an excellent response to Ig during the recurrence, unlike in the first episode. We do not know the reason for the lesser severity and better response to Ig during the relapse, although they may be related to immunological factors, as well as aetiological factors of the precipitating agent.

The causes predisposing to recurrent GBS are unclear. Patients younger than 30 years, with moderate impairment and MFS phenotype, are reported to present a higher risk of recurrence.[9] While the reasons for this are not clear, some human leukocyte antigens (HLA-Cw3 and HLA-DR2), as well as some immunological factors, are reported to be involved.[4,6,9,10]

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COVID-19 related strokes: *Pandora's Box may open as the p(c)lot thickens*!

Ictus y COVID-19: una nueva caja de Pandora

Dear Editor,

As I read through the interesting literature focusing on COVID-19 related strokes, featured recently in the *Journal*,[1,2] my inquisitiveness regarding the involved mechanisms of neuro-invasion escalates. Moreover, as the COVID-related 'clot' thickens (a result of the pro-inflammatory hypercoagulable mileu), almost in close conjunction does the 'plot' thicken interrogating whether the direct consequences of disseminated intravascular coagulation can alone explain the intriguing findings of most COVID-associated strokes being ischemic in nature with many researchers also suggesting these cerebrovascular events to be unrelated to age and associated with few vascular risk factors.[1—5]

In this context, while the thrombo-embolic sequel of COVID-19 is being ardently discussed,[1—4] paradoxical embolization into the cerebral circulation through an undetected probe patent foramen ovale (PPFO) (reported to be as common as 20—25% in the general population), can very well be an important contributing factor.[6] The premise for