International Journal of Health Science

CASE OF ACUTE DISSEMINATED ENCEPHALITIS WITH GUILLAIN-BARRÉ SYNDROME IN AN ADULT

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CASE PRESENTATION

A 54-year-old male patient, after having dinner with typical Chinese foods, started, on January 19, 2019, with high parietal headache, bilateral, pressure-type, of moderate intensity, daily, without phonophobia, photophobia, nausea and/or vomiting . On February 2nd, he developed acute ascending paraparesis and paresthesia, like tingling, in the lower limbs, presenting soon after with temporalspatial disorientation, acute urinary retention, dysphagia for solids and liquids, facial paralysis on the right and subsequent motor aphasia. Afterwards, he evolved with a lowering of the level of consciousness and tetraparesis with respiratory impairment, remaining in the ICU of a Chinese hospital for 15 days. Upon returning to Brazil on September 18, 2020, he was admitted to the Hospital Universitário Getúlio Vargas (HUGV).

On the neurological examination, he was oriented, with paraplegia in the lower limbs associated with hyperreflexia, tactile hypoesthesia level V1 on the right, with anesthesia level T11 bilaterally, anarthresthesia in the lower limbs, hypopalesthesia level T10 bilaterally and apalesthesia level T12 on the right, with intention tremor and posture with predominance to the left. Visual acuity 20/400 and bilateral dyschromatopsia. Fundoscopy evidenced bilateral ON pallor. Campimetry and MOE preserved.

During hospitalization, he underwent a neuraxial MRI showing a pseudotumoral lesion in the left frontotemporal region and longitudinally extensive myelitis from T4 to T12, serum levels of Anti-GM1, Anti-GD1 and anti-MOG (all positive), being diagnosed with Guillain-Syndrome. Barré and Acute disseminated encephalomyelitis secondary to anti-MOG antibody.

He underwent treatment with acyclovir, immunoglobulin for 5 days and methylpednisolone during hospitalization in

a Chinese hospital. After returning from the coma, treatment with azathioprine 100 mg/ day and prednisone 20 mg/day was started, remaining on this treatment until September 18, 2020, when he returned to Brazil and suspended the medication. During the evaluation in the HUGV ward, the patient remained with urinary retention and chronic constipation, and was advised to return to azathioprine 200 mg/day and prednisone 1 mg/Kg/day and requested imaging and laboratory tests.

DISCUSSION

Acute Disseminated Encephalomyelitis (ADEM) is a rare autoimmune syndrome of the central nervous system (CNS) that courses with inflammation and demyelination. It manifests acutely or subacutely, through encephalopathy and multifocal neurological Oligodendrocyte deficits. glycoprotein (MOG) is an important protein involved in the myelination process of the CNS. Anti-MOG autoantibodies are associated with a broad spectrum of CNS demyelinating diseases, such as ADEM, Optic neuritis (NO), Transverse myelitis (TM), as well as Neuromyelitis optica and the Spectrum of neuromyelitis 2،4 ۰6 Guillain-Barré optica diseases. Syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy and is an autoimmune disease. Its development has been highly associated with post-infectious autoimmune responses against gangliosides in peripheral nerves. Autoimmune antibodies that recognize gangliosides are found in a large proportion of GBS patients (62%) and are believed to contribute to the final pathology by inducing complement-mediated axonal injury and demyelination.⁵ Acute encephalomyelitis disseminated (ADEM) and Guillain-Barré syndrome (GBS) are commonly recognized as separate entities involving different parts of the nervous system.

However, they share some characteristics, such as: autoimmune pathogenesis, myelin damage, and previous history of viral infections or vaccination^{1/3}.

We describe the case of a 54-year-old patient with an initial picture of acute ascending paraparesis and tingling-like paresthesia in the lower limbs, who evolved with temporalspatial disorientation, acute urinary retention, dysphagia for solids and liquids, facial paralysis on the right and posterior motor aphasia. . Afterwards, he presented lowering of the level of consciousness and tetraparesis with respiratory impairment, remaining in the ICU for 15 days. Based on the temporal association and semiology showing involvement of the central (CNS) and peripheral systems, the diagnosis of GBS and ADEM was made, confirmed by the presence of anti-GM1 and anti-MOG antibodies.

CONCLUSION

The importance of this case report resides in the fact that the concurrence of ADEM and GBS is rare, however, it should be investigated in post-infectious conditions followed by CNS and Peripheral involvement.

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