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MYELIN OLIGODENDROCYTE GLYCOPROTEIN ANTIBODY-ASSOCIATED DISEASE (MOGAD) AFTER RECENT COVID-19 INFECTION AND COVID-19 VACCINATION: A CASE REPORT

Hiago Antunis Silva
Samuel Libânio Clinical Hospital - Pouso Alegre

Patricia Marques Mendes
Samuel Libânio Clinical Hospital - Pouso Alegre

Yan da Silva Raposo
Samuel Libânio Clinical Hospital - Pouso Alegre

Ilana Werneck Augsten
Samuel Libânio Clinical Hospital - Pouso Alegre

Maycon Melo Lopes
Samuel Libânio Clinical Hospital - Pouso Alegre

Hiara Gabrielle Silva
Samuel Libânio Clinical Hospital - Pouso Alegre

Presentation of the case: Female patient, 53 years old, previously diabetic, hypertensive, and dyslipidemic; with a clinical picture of gradual visual loss installation of central predominance in the left eye (LE), pain at ocular mobilization in the LE, and cephalea. On physical examination, she presented visual acuity (VA) of counting fingers in the LE, presence of a relative afferent pupillary defect in the LE, papilledema in the LE on dilated eye examination, and remaining neurological examination without alterations. Symptoms began 22 days after the first dose of the COVID-19 vaccine (AstraZeneca) and 15 days after COVID-19 infection (SARSCOV-2 antigen reagent test) with bilateral mild-moderate lung impairment. She denies previous neurological events. She was admitted to the Neurology Department of Samuel Libânio Clinical Hospital in Pouso Alegre - Minas Gerais, with laboratory tests presenting serologies for COVID-19 IgM Reagent and IgG Reagent, increased PCR, other general tests without alterations (blood count, thyroid, hepatic and renal functions), other infectious serologies without acute conditions (HIV, arboviruses, Herpes simplex virus 1 and 2, cytomegalovirus, toxoplasmosis, VDRL, Hepatitis B, and C), negative rheumatological tests, and liquor with a normal result (including serologies and infectious cultures). Magnetic resonance imaging (MRI) of the orbits shows signs of optic neuritis on the left, with paramagnetic contrast enhancement, without extension for optic chiasm. The MRI of the brain, cervical and thoracic spine was performed without any relevant changes. Opted for pulse therapy with methylprednisolone one gram/day for five days, and the patient evolved with partial recovery of VA in the LE (20/200). Due to atypical optic neuritis (acute loss of VA, poor recovery with corticosteroid therapy, age of the patient), serum tests of antiaquaporin 4 antibody with negative result and anti-MOG antibody with positive result were requested. Was performed a visual evoked potential evidencing demyelinating process in the pre-chiasmatic visual pathways of the LE. In view of the findings, a diagnosis of Myelin Oligodendrocyte Glycoprotein Antibody-associated Disease (MOGAD) was made, and subsequently, azathioprine was prescribed associated with corticosteroid therapy, without new neurological events and with progressive improvement of the visual picture. **Discussion and Conclusion:** The aforementioned patient presented a picture of optic neuritis in the LE with diagnostic possibilities of post-vaccination optic neuritis, post-infectious COVID-19 or first event of demyelinating disease. The visual evoked potential with signs of demyelinating optic neuritis and the positivity of the anti-MOG antibody contributed to the diagnosis of MOGAD and probably the previous infection and/or previous vaccination worked as triggers for the manifestation of the autoimmune disease. Although the diagnostic criteria for MOGAD are not yet well defined, the clinical manifestations in association with the positivity of the anti-MOG antibody assist in its diagnosis, as well as the exclusion of other conditions.