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COMPREHENSIVE WORK-UP FOR ACUTE OPHTHALMOPARESIS WITH ELEVATED GQ1B ANTIBODIES IS WARRANTED IN TIMES OF THE PANDEMIC

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Letter to the Editor

With interest we read the article by Garg et al. about a 24 years old male with sudden-onset, painless ophthalmoplegia without ataxia, areflexia, or limb weakness (acute ophthalmoparesis (AO)) who tested positive for GQ1b [1]. The patient profited from methyl-prednisolone [1]. It was concluded that AO with positive anti-GQ1b antibodies is a rare immune-mediated syndrome, which can be cost-effectively treated with steroids [1]. We have the following comments and concerns.

The main shortcoming is that the patient has not been tested for SARS-CoV-2. Though SARS-CoV-2 infected patients with OA have not been reported, several patients with Miller-Fisher syndrome (MFS) with or without affection of other cranial nerves or the limb or respiratory muscles have been published. MFS has been reported as a manifestation of neuro-COVID in a 45 years old patient who was immuno-suppressed prior to the SARS-CoV-2 infection [2]. The patient developed limb weakness after onset of MFS during the further disease course [2]. MFS as a manifestation of a SARS-CoV-2 infection was also reported in a 31 years old male with limb weakness already two months prior to onset of MFS [3]. In addition to cranial nerves VI, cranial nerves VII and XII were compromised [3]. Two other patients with cranial nerve involvement have been reported by Pascual-Goni et al. [4]. In a 36 years old male with SARS-CoV-2 associated MFS, MRI of the orbita and the retro-orbital region was notable for striking enlargement, prominent enhancement with gadolinium, and T2-hyperintensity of the left oculomotor nerve [5]. Several other COVID-19 patients with affection of cranial nerves have been published. CSF findings in patients with SARS-CoV-2 associated cranial nerve involvement is usually normal [4].

A further shortcoming is that no second spinal tap was carried out during follow-up. Cerebro-spinal fluid (CSF) protein may be normal at onset of Guillain Barre syndrome (GBS) to become elevated during the disease course [6].

Missing is a cerebral MRI to rule out Bickerstaff brainstem encephalitis (BBE) or hyperintensities of cranial nerves III, IV, and VI, as has been previoulsy reported [5].

AO is usually preceeded by infectious disease. In a study of 21 patients with AO, 17 had a history of an antecedent infection [7]. Thus, the index patient should have been tested for antibodies against campylobacter jejuni. Gastro-intestinal infections with this agent frequently precede the development of GBS. Infection with campylobacter jejunii may even go asymptomatic, why it is crucial to test patients with GBS propsectively for this agent.

GQ1b antibodies may not only be elevated in MFS but also in other subtypes of GBS, such as AO, acute ptosis, acute mydriasis, acute oropharyngeal palsy and acute ataxic neuropathy (AAN), pharyngeal-cervical-brachial (PCB) weakness, BBE, MFS/GBS overlap, and BBE/GBS overlap [8]. GQ1b antibodies are also elevated in post-infectious GQ1b antibody syndrome presenting with optic neuritis and palatal dysarthria [9].

Since cases of AO with spontaneous recovery have been reported [10], it cannot be excluded that complete recovery in the index patient was due to spontaneous recovery and not related to the application of steroids.

Overall, the presented case report is appealing but has several limitations which should be met before drawing final conclusions. Neuro-COVID needs to be excluded, follow-up CSF investigations and cerebral MRI should have been done, and antibdodies against campylobacter jejuni should have been determined. Spontanoeus recovery in the index patient should be considered.

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