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Before Attributing Polyradiculitis to an Anti-SARS-CoV-2 Vaccine, Differential Etiologies need to be Ruled Out

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Dear editor,

We read with interest the article by Khadka et al. about a 44 years-old male who developed Guillain-Barre syndrome (GBS) 9 days after receiving the Johnson and Johnson anti-SARS-CoV-2 vaccine (JJV).[1] The patient presented with paraparesis, paresthesias, and reduced tendon reflexes of the lower limbs.[1] Cerebrospinal fluid (CSF) investigations showed dissociation cyto-albuminique and nerve conduction study (NCS) showed demyelinating neuropathy.[1] Acute, inflammatory demyelinating polyneuropathy (AIDP) was diagnosed and the patient benefited from intravenous immunoglobulins (IVIG).[1] The study is attractive but raises concerns that should be discussed.

Clinical neurologic exam on admission was described as normal regarding both upper limbs.[1] However, "nerve conduction study (NCS) showed electrophysiological evidence of sensorimotor axono-myelinic polyneuropathy of severe severity on both arms and legs, and prolonged F-waves".[1] An explanation should be provided for the discrepancy between severely reduced nerve conduction in upper limb nerves but normal muscle force on clinical exam.

The rationale for applying a pre-medication with intravenous paracetamol, chlor-phenaramine, ranitidine and hydrocortisone prior to IVIG is not comprehensible. We should be informed about the indication for giving these drugs. For how long were these drugs given and why were steroids added to IVIG. From steroids it is well known that they are ineffective for GBS.[2]

CFS studies in patients with SARS-CoV-2 associated GBS have shown that cytokines, chemokines, or glial factors can be elevated in the CSF. Upregulation of the chemokine CXCL5, and of the interleukines IL-2, IL-3, and IL-8 have been recently demonstrated in a patient experiencing acute, necrotising encephalopathy following a vaccination with the ChAdOx1 (Astra Zeneca vaccine (AZV)).[3] We should be informed if CSF cytokines or chemokines were elevated in the index patient. Additionally, we should know if ganglioside antibodies were elevated in the serum.

Since it is well-known that SARS-CoV-2 infections can be complicated by GBS,[4] an acute SARS-CoV-2 infection has to be ruled out before establishing a causal relation between vaccination and GBS.

We should be told if contrast medium was applied for the spinal MRI. In the acute stage, enhancing lumbar radices could be seen on MRI of the lumbar spine.[5]

Overall, the interesting study has some limitations and inconsistencies that call the results and their interpretation into question. Addressing these limitations could further strengthen and reinforce the statement of the study. Differential causes of GBS need to be ruled out before attributing GBS to the vaccination.

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