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Letters to the editor

Diagnosing SARS-CoV-2 vaccination associated aseptic meningitis is intricate



We read with interest the case report by Reis Carneiro et al. about a 62 years old female who developed fatigue, impaired concentration, dizziness, myalgia, unstable gait, and mild headache one day after the first jab with the BNT162b-Pfizer anti-SARS-CoV-2 vaccine [1]. Upon the clinical presentation and pleocytosis in the absence of documenting an infectious agent, aseptic meningitis was diagnosed [1]. Under steroids complete recovery was achieved [1]. The study is appealing but raises comments and concerns.

We do not agree with the diagnosis aseptic meningitis [1]. Since the erythrocyte count was high with each of the spinal taps, contamination of the specimen with peripheral blood leucocytes is quite likely and thus a false positive leukocyte count in the cerebro-spinal fluid (CSF) cannot be excluded.

Missing are the time points at which the three spinal taps were taken. To assess if steroids were truly effective, it is crucial to know which of the three spinal taps have been done before and which after initiation of steroids.

Missing are the results of the magnetic resonance venography (MRV). Since SARS-CoV-2 vaccinations can be complicated by venous sinus thrombosis (VST) [2], and since the clinical presentation is also compatible with VST, it is essential that VST had been appropriately excluded by MRV and determination of the D-dimer.

Missing are the profiles of the cytokines and chemokines in the CSF. Since cytokines and glial markers can be elevated or reduced in immune-reactions to a SARS-CoV-2 vaccination [3], it would be helpful to know the titres of these markers in the CSF.

We also disagree with the notion that the index patient had postural tachycardia [1]. Tachycardia is defined as a heart rate > 100. However, the patient presented with heart rates < 100.

The patient had myalgias, which do not comply with the diagnosis of a septic meningitis. A pathophysiological explanation of the myalgias should be provided.

There is a discrepancy between the statement “there was no fever or systemic complaints” and the symptoms fatigue and myalgia [1]. Fatigue and myalgia are regarded as systemic symptoms.

There is a discrepancy between the normal clinical, neurologic examination and the symptom “unstable gait”.

We should know if the patient had a gait disturbance or not.

It is not mentioned if the cerebral MRI was carried out with or without contrast medium. Application of contrast medium is crucial to exclude encephalitis and whether the meninges enhanced or did not.

Overall, the interesting study has limitations which challenge the results and their interpretation. Addressing these limitations would strengthen the conclusion.

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Author contribution

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Competing interests

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Ethical approval and consent to participate

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Consent for Publication

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Availability of data and material

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COVID-19: A putative trigger for neuralgic amyotrophy



A 63-year-old man with medical history of diabetes mellitus presented with acute left shoulder pain that began three weeks after a first COVID-19 vaccination (AZD1222) in the left deltoid. The pain was severe, restricted to the left deltoid region and lasted for about three to four days. One week later, he developed left shoulder abduction weakness (Medical Research Council scale 1/5) and left arm external rotation defect, respectively related to supraspinatus and infraspinatus motor deficit, with no passive movement limitation. Neuralgic amyotrophy (NA) was retrospectively evoked when overt amyotrophy of the infraspinatus fossa and dynamic discrete scapular winging appeared. Magnetic resonance imaging (MRI) performed six weeks after pain onset found Short TI Inversion Recovery (STIR) hypersignal in the homolateral supraspinatus and infraspinatus muscles that evoked muscle edema.

The first electrodiagnostic (EDX) examination performed six weeks after onset revealed acute denervation in both left upper trapezius and anterior serratus muscles affirming spinal and long thoracic nerve injury in addition to supra-scapular nerve lesion. Needle examination performed two months after onset, as part of EDX testing, revealed an acute denervation pattern in the left infraspinatus and supraspinatus muscles, respectively total and with early reinnervation signs, consistent with NA diagnosis. Given the late diagnosis, no steroids were administered. Physiotherapy was recommended. The patient received the second injection of the COVID-19 vaccination (AZD1222) with no additional side effect.

In the absence of any other potential cause, our patient's NA was considered as possibly related to COVID-19 vaccination. SARS-Cov2 is responsible for neurological manifestations from imprecise mechanisms. It is unclear whether it is related to molecular mimicry, small vessel vasculitis secondary to the cytokine release, hypercoagulable state, auto-immunity, direct effect of the virus, or several of these mechanisms combined [1]. Siepmann et al. discuss possible singularity of COVID-19-associated NA, more likely to cause pure sensory involvement, which remains to be confirmed [2]. The incidence of post-vaccination NA is considered to be very rare. A few cases of NA occurring after COVID-19 vaccination have been reported so far [3]. Previously, NA cases following seasonal influenza vaccination have been reported (vaccine adverse effect reporting system database – VAERS). Exact relationships between NA and vaccination remain unproven but it could be in favor of some immunological mechanism as a trigger of NA. As demonstrated in our case and in the literature, paraclinical confirmation of NA (especially when a differential diagnosis is suspected) can be provided by different modalities, such as EDX or MRI [4,5]. In conclusion, physicians working in the field of COVID-19 should be aware of possible post-vaccination NA.

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