Letter to the Editor: Pre-Existing Neuropathy Favours SARS-CoV-2 Vaccination Associated Guillain-Barre Syndrome

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Dear Editor,

We read with interest the article by Kim et al. about two patients with SARS-CoV-2 vaccination associated Guillain Bare syndrome (SC2VG). Patient-1 was a 42-year-old male who developed bilateral facial palsy, dysphagia, quadriparesis, and respiratory insufficiency requiring intubation and mechanical ventilation 2 weeks after the first dose of the AstraZeneca vaccine (AZV).

Upon cerebrospinal fluid (CSF) investigations and nerve conductions studies (NCSs) the patient was diagnosed with GBS received intravenous immunoglobulins, with a beneficial effect.

Patient-2 was a 48-year-old female with a history of diabetes and arterial hypertension who developed left facial palsy, dysarthria, severe myalgia, numbness, and muscle weakness two weeks after the first dose of the Biontech Pfizer vaccine (BPV). SC2VG was suspected and confirmed by CSF and NCSs. IVIG were given with a significant beneficial effect.

The study is appealing but raises concerns that need to be discussed. Patient-1 had received tuberculostatic treatment prior to onset of GBS. Particularly isoniacid and ethambutol are neurotoxic. Though ethambutol predominantly causes optic neuropathy, some cases with neuropathy of peripheral have been reported. Also isoniacid can cause polyneuropathy. We should be told which tuberculostatics the patient received, how many days prior to onset of GBS, for how long, and which dosages. It is essential to know if patient-1 developed side effects to any of the tuberculostatics, particularly sensory or motor dysfunction. Patients with previous neuropathy seem to be prone to develop GBS. It would be also interesting to know if patient-1 had undergone NCSs prior to onset of SARS-CoV-2 vaccination associated GBS (SC2VG).

Missing is the subclassification of GBS in patient-1. Since sensory functions were described as preserved, and since NCSs revealed an axonal lesion in most of the investigated nerves, GBS should be classified as acute, motor, axonal neuropathy (AMAN).

At onset patient-1 had left-sided facial palsy but on admission to the tertiary unit he had bilateral facial palsy. Was this due to progression within a few days or was bilateral facial palsy initially subtle?
We do not agree that the nerve action potential amplitude from the right sural nerve was 42.4 mV respectively 35.0 mV in the left sural nerve as indicated in Table 1. Do the authors mean microV?

Patient-2 had diabetes which did not require treatment. However, the HbA1c values were not presented to assess if blood glucose levels were in the normal range on the long run. We should also know if patient-2 had any indications for diabetic polyneuropathy on NCSs prior to onset of SC2VG.

Missing is the subclassification of GBS in patient-2. Since nerve conduction velocities were largely normal, since compound muscle action potentials were reduced, and since motor and sensory fibers were affected, the most probable GBS subtype is acute, motor and sensory, axonal neuropathy (AMSAN).

Myalgia in patient-2 remained unexplained. Were there any indications for myositis, which has been previously reported as a neurological complication of SARS-CoV-2 vaccinations. Were there any indications for myositis on needle EMG in patient-2? Was the creatine-kinase normal or elevated?

Overall, the interesting study has some limitations and inconsistencies which challenge the results and their interpretation. Addressing these issues would strengthen the conclusions and could increase the status of the study.

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Dear Sir,

Thank you for your interest in our article, “Guillain-Barre Syndrome After Two COVID-19 Vaccinations: Two Case Reports With Follow-up Electrodiagnostic study”. We would like to explain the issues you have raised.

Patient-1 did not have medical record of tuberculosis treatment. He said it was few decades ago and we could not get his exact previous medical record of tuberculosis treatment.

Patient-1 did not have any sign of neurologic deficits before admission. Also he told us this was the first experience of nerve conduction study.

We classified GBS subtypes in patient-1 and patient-2 according to electrodiagnostic classification proposed by Uncini et al. Both patient-1 and patient-2 were classified as acute inflammatory demyelinating polyneuropathy (AIDP) subtype.

At symptom onset, patient-1 showed only left facial palsy. After transmission to our hospital, facial palsy progressed bilaterally. We were not able to know the exact onset of right facial palsy due to drowsy mental state during the ICU care. At the rehabilitation unit, his left facial palsy was partially resolved and right facial palsy was progressively deteriorated.

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Patient-2 had a history of diabetes mellitus but no medication applied. On admission lab, HbA1c was 6.7 and glucose levels were 143 (06:00), 141 (10:00), 160 (15:00), and 195 (20:00). She was applied with Diabex 500 mg bid and at the discharge HbA1c was 6.0, day glucose levels were 95 (06:00), 105 (10:00), 153 (15:00), and 149 (20:00). She was diagnosed as diabetes mellitus a few years ago at a local clinic. But her glucose level was in the normal range and she did not take any medication. Although patient-2 did not undergo nerve conduction study prior to this admission, she did not show any symptoms suggestive of diabetic polynuropathy.

On needle EMG, patient-2 did not show any findings of myopathy such as early recruitment and small polyphasic motor unit action potentials. At the admission, her CK/LDH was 107/224 and ESR was 21, all in the normal range. There were no signs of myositis.

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https://doi.org/10.3346/jkms.2022.37.e217
In Table 1, the unit of sural nerve action potential amplitude was misspelled. MicroV ($\mu$V) is the correct expression. Thank you very much again for your excellent suggestions.

REFERENCES

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