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Letter to the Editor

Before Diagnosing COVID-19-Associated Femoral Nerve Compression due to psoas Hematoma, Causalities and Pathophysiologies Must Be Established

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We were interested to read the article by Alsaqobi *et al.* about a 41-year-old man with severe SARS-CoV-2 infection who required mechanical ventilation and extracorporeal membrane oxygenation (ECMO) complicated by jugular vein thrombosis, psoas hematoma, critically ill neuropathy, and bilateral femoral nerve compression neuropathy [1]. He had a history of diabetes, obesity and asthma [1]. In addition, he was diagnosed with critically ill neuropathy [1]. At the one-year follow-up, he had not fully recovered [1]. The study is excellent, but some points should be discussed.

The first point is that the diagnosis of a psoas hematoma is not convincing. Blood on CT images is usually hyperdense [2]. However, the CT images shown in Figure 1 and Figure 2 do not show hyperdensity with Hounsfield units of 50-60 for whole blood or 60-80 Hounsfield units for coagulated blood. What is shown has at most 10-20 Hounsfield units. This discrepancy should be explained. We should also know what the lesions shown on CT looked like on MRI. Was muscle edema and plexitis of the lumbosacral plexus completely ruled out?

The second point relates to the diagnosis of critically ill neuropathy [1]. As the patient was diabetic, it is essential that the HbA1c values are known on admission and on discharge. Was the diabetes well or poorly controlled? Is it conceivable that the axonal neuropathy was due to a worsening of the diabetic neuropathy rather than a critically ill neuropathy?

The third point is that Guillain-Barre syndrome (GBS) was not considered as a differential diagnosis of axonal neuropathy and was not definitively ruled out. Has the patient undergone cerebrospinal fluid (CSF) testing to detect or rule out cyto-albuminous dissociation? Has proximal neurography been performed to detect a proximal lesion of motor or sensory nerve fibers? As SARS-CoV-2 infections can be complicated by GBS [3], it would have been imperative to exclude this differential diagnosis.

The fourth point is that not all medications administered during the ICU stay were reported. In order to assess whether a particular drug or combination was responsible for the axonal neuropathy, it is essential to know all the drugs administered in detail and which of them were neurotoxic.

The fifth point is that no information was provided as to whether the axonal neuropathy was already present before the SARS-CoV-2 infection or was only exacerbated by the severe COVID-19 infection.

The sixth point is that an isolated femoral compression neuropathy due to a psoas hematoma is unlikely for several reasons. First, a psoas hematoma usually affects not only the femoral nerve in isolation, but the entire lumbosacral plexus [4]. Therefore, if a psoas hematoma was really responsible, it should also have affected the sciatic nerve, the peroneal nerve, the tibial nerve and the obturator nerve. The fact that the patient also complained of sensory disturbances in the lower limbs immediately after the hematoma suggests that the sural nerve and the saphenous nerve were also affected.

The anti-factor Xa value at the time of the suspected haemorrhage is missing. Was the value in the therapeutic range or below? Was the heparinization actually responsible for the drop in haemoglobin?

To summarize, this interesting study has limitations that put the results and their interpretation into perspective. Addressing these limitations could strengthen the conclusions and support the message of the study.

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