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Letter to the Editor

Before an Embolic Infarction of the Cervical Spinal Cord is Diagnosed, Ischemia should be Documented and the Source of the Embolism Identified

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Letter to the Editor

We read with interest the article by Golder *et al*. regarding a 66-year-old female patient presenting with progressive tetraparesis, tetraspasticity, numbness in all four extremities, and bladder dysfunction; the clinical picture was initially interpreted and treated as Guillain-Barré syndrome (GBS) but was subsequently reclassified during the hospital stay as an embolic infarction of the cervical spinal cord (segments C2–C7) [1]. A deep vein thrombosis was suspected as the source of the embolism, given the confirmed presence of a patent foramen ovale (PFO) [1]. Although the study is noteworthy, several points warrant discussion.

A first point concerns the contradictory description of the temporal progression of symptoms and clinical findings [1]. According to the case description, the patient was already experiencing diffuse numbness across her entire body 12 hours prior to presentation [1]. However, the following sentence states that the patient developed ascending paresthesia and weakness in the lower extremities during the night before presentation [1]. Furthermore, there is a discrepancy between the statement that tetraparesis was present upon admission and the report that paresis of the left arm developed later during the hospital stay [1]. Equally contradictory is the report of hyperreflexia at admission, while it is simultaneously noted that the patient developed progressive tetraspasticity over the subsequent days [1]. These inconsistencies should be clarified.

A second point is that the diagnosis of spinal cord infarction is not sufficiently substantiated [1]. The initial MRI of the cervical spine was unremarkable; Evidence of edema in segments C2–C7 became apparent only during a second MRI examination [1]. However, no diffusion-weighted images (DWI) or apparent diffusion coefficient (ADC) maps were acquired to determine whether the suspected cervical edema was cytotoxic or vasogenic in nature. Cytotoxic edema is characteristic of ischemia (DWI-hyperintense, ADC-hypointense), whereas vasogenic edema (DWI- and ADC-hyperintense) is typical of neoplasms, inflammation, trauma, PRES, blood-brain barrier disruption, or hemorrhage [2].

The third point is that no results from magnetic resonance angiography (MRA) or digital subtraction angiography (DSA) were reported [1]. MRA or DSA of the anterior spinal artery, which is formed at the craniocervical junction by the union of branches from both vertebral arteries, is crucial for ruling out stenosis, occlusion, or dissection of the vertebral arteries or the anterior spinal artery [3]. Unless an angiographic examination of the arteries supplying the cervical spinal cord has been performed, the stroke mechanism cannot be attributed to cardioembolism.

The fourth point is that the nature and cause of the pleocytosis were not clarified [1]. What was the leukocyte count in the cerebrospinal fluid (CSF)? Was the pleocytosis due to an increased number of granulocytes or lymphocytes? Was there intrathecal IgG production, were oligoclonal bands detectable, was there a blood-CSF barrier disturbance, was the CSF culture negative, and were neurotropic viruses detectable in the CSF?

The fifth point is that it is unclear why the patient was initially diagnosed with GBS [1]. Tendon reflexes were initially preserved or exaggerated, and pleocytosis was present. No nerve conduction studies were performed. Thus, the patient did not meet the Brighton criteria for a diagnosis of definite GBS, and there was no indication for the administration of intravenous immunoglobulins (IVIG) [4].

The sixth point is that no explanation was provided as to why the initial MRI of the brain and cervical spine was unremarkable but showed pathological findings on follow-up imaging; this suggests that the cerebellar infarction and the suspected cervical infarction occurred only during the hospital stay. Was there any evidence of cardiovascular risk factors, dehydration, or coagulopathy during the stay? Is it conceivable that the administration of IVIG played a role in the development of the ischemia, as previously reported [5]?

Finally, the patient's current medication regimen was not specified. Given that her medical history included allergic asthma, eosinophilia, GERD, immunoglobulin G deficiency, osteoporosis, peptic ulcer disease, and osteoarthritis, it can be assumed that she was taking various medications regularly. Since she presented with no classic cardiovascular risk factors, it is crucial to assess whether any of her medications contributed to the development of the spinal cord infarction.

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