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Letter to the Editor

Before a Diagnosis of Guillain-Barré Syndrome is Made in a Patient with Lupus Erythematosus, A Demyelinating Neuropathy Must be Ruled Out

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Letter to the Editor

We read with interest the article by Weng *et al.* regarding a 43-year-old female patient with systemic lupus erythematosus (SLE) who developed Guillain-Barré syndrome (GBS), subtype acute inflammatory demyelinating polyneuropathy (AIDP), complicated by trigeminal neuropathy [1]. While the study is noteworthy, several points warrant discussion.

First, the diagnosis of GBS appears questionable [1]. Given that SLE can be associated with demyelinating polyneuropathy [2], it is crucial to know whether nerve conduction velocity (NCV) studies had been performed prior to the patient's current deterioration and whether a demyelinating polyneuropathy had previously been diagnosed. The reported NCV findings can also occur in cases of demyelinating polyneuropathy. Cerebrospinal fluid (CSF) protein levels can also be elevated in SLE if there is cerebral involvement [3]. Furthermore, elevated CSF protein concentration can result from severe hyponatremia. Additionally, magnetic resonance imaging (MRI) did not document any swelling or contrast enhancement of the cervical and lumbar nerve roots.

Second, the cause of the hyponatremia was not elucidated [1]. Although it is mentioned that the hyponatremia was attributable to the syndrome of inappropriate antidiuretic hormone secretion (SIADH), the underlying cause of the SIADH was not specified. Common causes of SIADH include ADH-producing malignancies (e.g., small-cell lung carcinoma), stroke, traumatic brain injury, brain tumors, meningitis, neurosurgical procedures, pneumonia, tuberculosis, other pulmonary infections, medications (e.g., mood stabilizers, anticonvulsants, anesthetics), severe pain, nausea, and stress [4]. Were all these causes thoroughly ruled out in the patient in question? Were serum ADH levels elevated or low? Was the SIADH attributable to SLE?

The third point is that it is questionable whether the patient actually met all the criteria for SIADH [1]. Diagnostic criteria for SIADH include: serum sodium <135 mmol/L; serum osmolality <275 mOsm/kg; urine osmolality >100 mOsm/kg; urine sodium >30 mEq/L despite normal salt and water intake; no signs of dehydration or fluid overload; and normal cardiac, renal, hepatic, adrenal, and thyroid function. Did the patient actually meet all these diagnostic criteria?

The fourth point is that no information regarding the cerebral vessels was provided [1]. Since SLE can be associated with cerebral vasculitis [5], performing magnetic resonance angiography, computed tomography angiography, or digital subtraction angiography would have been essential.

The fifth point is that the patient's current maintenance medication was not specified [1]. Since diuretics can cause hyponatremia, it is essential to know the medications the patient was taking regularly.

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