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Letter to the Editor

Before Diagnosing a Lidocaine-Induced Myasthenic Crisis in a Multimorbid Patient, all Alternative Etiologies should be Thoroughly Ruled Out

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Letter to the Editor

We read with interest the article by Benito-Leon *et al*. about a 68-year-old female patient with a six-month history of proximal tetraparesis, fatigue, dysphagia, mild hypercreatine-kinase (CK)emia, and a myogenic electromyogram (EMG). Following subcutaneous administration of 80mg lidocaine, she suddenly developed acute respiratory failure, altered mental status, facial palsy, and worsening of the tetraparesis, requiring intubation and mechanical ventilation [1]. The acute deterioration after lidocaine was attributed to a myasthenic crisis (MC) based on a slightly positive neostigmine test, decremental repetitive nerve stimulation, increased jitter, and elevated acetylcholine receptor antibodies (AChR-Abs) [1]. The patient partially recovered after administration of intravenous immunoglobulins (IVIG) and pyridostigmine [1]. The study is interesting, but some points require further discussion.

First, it was not specified whether the respiratory failure had cerebral, cardiac, pulmonary, metabolic, electrolyte, or myogenic causes. Determining the cause of acute respiratory failure is crucial, as the type of treatment can depend heavily on the underlying cause. Although the patient underwent cerebral computed tomography (CT) and CT angiography, this does not definitively rule out a brainstem lesion or multiple small embolic infarcts. Given the patient's paroxysmal atrial fibrillation and mitral stenosis, it would have been essential to exclude an embolic stroke due to endocarditis, myocarditis, or atrial fibrillation using multimodal magnetic resonance imaging. Additionally, pulmonary embolism should have been ruled out by contrast-enhanced spiral CT of the lungs. Were the D-dimer levels and other coagulation parameters within the normal range or were they pathological? Since the patient was regularly taking acenocoumarol, it should be known whether the INR was within the therapeutic range. It should also be reported whether metabolic (e.g. lactic acidosis) or respiratory acidosis, and whether the levels of CK-MB, troponin, pro-brain natriuretic peptide, and serum procalcitonin were normal or elevated.

The second point concerns the incomplete investigation of the hyper-CKemia. It should be clarified whether the hyper-CKemia was muscular, cardiac, or cerebral in nature. Was the hyper-CKemia due to primary or secondary myopathy resulting from the use of atorvastatin, deflazacort, or chloroquine? Was a muscle biopsy performed after recovery, and what were the results? Since the patient has been suffering from upper extremity paraparesis for six months, it is unclear why the cause of the paraparesis was not clarified during this period. It should also be explained why the first EMG was myogenic and the second normal. Were myocardial infarction and Takotsubo syndrome completely ruled out? Were there any clinical or echocardiographic signs of heart failure? It should also be reported whether the CK levels remained elevated after the acute deterioration.

The third point is that the diagnosis of myasthenia gravis (MG) and MC remains questionable. In particular, it should be clarified whether the pupils were dilated or constricted at the time of acute respiratory failure. MC is usually associated with pupillary dilation [2]. Since the pupils were described as normal during the clinical neurological examination, MC is rather unlikely. As the patient suffered from systemic lupus erythematosus (SLE), and SLE can be associated with a thymoma [3], the results of the mediastinal CT scan should be available. It should also be clarified whether the investigations that led to the diagnosis of MG were false-positive. Increased jitter can also occur in myositis, reinnervation, congenital myasthenic syndrome, or botulinum toxin infection [4]. A decremental response can also be observed in presynaptic motor endplate disorders (Lambert-Eaton syndrome, botulism), motor neuron diseases, acetylcholinesterase deficiency, or metabolic myopathy [5]. AchR-Abs may be falsely positive in Lambert-Eaton syndrome, thymoma, autoimmune diseases, amyotrophic lateral sclerosis, multiple sclerosis, infections, or with the use of certain medications (e.g., penecillamine). It should be reported

whether elevated AchR-Abs persisted after resolution of respiratory failure or not.
Finally, we should know the patient's long-term course and treatment outcome. Has she fully or only partially recovered from respiratory failure, tetraplegia, facial paralysis, and dysphagia? What was the long-term course of the AchR-Abs? Of particular interest is the long-term treatment of the suspected MG. In addition to deflazacort, did the patient receive steroids, immunosuppressants such as azathioprine, mycophenolate mofetil, or rituximab, or Fc receptor blockers or complement inhibitors?

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