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Letter to the Editor

Other Autoimmune Diseases Along with Myasthenia Gravis are not Uncommon

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Letter to the Editor

We read with interest the article by AbuAlrob *et al*. about a 58-year-old woman with myasthenia gravis (MG) who developed venous sinus thrombosis (VST) shortly after starting plasmapheresis [1]. The VST was attributed to heparin-induced thrombocytopenia (HIT) [1]. The patient benefited from the immediate discontinuation of heparin and the administration of fondaparinux and later apixaban [1]. The study is noteworthy, but some points require discussion.

The first point is that it is not clear why the patient was treated only with pyridostigmine and a thymectomy for her MG [1]. We should know whether antibodies against acetylcholine receptors (AchR), muscle-specific kinase (MUSK), low-density lipoprotein-related protein 4 (LRP4), or titin were elevated, and if any of these were elevated, we should know the titers of these antibodies. If any of these antibodies were elevated, the patient should also have been treated with immunosuppressants.

The second point is that there was no report on why the patient suddenly underwent plasmapheresis. Did she have a sudden worsening of MG, did she have a myasthenic or cholinergic crisis? What were the quantitative myasthenia gravis score (QMGS) and myasthenia gravis composite score (MGCS) shortly before the start of plasmapheresis? What was the last dose of pyridostigmine she took regularly? The standard treatment for generalized MG is with an acetylcholinesterase inhibitor such as pyridostigmine, steroids, and an immunosuppressant such as azathioprine, mycophenolate mofetil, methotrexate, cyclosporine, tacrolimus, or rituximab. Which of these immunosuppressants did the patient take regularly? We should also know why she underwent plasmapheresis but did not receive intravenous immunoglobulins.

The third point is that MG is known to be associated with other immunological diseases such as thyroid disease, systemic lupus erythematosus (SLE), rheumatoid arthritis, inflammatory bowel disease (IBD), vitiligo, neuromyelitis optica spectrum disorders (NMOSD), multiple sclerosis (MS), and autoimmune encephalitis (AIE) [2]. Did the patient have clinical symptoms of these diseases, or were antibodies associated with these diseases also positive?

The fourth point is that we disagree with the view that the index patient is unique [1]. The association between MG and HIT-induced thrombosis has been described previously [3]. A 43-year-old woman with MG developed deep vein thrombosis and pulmonary embolism 12 days after her first exposure to heparin during plasmapheresis due to worsening MG [3]. There is also the case of a 73-year-old man with MG who suffered a pulmonary embolism during daily plasmapheresis due to worsening MG [4].

The fifth point is that alternative causes of coagulopathy have not been sufficiently ruled out. Since the patient apparently travelled from Iran to Qatar after plasmapheresis and air travel is associated with an increased risk of thrombosis, exsiccosis, immobility, or hereditary coagulopathy should be ruled out as alternative causes of VST.

In summary, MG is frequently associated with autoimmune diseases and has also been reported in conjunction with HIT. MG patients require close monitoring and adequate immunosuppressive treatment to avoid complications from more invasive treatment.

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