Peer Review File

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Reviewer A

The manuscript describes a possible new association of malignancy (pancreatic carcinoma) with a typical paraneoplastic syndrome involving the neuromuscular junction. I partially agree with the authors that this manuscript brings a new paraneoplastic association with Lambert-Eaton myasthenic syndrome. The major concern is that the authors considered only clinical and serum biomarker (positive anti-VGCC antibody) as definitely diagnostic of Lambert-Eaton myasthenic syndrome. However, this explanation is not possible, even with the absence of tendon reflexes characteristic of presynaptic membrane compromise of the neuromuscular junction. Neurophysiological testing evaluation is a key step to define this diagnosis. There are several cases of individuals with positive serum anti-VGCC antibody titles in the context of oat-cell lung carcinoma and no clinical features of Lambert-Eaton myasthenic syndrome. I suggest the authors adding information about the neurophysiological studies which were performed during the patient's diagnostic work-up.

Response: Thank you for pointing this out. We agree with this comment. Therefore, we have provided some more data about the neurophysiological study that was conducted. In the revised manuscript you can find more information in page 4 and from line 115 to 121. Other neurophysiological studies weren't able as a pacemaker was implanted (page 4, line 130) Moreover, replying to your comment about the possibility of a lung carcinoma as we stated in the manuscript, we performed a CT Thorax without evidence of malignancy and furthermore a diagnostic serum test of Ati-SOX-1 Antibodies was negative. A further immunological laboratory diagnostic with Antibodies associated with other neurological disorders was also negative, as it stands in page 4, lines 136-138.

Reviewer B

This clinical case is handy in our medical work because the occurrence of neuromuscular disorders in pancreatic cancer is not usually described because of its malignancy and the delay in diagnosing this type of cancer. The presentation of the case is very detailed, but the authors should describe more findings in the neurophysiological test because it was the first test to support the diagnosis of a neuromuscular disorder. If they performed repetitive stimulation, on what nerve did they perform this test: on a distal or peripheral nerve? What frequency of stimulation did they use? The diagnostic hallmark of Eaton-Lambert myasthenic syndrome is≥60% increment at high-rate stimulation or postexercise facilitation; however, authors describe 'a fading'. Because of these findings, the authors should explain more carefully why they suspected Eaton-Lambert syndrome after performing a Train of four or if they suspected initially having Myasthenia Gravis, but because of the laboratory results, they diagnosed Eaton-Lambert syndrome**.**

Response*:* We appreciate the reviewer's insightful suggestion and agree that it would be useful to demonstrate more information about the neurophysiological study that was conducted. We demonstrate all these findings in page 4, line 115 to 121.