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LUNG ADENOCARCINOMA WITH THE LAMBERT–EATON MYASTHENIC SYNDROME, A CASE REPORT

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Introduction - Purpose : Introduction: The Lambert–Eaton myasthenic syndrome (LEMS) is a neuromuscular disease caused by pathogenic autoantibodies directed against voltage-gated calcium channels (VGCCs) in presynaptic nerve terminals. Neuroendocrine tumors, including SCLC and MCC, are the most common solid tumors associated with paraneoplastic neuropathies. However, LEMS rarely occurs with non-SCLC. In this case, we presented a patient with lung adenocancer who developed LEMS

Findings : Case: In March 2017, multiple metastatic lesions were detected in brain CT of a 68-year-old male patient who presented with blurred vision and dizziness. Excisional biopsy of the occipital lobe was performed, and the result was compatible with adenocarcinoma metastasis. In thorax CT, a mass of 6x3 cm was detected in the right superior lobe of the lung. The bronchoscopic biopsy performed was consistent with non-SCLC. EGFR mutation, Ros1 and ALK rearrangement were negative. Patient diagnosed with metastatic lung adenocarcinoma, started cisplatin + pemetrexed chemotherapy in April 2017. After 10 days, the patient had proximal muscle weakness, especially in the lower extremities symmetrically. Findings supporting Lambert Eaton myasthenic syndrome were detected in the electromyography (EMG). LEMS was diagnosed with clinical and EMG findings and intravenous immunoglobulin (IVIG) treatment was started. The treatment received a dramatic response and the patient began to walk again. The patient who was treated at another center for primary disease was excluded from our follow-up.

Discussion : Discussion: Lambert-Eaton myasthenic syndrome was first described as a paraneoplastic syndrome in patients with lung cancer, now known to be idiopathic in nearly half the cases. Paraneoplastic LEMS is usually observed in older patients, with smoking history and who have developed small cell lung cancer. However, our patient had adenocarcinoma of the lung. The LEMS diagnosis was ultimately based on characteristic clinical findings, electrophysiological studies, and antibody testing. The most important clinical feature of LEMS is a significant reduction in the CMAP amplitude of motor nerves that is caused by the lack of ACh release at the nerve junction. Treatment of LEMS must be custom-made per disease severity, comorbidities, coexistence of cancer, and life expectancy. Since LEMS associated with cancer is a paraneoplastic phenomenon, primary treatment should be targeted to cancer because LEMS frequently improves with successful cancer therapy. Immunotherapy of LEMS itself usually produces little or no improvement in strength without effective treatment of underlying malignancy.

Keywords: Lung Adenocarcinoma, Lambert–Eaton, Myasthenic Syndrome