determine the efficacy of combined HRT/ACT versus HRT alone.

2701 BOTULISM, A MIMIC OF MILLER FISHER SYNDROME AND ACUTE BRAINSTEM STROKE

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Introduction Botulism is a rare neuromuscular junction disorder that causes descending flaccid paralysis, dysautonomia and cranial nerve palsies.[1] Guillain-Barre Syndrome (GBS) is an immune-mediated polyradiculoneuropathy with ascending paralysis, areflexia, dysautonomia and albuminocytological dissociation on cerebrospinal fluid (CSF) analysis.[2] Miller Fisher variant of GBS causes ophthalmoplegia, areflexia and ataxia.[2] Brainstem strokes may cause cranial nerve palsies and quadriplegia.[3] We present a case of botulism that presented a diagnostic dilemma, confounded by a delayed history of spoiled milk consumption, mimicking GBS and brainstem stroke.

Case A 61-year-old man with moderate stroke risk factors, reported acute diplopia, ataxia and dysarthria. No infective prodrome or suspected food poisoning was initially disclosed.

Day 2, he developed dysphagia and severe respiratory distress requiring intubation. He developed rapidly progressive ophthalmoplegia and descending paralysis, requiring ventilation. Sequential intravenous immunoglobulin and plasma exchanges were minimally effective.

Day 12, his partner recalled consumption of expired almond milk, 2 days before admission. He was provisionally diagnosed with botulism and given botulin antitoxin. He continues to slowly recover.

Clostridium botulinum mouse bioassay was eventually confirmatory. Stool *C. difficile* antigen and toxins were negative. CSF was bland. Anti-ganglioside antibodies, including GQ1-b were negative. Nerve conduction studies and electromyogram confirmed generalized predominantly motor neuropathy. MRI brain and spine/plexus were normal.

Discussion This case was diagnostically challenging, given limited history at the time and rapidly progressive signs that overlapped between botulism, GBS and brainstem stroke. Tests to distinguish these conditions are not always rapidly available or reliable, so empiric treatment should not be delayed.

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2702 TROUBLESOME PTOSIS: AN ATYPICAL PREDOMINANTLY OCULAR PRESENTATION OF LAMBERT-EATON MYASTHENIC SYNDROME

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Objectives Lambert-Eaton myasthenic syndrome (LEMS) is a rare, often paraneoplastic, presynaptic neuromuscular junctionopathy that typically presents with proximal weakness. We present a case with prominent ocular manifestations, to highlight this important yet atypical presentation.

Methods Case report.

Results A 66-year-old female was transferred to our service for investigation of a mediastinal mass, in the setting of 3-months of progressive ptosis and diplopia, later accompanied by proximal weakness. She had a background of a fifty pack-year smoking history.

Examination revealed bilateral ptosis which improved with sustained upgaze, mixed horizontal/vertical diplopia, non-fatigable 4+/5 power in the proximal limbs, and reduced lower limb reflexes which improved with repetition.

Nerve conduction studies revealed reduced compound muscle action potential (CMAP) amplitudes, with peroneal and ulnar CMAP amplitudes increasing >100% following 10 seconds exercise, and abductor digiti minimi CMAP amplitudes incrementing >100% with high-frequency repetitive nerve stimulation. Voltage-gated calcium channel antibodies were positive (462 pM) (normal <30 pM). Neuraxial imaging was normal. PET-CT scan and biopsy of the mediastinal mass revealed extensive stage small cell lung cancer.

With a diagnosis of paraneoplastic LEMS confirmed, she was commenced on amifampridine 10 mg twice-daily and had remarkable improvement within one week, back to baseline.

Conclusions Ocular symptoms in LEMS is uncommon, present in approximately one-quarter of patients in the largest case series.[1] A predominantly ocular presentation, as in our case, is considered rare. As our case highlights, the presence of paradoxical lid elevation after sustained upgaze may be a particularly useful clinical sign in diagnosing these patients.

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2703 THE MANY FACES OF NEUROSARCOIDOSIS: UNDERSTANDING ITS CLINICAL DIVERSITY

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Neurosarcoidosis is a manifestation of sarcoidosis that affects the nervous system. It presents with a heterogeneous clinical picture. The diagnosis is established through clinical evaluation, imaging, and tissue biopsy in most cases. We discuss two cases that presented with symptoms suggestive of multiple sclerosis (MS) and stroke like features, but in fact yielded a diagnosis of neurosarcoidosis.

The first is a 54-year-old male with a background of diplopia and paraesthesia of his limbs, who subsequently had acute ataxia with gait imbalance, and new onset of vertical diplopia. Neuroimaging revealed multiple pontine, cerebellar, and temporal lobe acute infarcts, and leptomeningeal spinal enhancement. Cerebrospinal fluid analysis was consistent with diagnosis of neurosarcoidosis. The second is a 45-year-old female presenting with lower limb paraesthesia and shock like symptoms when she flexed her neck. She had no objective clinical deficit. Neuroimaging was performed and