Conclusion This case highlights the diagnostic challenges in patients with MNGIE which often delays the initiation of appropriate treatment.

2695 CLINICAL AND ELECTROPHYSIOLOGICAL PROFILE OF ANTI-MAG NEUROPATHY AT WELLINGTON REGIONAL HOSPITAL

Purwa Joshi, Dhara Knight*. *Wellington Regional Hospital, New Zealand, Wellington, Wellington, New Zealand*

10.1136/bmjno-2023-ANZAN.97

Objectives Recent studies of neuropathy associated with anti-myelin-associated-glycoprotein (anti-MAG) antibodies, have observed that the electrophysiological features are more heterogeneous than originally described. Our aim was to evaluate the clinical and electrophysiological features of anti-MAG neuropathy at our hospital.
Methods Patients followed up for anti-MAG neuropathy at Wellington Regional Hospital were identified by searching the Neurology outpatient clinic database. Their electronic records were reviewed to collect demographic, clinical, laboratory, electrophysiological, and treatment variables.
Results Five patients with anti-MAG neuropathy were identified; all Caucasian males, aged between 58 and 86 years at diagnosis. Neuropathy symptoms were present for between 2 and 10 years at diagnosis. All had a distal, sensory-predominant neuropathy, with asymmetric lower limb involvement in 3/5. All had an IgM-kappa paraprotein, and 2/5 were diagnosed of Waldenstrom's macroglobulinaemia. Anti-MAG titres ranged between 5218 to >70,000 BTU. Lower limb nerve conduction studies demonstrated axonal loss, with absent sensory responses, and absent or very attenuated distal motor amplitudes (<1.0 mV) in all patients. Median nerve distal motor latencies ranged between 5.25 and 17.8 mV, and median nerve motor conduction velocities ranged between 20.2 and 46.0 m/s. Three patients received immunosuppression in the form of rituximab and/or chemotherapy, and one had a partial improvement in symptoms. Of note, all patients were ambulatory at last follow-up, at least 8 years from symptom onset.
Conclusion Our small cohort of patients with anti-MAG neuropathy demonstrated a fairly typical clinical syndrome, but a heterogeneous electrophysiological profile. All patients remained ambulatory in the long term.

2696 PROGRESSIVE ATAXIA AND PALATAL TREMOR WITH HYPERTROPHIC OLIVARY DEGENERATION

Tony Zhang*, Bernard Liem, Neil Anderson. *Auckland City Hospital, Grafton, New Zealand*

10.1136/bmjno-2023-ANZAN.98

Background Progressive ataxia and palatal tremor (PAPT) is characterized by adult-onset ataxia and palatal tremor typically due to a lesion affecting the Guillain-Mollaret Triangle. This pathway consists of the ipsilateral inferior olivary nucleus and red nucleus, as well as the contralateral cerebellar dentate nucleus. PAPT may be idiopathic or as a result of stroke, tumor, infection or demyelination. Familial causes of PAPT have also been rarely described.
Case A 70 year-old European man presented with a one year history of progressive gait unsteadiness. There were no ocular or bulbar symptoms, hearing loss, tinnitus, vertigo or vomiting. Past medical history was significant for controlled hypertension and an alcohol intake of 21 standard drinks per week. Family history was unremarkable. Examination of his palate revealed oscillatory movements of his soft palate at a frequency of 2 hertz without the sensation of 'ear clicks'. There was moderate truncal and gait ataxia but no limb ataxia. MRI demonstrated increased T2 and FLAIR hyperintensities in bilateral medullary olives consistent with hypertrophic olivary degeneration. Genetic testing was requested for glial fibrillary acid protein, POLG mutation and spinocerebellar ataxia panels to exclude familial forms of PAPT.
Conclusion We report a patient with the clinical syndrome of PAPT and corresponding MRI findings suggestive of bilateral hypertrophic olivary degeneration. Descriptions of this are limited to case series in the literature. Palatal tremor should be carefully observed for in all patients presenting with ataxia as it can often be asymptomatic.

2698 COMBINED HABIT REVERSAL TRAINING AND ACCEPTANCE AND COMMITMENT THERAPY FOR TREATMENT OF TICS IN TOURETTE SYNDROME

[1]Jenny Eisenhauer, [1]Ali Buckland*, [2]Stuart Watson, [1,3,4,5]Rick Stell. *[1]Perron Institute of Neurological and Translational Science, Nedlands, WA, Australia; [2]Watson Research and Statistics Consulting, Perth, WA, Australia; [3]Consultant Neurologist, Sir Charles Gairdner Hospital, Nedlands, WA, Australia; [4]Director of Movement Disorder Unit, Perron Institute of Neurological and Translational Science, Nedlands, WA, Australia; [5]Clinical Associate Professor, Nedlands, WA, Australia*

10.1136/bmjno-2023-ANZAN.99

Objectives This pilot study aimed to investigate the efficacy of a combined behavioural intervention to improve severity of tic symptoms in adults with Tourette Syndrome. The treatment protocol combined elements of Habit Reversal Therapy (HRT) and Acceptance and Commitment Therapy (ACT). HRT is a behavioural approach to the treatment of tics and other habit disorders (Woods, 2001). The current study combined HRT with ACT, an empirically based psychological intervention and useful adjunct to HRT given its focus on acceptance, rather than avoidance, of internal experiences (such as urges to tic). It was hypothesised that the treatment would result in reduced tic severity.
Methods Tic symptoms were assessed at baseline, post-intervention, and at 6- and 12-months follow-up on 11 participants. The primary outcome measure was the Yale Global Tic Severity Scale (YGTSS) and validated using a video assessment. Intervention consisted of eight, 1-hour weekly individual treatment sessions.
Results Using mixed-effects regression, results indicated that compared to pre-treatment scores, participants experienced an average significant reduction in tic severity (YGTSS) at post-treatment (b=-10.36, p=.002), maintained at six- (b=-8.19, p=.012) and 12-month follow up (b=-8.82, p=.009). Results of the video assessment demonstrated a similar significant treatment effect that was maintained at 6- and 12-month follow-up.
Conclusion Results suggest that the combined HRT/ACT treatment protocol is effective in reducing tic severity in adults with Tourette Syndrome, with maintained improvements up to 12 months post-intervention. These results have provided data for a future comprehensive randomised-controlled trial to

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

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

[1]Chris Kwan, [2]Anthony Elias, [1]Michal Lubomski*. [1]*Department of Neurology, Royal North Shore Hospital, St Leonards, NSW, Australia*; [2]*Department of Infectious Diseases and Microbiology, Royal North Shore Hospital, St Leonards, NSW, Australia*

10.1136/bmjno-2023-ANZAN.100

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.

REFERENCES

1. Sobel J. Botulism. *Clin Infect Dis.* 2005;**41**(8):1167–73.
2. Dimachkie MM, Barohn RJ. Guillain-Barré syndrome and variants. *Neurol Clin*. 2013;**31**(2):491–510.
3. Searls DE, Pazdera L, Korbel E, Vysata O, Caplan LR. Symptoms and signs of posterior circulation ischemia in the new England medical center posterior circulation registry. *Arch Neurol.* 2012;**69**(3):346–51.

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

Rohit Sharma*, Michael Ginevra, Lakshini Gunasekera, Abhishek Malhotra. *Neurosciences, University Hospital Geelong, Barwon Health, Geelong, VIC, Australia*

10.1136/bmjno-2023-ANZAN.101

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.

REFERENCE

1. Young J, Leavitt J. Lambert-Eaton Myasthenic Syndrome. *J Neuroophthalmol.* 2016;**36**(1):20–2.

2703 THE MANY FACES OF NEUROSARCOIDOSIS: UNDERSTANDING ITS CLINICAL DIVERSITY

John Girgis*, Koshy George. *Neurology, Gold Coast University Hospital, Gold Coast, QLD, Australia*

10.1136/bmjno-2023-ANZAN.102

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