follow-up 9 months after diagnosis. This case highlights the need to maintain a high index of suspicion for alternative aetiologies when evaluating a patient with MS who presents with progressive neurological symptoms, especially with the breadth of treatment options and rising numbers of older patients at risk of immunosenescence.

2342 ACUTE SPINAL CORD MRI DIFFUSION RESTRICTION IN A CASE OF MOGAD

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Introduction Although myelin oligodendrocyte glycoprotein associated disease (MOGAD) has well described spinal cord MRI features, the utility of diffusion-weighted imaging in acute MOGAD is not clear. Here we describe a case of MOGAD with true restricted diffusion on spinal cord MRI and consider therapeutic and prognostic implications.

Case A 45-year-old man presented with acute urinary retention and over several days developed predominantly complete paraplegia and anaesthesia below T4 level with acute and severe sphincter dysfunction. Two weeks prior he had upper respiratory tract infection symptoms. An admission MRI spine demonstrated longitudinally extensive transverse myelitis from C3-C7 and T1-T10. Serum MOG antibodies were confirmed using a live cell-based assay. He was treated with pulsed intravenous methylprednisolone followed by a slow oral steroid taper, plasma exchange, IVIg, and rituximab. A repeat scan 4 weeks from onset, whilst our patient remained paraplegic, showed patchy foci of true diffusion restriction (hyperintense on diffusion-weighted imaging and hypointense apparent diffusion coefficient) from T6-T8 without contrast enhancement. Six months later he has no clinical or radiological evidence of disease relapse and can walk short distances independently, although has residual sphincter dysfunction.

Conclusions Restriction on diffusion-weighted imaging may reflect a greater degree of acute inflammation, and thus potentially be useful as a prognostic marker in MOGAD. We review the scant literature on diffusion restriction in longitudinally extensive traverse myelitis, and in neuroinflammatory conditions more broadly, and consider implications for guiding proactive immunotherapy regimes at disease onset.

2343 IS IMBALANCE IN CIDP CAUSED BY LOWER LIMB TREMOR?

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Objectives Neuropathy causes imbalance to a greater degree than expected based on clinical and neurophysiological findings alone. Our hypothesis was that inflammatory neuropathy patients might experience subclinical lower limb tremor that then causes imbalance, similar to primary orthostatic tremor.

Methods Patients with CIDP were consecutively recruited from neuromuscular clinics in Sydney. Tremor studies with surface electromyography (EMG) to rectus femoris, biceps femoris, tibialis anterior, and medial gastrocnemius were performed, as well as posturography using force platform analysis. Nerve conduction studies were performed to correlate tremor findings with neuropathy. Berg Balance Scale (BBS) was undertaken to quantify balance.

Results Twenty-six patients with CIDP were recruited, mean age 65±2.6 years. They were moderately impacted by imbalance, with mean BBS 43/56. A spectral peak on Fast Fourier Transform was found between 10–12 Hz in 11/26 (42%) patients when seated with legs held out in front. Posturography disclosed a high frequency peak from 12–18 Hz in the 'z' axis (up-down) in 9/26 (34%) patients while standing, similar to primary orthostatic tremor. Interestingly, however, a corresponding spectral peak on EMG was only present in 4/9 patients. Overall, tremor was detected in 58% of patients. There were no correlations between peripheral nerve conduction studies and these findings.

Conclusions Patients with CIDP suffer moderate imbalance. Lower limb tremor was detected in 58% of patients, and a subset had similar neurophysiological hallmarks to primary orthostatic tremor. This raises the question of whether orthostatic tremor is the cause of imbalance in CIDP, or a physiological compensatory mechanism in those with imbalance.

2345 MENINGIOMA ENCASEMENT OF THE INTERNAL CAROTID ARTERY AS A RARE CAUSE OF STROKE

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A 62 year old woman with a history of hypertension presented to the hospital with sudden onset left sided hemiplegia. She also complained of headaches and visual blurriness days leading up to admission. On examination, she was found to have dense left-sided hemiplegia, hemianopia and neglect with a National Institute of Health Scale Score (NIHSS) of 12. CT of the brain and neck angiography revealed an occlusion of the right internal carotid artery (ICA) and bifrontal oedema. Subsequent MRI (including DWI, SWI, T1 with gadolinium contrast, and T2 FLAIR) showed infarction overlying the right M2 territory with a large frontal meningioma. The meningioma was also found to have encased the right ICA. The patient was commenced on aspirin and dexamethasone. The patient then underwent inpatient rehabilitation was referred to neurosurgery for further observation. The case highlights meningioma encasement of ICA as a rare cause of ICA occlusion, leading to stroke

Meningiomas more commonly cause transient ischaemic attacks (TIAs) due to encasement of carotid arteries, leading to vascular insufficiency. There are few reported cases of meningiomas causing strokes.[1] [2] The internal carotid artery