Case An 18-year-old male presented with 3-month history of intermittent visual blurring and headaches. He had absent upward gaze. Fundoscopy showed focal optic disc oedema with phlebitis in the right eye. MRI brain revealed bilateral enhancing thalamic lesions with minor cystic change in the left thalamus. Cerebrospinal fluid (CSF) showed normal biochemistry, 27 white cells (96% lymphocytes). CFS anti-neuronal and limbic encephalitis antibodies were negative, oligoclonal bands were detected. HLAB51, ANCA were not detected and ACE level was normal.

Treatment involved 5-day course of 1g intravenous methylprednisone followed by tapering steroid dose with improvement in the ocular findings. PET scan and thoracic/mesenteric angiogram and testicular ultrasound were unremarkable. Three monthly progress MRI's showed gradual increase in the size of the left thalamic lesion with worsening oedema and involvement of the left midbrain. Repeated CSF analysis showed leukocytosis with restricted oligoclonal bands and negative flow cytometry. Decision was made to proceed with biopsy of the left thalamic lesion. Histopathology demonstrated sheets of loosely cohesive cells with large nuclei and nulcleoli consistent with an intracranial germinoma. The patient was referred to oncology team for further treatment.

Conclusion This case highlights the presence of focal optic disc swelling as a primary presenting manifestation of intracranial germinoma. In addition, it demonstrates that intracranial germinoma can mimic CNS inflammatory lesions and that persistence of MRI changes should prompt consider of biopsy for tissue diagnosis.

2454 THE ROLE OF THE NEUROLOGIST FOR ADULTS WITH SPINAL MUSCULAR ATROPHY

[1,2]Lauren M Sanders*, [1,2]Katrina A Reardon, [3,4]Katrina A Morris, [5]Gayatri Jain, [4,6]Matthew C Kiernan, [7]Christina Liang, [3,4]Alastair J Corbett. *[1]Medicine, University of Melbourne, Fitzroy, VIC, Australia*; *[2]Neurosciences, St Vincent's Hospital, Melbourne, Fitzroy, VIC, Australia*; *[3]Neurology, Concord Repatriation General Hospital, Concord, NSW, Australia*; *[4]University of Sydney, Sydney, NSW, Australia*; *[5]Neurology, Austin Hospital, Heidelberg, Vic, Australia*; *[6]Insititute of Clinical Neurosciences, Royal Prince Alfred Hospital, Camperdown, NSW, Australia*; *[7]Neurology, Royal North Shore, St Leonards, NSW, Australia*

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Objective Management priorities of neurologists and patients do not always align. We sought to understand the neurologist's role from the perspective of adults living with spinal muscular atrophy (SMA).

Methods Focus groups were conducted with adults with SMA covering management expectations, assessments and treatments. Sessions were facilitated by neurologists via zoom. Groups were structured according to SMA severity. Participants were provided with a pre-session survey which sought opinions on aspects of neurological care. Results from the survey were used to initiate discussion within the focus groups. The neurologist's role was the first discussion topic. Sessions were recorded for verbatim audio transcription. Data were explored using reflexive thematic analysis.

Results Twenty-eight adults with SMA participated (ages 25–68). There was significant variability in experiences of engagement with neurologists. Some participants had not seen a neurologist for many years, perceiving consultations to have limited benefit. Frustration was expressed at the inequity of access to neuromuscular expertise across Australia. There was consensus that neurologists have a coordinating role within a multi-disciplinary care team that includes the person with SMA. Multi-disciplinary clinics were preferred, although few had experienced this model of care. Participants valued consultations where their neurologist provided updates on treatments and advances and demonstrated attention to individualisation of management. In general, mental wellbeing was experienced to be either ignored or poorly managed by neurologists.

Conclusion We identified a variable disconnect between needs of adults with SMA and neurologist provided care. Further work is required to ensure equitable access to neuromuscular expertise across Australia.

2455 CASE REPORT: AN UNUSUAL PROGRESSIVE GAIT ATAXIA

Xi (Jessica) Jia*, Sameen Haque. *Nepean Hospital, Kingswood, NSW, Australia*

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Case Description Ms PM is a 79yo lady with a 6-weeks history of progressive ataxia. Her mobility deteriorated rapidly from walking independently to requiring assistance. Her past medical history is significant for breast cancer, hypertension, and hypercholesterolemia. On initial examination, Ms PM has significant truncal and limb ataxia with shuffling gait. She also has cogwheel rigidity in her upper limbs and increased tone in her lower limbs. Her power and sensation were normal. Basic biochemistry and CSF analysis were unremarkable.MRI Brain had shown brain atrophy and chronic microvascular ischaemic changes. MRI spine had shown no cord compression. EEG was normal. Ms PM was trialed on levodopa, IVIG and methylprednisolone with no improvement in her mobility. Alarmingly, Ms PM developed rapid cognitive decline and cerebellar signs in her upper limbs over 3-week period while stayed as an inpatient.

Discussion Ms PM's initial presentation was suggestive of Parkinson-plus syndrome. It accounts for 10% of clinical parkinsonism. It normally has a faster progression and lack of response to levodopa treatment compared to Parkinson disease. However, it is normally not associated with rapid cognitive decline. Therefore, prior disease was considered. Creutzfeldt-Jakob disease (CJD) is the most frequent of prior diseases with an occurrence rate of one in a million per year. To diagnose probable sCJD, one needs to have progressive dementia and at least two of the four clinical features: myoclonus, visual or cerebellar disturbance, pyramidal or extrapyramidal dysfunction and akinetic mutism. Ms PM's presentation fits well with the clinical diagnosis of sCJD.

2456 CERVICAL CORD COMPRESSION: A UNIQUE PRESENTATION OF MCARDLE SIGN

Indumathi Singh*, Sai Nagaratnam, Hugo MoralesBriceno. *Department of Neurology, Westmead Hospital, Westmead, NSW, Australia*

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Objectives We report a unique case of McArdle sign secondary to extrinsic cervical cord compression. McArdle sign is the presence of rapidly reversible weakness induced by neck flexion. It is thought to be entirely specific and 65% sensitive for