days followed by weaning high dose oral prednisolone and maintenance intravenous immunoglobulin (IVIg). Progress MRI demonstrated ongoing inflammatory changes and so an FDG-PET scan was performed which demonstrated no suspicious FDG avid malignancy. Two months later her clinical symptoms had largely resolved, with marked improvement in her cognitive function.

Conclusion In contrast to other reported cases of post-COVID-19 ADEM, our patient had mild antecedent infection, was relatively young and had an excellent neurologic outcome. Furthermore, 'cloud-like enhancement' was seen on neuroimaging, a finding previously thought specific to NMOSD.

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2451 DIAGNOSTIC CHALLENGE – JAPANESE ENCEPHALITIS MASQUERADING AS POST SARS-COV-2 ENCEPHALITIS

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Objectives and Methods Para or post-infectious encephalitis is a neurological syndrome associated with a concomitant or antecedent infection, usually viral. The damage to the nervous system might be caused directly by the virus or by the host immune response to the infection. We report a case of encephalitis in a 68-year-old male who presented with a 4-day history of progressive paraparesis, bilateral cerebellar ataxia, and unilateral lower motor neuron pattern facial nerve palsy, 1 week after resolution of mild SARS-CoV-2 infection.

Results Non-contrast CT of brain was unremarkable. MRI showed T2/FLAIR hyperintensities in the brainstem at bilateral cerebellar peduncles, left thalamus, left midbrain, and periventricular regions. There was no evidence of transverse myelitis. CSF analysis revealed pleocytosis and increased protein with negative BioFire viral PCR. High dose corticosteroid therapy was commenced due to clinical suspicion of post-SARS-CoV-2 encephalitis and significant clinical improvement followed. Due to increasing Japanese Encephalitis Virus (JEV) cases in the region, additional JEV tests were performed and returned positive CSF JEV PCR.

Conclusion Post-SARS-CoV-2 and Japanese encephalitis are both rare and debilitating neurological conditions in Australasian setting. Diagnosis of post-SARS-CoV-2 relies on exclusion of geographically common infective aetiologies and may be akin to acute disseminated encephalomyelitis in pathophysiology. This case underscores importance of remaining vigilant on emerging infections such as JEV in Australasia. There are limited high quality evidence-based therapies for both conditions and further studies are urgently required to guide the diagnosis and choice of therapies.

2452 TICK-BORNE ENCEPHALITIS PRESENTING AS HEMICHOREA

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Introduction Tick-borne encephalitis (TBE), caused by infection with Flaviviridae vector-borne viruses, is a rising health concern in parts of the world.[1] TBE viruses may cause meningitis, encephalitis, myelitis and polyradiculitis.[2] We describe an unusual presentation of TBE with chorea.

Methods Case report.

An 82 year-old female presented with acute right-sided hemichorea and confusion. Her background was significant for treated polymyalgia rheumatica, hypertension on verapamil and restless legs syndrome on pramipexole. She described numerous tick bites across her scalp three weeks prior to presentation, treated with a week's course of doxycycline. Examination revealed severe right-sided chorea with no other extrapyramidal signs.

Results There was no stroke, or other cause for her symptoms on MRI. The CSF was abnormal with a mononuclear pleocytosis (30 x 10^6/L), normal protein, negative culture and negative multiplex PCR for viruses. Serological and CSF work-up for alternate causes of chorea was negative. An FDG-PET CT brain demonstrated no regional cerebral glucose hypometabolism. After exclusion of all other causes, she was diagnosed with encephalitis with hemichorea. Following treatment with intravenous pulse methylprednisolone her symptoms resolved one week later.

Conclusion To our knowledge there are only two other case reports of TBE causing chorea.[2] [3] This is the first case reported in Australia. The mechanism of chorea remains unclear, but responded to immunotherapy so quickly and comprehensively that neuronal molecular mimicry with a viral or bacterial pathogen, or venom protein seems more likely than a productive viral infection.

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2453 INTRACRANIAL GERMINOMA RESEMBLING AN INFLAMMATORY CENTRAL NERVOUS SYSTEM LESION

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Introduction Intracranial germinoma is a rare but important differential in the work of up central nervous system (CNS) lesions.

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

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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

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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

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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