recommended regular physiotherapy and ongoing respiratory support.

Conclusion Multiminicore myopathy is a rare inherited condition typically presenting in childhood. Respiratory failure is common in large case series, though onset is usually before adolescence. As in the present case, muscle biopsy features can be non-specific and further genetic testing may be required for diagnosis. With genetic panels now more widely available, it is likely that diagnosis rates of rare diseases will increase, thus changing our understanding of disease demographics.

REFERENCE

1. Ogasawara M, Nishino I. A review of core myopathy: central core disease, multiminicore disease, dusty core disease, and core-rod myopathy. *Neuromuscul Disord.* 2021 Oct;**31**(10):968–977. doi: 10.1016/j.nmd.2021.08.015. Epub 2021 Sep 17.

2818 RECURRENT MENINGO-ENCEPHALITIS DUE TO MOG-ANTIBODY ASSOCIATED DISEASE

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Introduction Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) typically presents with optic neuritis, myelitis, or intracranial demyelination. Recent literature has described presentations consisting of meningo-cortical inflammation associated with anti-MOG antibody. This syndrome is sometimes known as FLAMES – FLAIR hyperintense lesions in anti-MOG associated encephalitis with seizures.

Case report We present the case of a 39-year-old man who presented to hospital with one month of headache associated with photophobia and mild encephalopathy. During his admission the patient developed left-sided focal motor seizures. MRI of the brain showed FLAIR high signal in the right median occipital parasagittal sulci with post-contrast leptomeningeal enhancement extending to the left occipital lobe. CSF analysis showed raised white cells with a negative gram stain and negative viral PCR panel. Serum and CSF antibody tests were positive for anti-MOG antibody. Treatment with intravenous methylprednisolone resulted in rapid improvement in symptoms over days. Repeat MRI two months later showed normalization of the cortical change.

One year later, the patient represented with headache. Repeat MRI brain showed new leptomeningeal enhancement predominantly within the right occipital lobe, and repeat CSF was inflammatory. Treatment with intravenous steroids again rapidly improved symptoms, and long-term treatment with oral steroids and rituximab was instituted.

Conclusions Atypical MOGAD presentations include a spectrum of cortical and meningeal predominant manifestations. Our patient is a typical example of this, with a typically rapid response to treatment. Testing for anti-MOG antibodies is crucial in patients with cortical hyperintensities on MRI, as prompt diagnosis and treatment can result in improved outcomes

2821 BILATERAL DYSGEUSIA SECONDARY TO POST-COITAL UNILATERAL THALAMIC ISCHAEMIC STROKE

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Introduction This case report describes a twenty-five-year-old female with bilateral loss of taste secondary to right anterior ischaemic thalamic stroke.

Methods A single case report of a patient admitted to Mater Hospital, Australia with review of related published case reports.

Results A right-handed twenty-five-year-old female presented with post-coital acute onset headache, dizziness, dysarthria & left-sided facial droop. Initial symptoms resolved & the patient became aware of bilateral loss of taste. She described limited ability identifying foods previously enjoyed & subsequent dissatisfaction with oral intake.

Dysgeusia was evident on examination bilaterally with all tested food stimuli.

MRI revealed a focus of restricted diffusion in the right hemi-thalamus consistent with acute ischaemic stroke. CT head and neck angiogram were unremarkable with no evidence of vasospasm. Trans-oesophageal echocardiogram reported a positive agitated saline contrast study suggestive of patent foramen ovale & referred for closure.

Discussion The dorsomedial thalamic nucleus is involved in both intensity of taste perception and the hedonic nature of oral intake.[1] Both thalami provide bilateral taste perception, however there is marked variability in innervation, with rare reports of unilateral thalamic strokes resulting in bilateral loss of taste.[2]

The reward pathways associated with pleasant taste sensations are important for motivating oral intake and maintaining nutrition. Gustatory dysfunction generally improves, but is important to address in the acute stroke period including nutritional assessment, diet modifications & associated mood disorder.[1]

Conclusions Bilateral dysgeusia secondary to unilateral thalamic stroke is a very rare stroke complication with associated serious patient consequences including malnutrition.

REFERENCES

1. Uider FM, Sivagnanaratnam A. Bilateral loss of taste from a unilateral thalamic infarct. *Practical Neurology*. 2021;**0**:1–4.
2. Kogawa S, Yamakawa I, Nakajima A, *et al*. Bilateral ageusia caused by right thalamic infarction. *Rinsho Shinkeigaku*. 2013;**53**:24–8.

2822 FROM HOSPITAL TO HOME: SUPPORTING CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY PATIENTS ON SUBCUTANEOUS IMMUNOGLOBULIN THERAPY

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Objectives We review consumer satisfaction with the support provided to those with chronic inflammatory demyelinating polyneuropathy (CIDP) treated with subcutaneous immunoglobulin (SCIg) at our hospital.
Methods We performed a chart review of all CIDP patients transitioned from intravenous immunoglobulin (IVIg) to SCIg from 2019–2022 at the Royal Melbourne Hospital. We reviewed the education and support process, medication and consumable supply to patients, and assessed patient-reported satisfaction through interview.
Results Five patients were transitioned onto SCIg, 3 patients continuing SCIg at the end of the period and a further 3 patients had commenced education in preparation for transition to SCIg. The same approach was used in all cases: 'SCIg eligible' patients were identified by the neuromuscular medical team, and with consent, our SCIg nurse provided initial in-person education at a subsequent IVIG infusion or via telehealth. Patients underwent two supervised SCIg infusions with the SCIg nurse; further training was not required, but could be provided upon evaluation of patient's experience and self-administering competence. Consumables and SCIg vials are provided by RMH infusion centre and pharmacy, and the patients are reviewed every 8–12 weeks by the treating team and/or SCIg nurse. Four out of 5 patients were available for interview, and all reported the medication was 'very easy' to administer as instructed, and expressed satisfaction with the process of SCIg education, transition and nursing support.
Conclusions Consumers consistently report a high degree of satisfaction with the process of SCIg education, treatment initiation and ongoing nursing support at our centre.

2824 REFRACTORY CHRONIC INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY UNMASKED BY CHADOX1 NCOV-19 VACCINE

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Introduction Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an acquired autoimmune condition affecting the peripheral nerve and nerve roots. Vaccination for the SARS-CoV-2 virus has been associated with incident CIDP diagnoses, though the mechanism is unclear. We present a case of refractory CIDP following administration of the ChAdOx1 nCoV-19 vaccine and propose that vaccination unmasked a previously asymptomatic neuropathy.
Case A 79-year-old man presented with acute onset lower limb weakness and sensory disturbance 2 weeks after administration of the ChAdOx1 nCoV-19 vaccine. Clinical and neurophysiological assessments were consistent with acute inflammatory demyelinating polyneuropathy (AIDP). Despite early treatment with first line therapy, the patient experienced relapses over 5 months with incomplete recovery. A sural nerve biopsy in the acute setting showed changes consistent with chronic demyelination/remyelination. The patient improved following plasma exchange, rituximab, and bortezomib and was independently mobile on discharge.
Discussion The development of inflammatory neuropathy following vaccination is an established association. CIDP is a rare association with the ChAdOx1 nCoV-19 vaccine, typically presenting with acute neuropathy that proceeds to chronicity, as in our patient. In the present case, sural nerve biopsy showed findings consistent with chronic neuropathy although it was taken in the acute setting. We propose that the COVID-19 vaccine unmasked CIDP in a previously asymptomatic patient. A high index of suspicion and regular follow for those who develop neuropathy following vaccination is recommended.

2826 CASE REPORT: THYROTOXIC NEUROPATHY, A RARE BUT UNDER RECOGNISED CONDITION

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Introduction Well known neurological associations with thyrotoxicosis include myopathy, periodic paralysis and ophthalmoplegia. Thyrotoxic neuropathy aka 'Basedow's Paraplegia' was described by Charcot in 1889 but remains rarely reported. We describe a case of subacute ophthalmoplegia, proximal and distal areflexic weakness in the setting of newly diagnosed thyrotoxicosis.
Case A 54-year-old previously well Chinese female presented with a 3-month history of progressive vertical diplopia, 20 kg of weight loss and symmetrical proximal limb weakness. Initial examination revealed a complex external ophthalmoplegia associated with peri-orbital swelling and lid retraction. Right eye elevation was impaired, and both vertical and horizontal movements were limited in the left eye. Symmetrical distal greater than proximal limb weakness including 3/5 ankle movements were recorded. Temperature and pain sensation was absent in a stocking distribution to the upper tibia associated with absent lower limb reflexes. Asymmetrical lower limb pitting oedema was also present. The gait demonstrated a waddling and high stepping quality. Investigations revealed a thyroid panel consistent with profound primary hyperthyroidism. Magnetic resonance imaging of the brain and orbits excluded cerebral lesions, cavernous sinus, or cranial nerve pathology. Interestingly, extra-ocular muscle enlargement was not present radiologically.
Conclusions This case represents a rare but described example of thyrotoxic neuropathy, which is likely an under recognised condition.

2827 REDUCED CALPAIN EXPRESSION IN A PATIENT WITH FASCIOSCAPULOHUMERAL MUSCULAR DYSTROPHY

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Background Limb girdle muscular dystrophies (LGMD) show phenotypic and genotypic variability. Autosomal recessive calpainopathies (LGMD R1) can mimic fascioscapulohumeral dystrophy (FSHD) and is an important differential in patients with a scapuloperoneal syndrome.