Conclusion This case highlights the importance of considering neurosyphilis in a variety of neurological presentations, in this case encephalopathy, facial nerve palsy and gelastic seizures localizing to the frontal lobe. Neurosyphilis is increasing in prevalence and is treatable.

3160 TARGETS FOR INPATIENT STROKE CARE: ARE THEY BEING MET, ARE THEY APPROPRIATE? DATA FROM AN AUCKLAND HOSPITAL

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Background/Objectives New Zealand sets three inpatient quality metrics as benchmarks to evaluate clinical performance in the field of stroke care: admission to an organised stroke unit (80% within 24 hours of presentation), appropriate reperfusion therapy (12% of patients with ischaemic stroke), and early transfer to rehabilitation services (80% within 7 days of admission). This study assesses the performance of an Auckland hospital with respect to these targets and examines association with a number of secondary variables. Further qualitative data was collected to contexualise the secondary variables.
Methods Two hundred consecutive patients with a discharge diagnosis of stroke were selected, a sample size large enough to estimate the proportions above within a 95% confidence interval. Ethics approval was granted and locality approvals obtained. Data abstraction was performed by study investigators. Statistical analysis was performed in the R programming language.
Results Patients admitted to the Stroke Unit within 24 hours: 50.0%

Patients with ischaemic stroke who received reperfusion therapy: 16.7%

Patients transferred to Rehabilitation service within 7 days of presentation: 40.8%

A Cox Hazards model for time to discharge showed two secondary variables with significant differences.
Conclusion Although only one target was met, secondary variables data suggests complex circumstances which question the value of targets as a measure of performance. There has been mixed data regarding ethnic, gender or age-related inequity of care in acute stroke management in New Zealand, there was no evidence of this in our study. Overall, data was suggestive of an "after-hours or weekend effect".

3161 SAME BUT DIFFERENT: A RARE CASE OF PRIMARY CEREBRAL AMYLOIDOMA

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Background Cerebral amyloidoma is a rare form of localised CNS tumefactive amyloid Aβ protein deposition with no evidence of systemic amyloidosis. It is suggested to occur due to misfolding of local proteins and subsequently deposited in tissue.

The distinct radiographic appearance is of a nodular mass focused in the supratentorial periventricular white matter. The lesion demonstrates avid contrast enhancement with perilesional vasogenic oedema, but without significant mass effect given lesion size.

In comparison to more common neurodegenerative amyloid deposition diseases, given the localised nature of the lesion, primary cerebral amyloidoma is potentially curable with aggressive targeted therapy.
Case We describe a forty-five-year-old female referred for assessment of progressive left-sided upper-motor-neurone features, with concern for intracranial glioma on peripheral imaging. She initially presented with left upper limb weakness and paresthesias and over three months developed left lower limb weakness and gait disturbance.

MRI revealed right frontoparietal confluent nodular irregular areas of enhancement most evident in the deep white matter, with extension to the periventricular region and cortex, with vessels transversing the lesion.

Brain biopsy demonstrated amorphous eosinophilic nodular deposits of amyloid, with positive congo-red staining consistent with a diagnosis of lambda light-chain cerebral amyloidoma.

Extensive investigations performed excluded evidence of systemic amyloidosis. Stereotactic radiotherapy was performed with clinical stability over the following eighteen months.
Conclusion This case highlights the rare but important differential of cerebral amyloidoma to be considered in the assessment of CNS mass-like lesions, which presents with a distinct clinico-radiological presentation.

3162 ALOPECIA UNIVERSALIS AFTER ALEMTUZUMAB IN MULTIPLE SCLEROSIS - A NINE YEAR FOLLOW UP AND REVIEW OF LITERATURE

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Objective and Background Alemtuzumab is a humanised monoclonal antibody against CD 52 with for highly aggressive multiples sclerosis with some undesirable side-effects. There have been few reports of skin disorders including the very rare *alopecia universalis*. We aim to report a case with this in long term follow up, explain the pathomechanisms and also present a comprehensive review of literature.
Methods We report a 36 year old male, started on alemtuzumab in 2015 and presented with Alopecia universalis in 2019. A systematic literature search in pubmed using the keywords "Alemtuzumab" "skin" "cutaneous" "alopecia" "hair loss" was done. Phase 3 studies and the side-effects during that time were also studied.
Results He started reporting isolated circular hairless areas in various regions of the body . Eventually, he lost hair all over his body with eyebrows being the last to go. He received baricitinib tablets, steroid injections and cyclosporin and is back to normal now at 9 years follow up. We have photographic evidence and a timeline of his evolution which we would like to present along with the hypothesized mechanisms.

Conclusion Due to the depletion of peripheral CD4 + and CD8 + T cells and initially of CD 19+ B cells, an increased risk of skin tumours after alemtuzumab is plausible. Our case report & review summarizes the mechanisms based on multiple case reports and series, highlights the need for registry data for such side-effects in large populations of MS who received alemtuzumab and emphasizes the need for multidisciplinary involvement in MS care.

3163 SARS-COV-2 INFECTION PRECIPITATING GANGLIONIC ACETYLCHOLINE RECEPTOR ANTIBODY SEROPOSITIVE AUTOIMMUNE AUTONOMIC GANGLIONOPATHY WITH CEREBELLAR ATAXIA

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Background Autoimmune autonomic ganglionopathy (AAG) is a rare disorder presenting with autonomic dysfunction secondary to alpha-3-ganglionic acetylcholine receptor antibodies (gAChR-Ab). Autonomic dysfunction and neuroimmunological complications following SARS-CoV-2 are reported although de novo pan-dysautonomia due to gAChR-Ab seropositive AAG following SARS-CoV-2 has not been described previously.
Methods We report a case of autonomic failure due to gAChR-Ab seropositive AAG triggered by SARS-CoV-2 infection.
Results A 53-year-old previously healthy male developed subacute autonomic symptoms including, sweating alterations, early satiety, xerostomia, dry eye, constipation and orthostatic hypotension following SARS-CoV-2 infection. These were accompanied by brain fog, gait instability, diplopia, and dysarthria. Examination revealed Adie pupils and mild cerebellar dysfunction.

Autonomic testing demonstrated inappropriate sinus tachycardia and orthostatic hypotension. Symptom management required fludrocortisone, ivabradine and famotidine. Nerve conduction studies, MR brain and CSF studies were unremarkable. Pathological gAChR-Ab were identified in serum by flow cytometric immunomodulation assay (25%, reference ≤18). Serum glutamic acid decarboxylase (GAD) antibody was positive at low titre (15 IU/mL, reference <5). Malignancy was excluded. The patient was diagnosed with AAG, and improved with prednisolone and intravenous immunoglobulin, although with end-of-cycle deterioration. Rituximab achieved sustained improvement in autonomic symptoms, ataxia and brain fog.
Conclusion SARS-CoV-2 infection may precipitate production of gAChR-Ab leading to dysautonomia. AAG is rare but important to identify as an immunotherapy-responsive cause of autonomic symptoms after SARS-CoV-2 infection. Positive serum gAChR-Ab and ancillary autonomic testing confirms the diagnosis. Ataxia is rare in patients with gAChR-Ab and its pathological mechanism is uncertain.

3164 UNILATERAL POST-HYPOXIC MYOCLONUS

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Post-hypoxic myoclonus is a highly concerning phenomenon, which confers a grave but not universally fatal prognosis. We present a case of a 72-year-old woman who developed persistent but fluctuating unilateral facial myoclonus 1-day post cardiothoracic and vascular surgery, that had been complicated by intraoperative haemorrhage and hypoxia. Whilst still intubated and sedated, clinical examination confirmed left lower face twitching that waxed and waned in frequency and amplitude. EEG demonstrated near-continuous, semi-rhythmic 1–2Hz spike-wave and sharp-wave discharges over the right hemisphere maximal centrally, moderate diffuse right hemisphere slowing but only mild background slowing over the left hemisphere. Left temporal leads demonstrated electromyographic artefact time-locked to right sided discharges. MRI-B revealed cortical and subcortical watershed infarcts in the right cerebral hemisphere, and increased signal along the right pericentral cortex, presumed to reflect laminar necrosis. Levetiracetam and subsequently valproate eventually suppressed myoclonus, the patient was able to be extubated after several days and begin communicating after several further days. Asymmetric cerebral hypoperfusion was hypothesised to be related to either underlying vascular disease or surgical redirection of blood flow intraoperatively. This case highlights the variable impact of post hypoxic myoclonus on neuroprognostication. Despite similarity in semiology, there is a spectrum to hypoxic-myoclonus, building on previously recognised entities including Lance-Adams Syndrome and Myoclonic-Status-Epilepticus.[1]

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3166 SPONTANEOUS INTRACRANIAL HYPOTENSION (SIH) DURING LATE PREGNANCY

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A previously healthy primiparous 29-year-old woman, presented at 35-weeks' gestation with a 12-day history of postural headache. There was no previous headache history and no preceding trauma or lumbar puncture. The pregnancy was uncomplicated. Initial MRI-B was interpreted as normal. Due to persistent and debilitating headache, MRI-B was repeated 2-weeks later and demonstrated profound features of intracranial hypotension. MRI-spine showed epidural collections at cervical and thoracic levels. Conservative management was