immunoglobulin, oral and intravenous corticosteroids, azathioprine, and mycophenolate.

Residual hip, abdominal and truncal weakness remained, affecting her gait and posture. CK levels during this period remained normal. MRI demonstrated extensive paravertebral muscle loss with fatty replacement without oedema. Small myopathic units without active features were present on EMG.

Conclusion This case illustrates several important aspects of NAM, which remains a rare disorder. Recovery can be incomplete in up to 40%, especially if control is delayed.² In our opinion, normalisation of CK is an important target and may be necessary for full recovery, and rising CK may predict relapse.³ We suggest MRI and EMG of weak muscles may assist in distinguishing persisting myositis from 'burnt-out' disease, clarifying whether immunotherapy should be increased or not. However relapse on weaning immunosuppression remains frequent. While SRP antibodies in patients who had also taken statins has been previously reported, the quick sequential onset in this case suggests possible causality.

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073 ACUTE SPINAL INFARCTION: TREATMENT AND OUTCOMES WITH AND WITHOUT HYPERBARIC OXYGEN THERAPY AT FIONA STANLEY HOSPITAL

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Introduction Spinal cord infarction is a potentially devastating disorder commonly presenting with anterior spinal artery syndrome developing over minutes to hours, accounting for an estimated 1% of stroke presentations. Aetiologies range from aortic surgical complications, systemic hypotension and other rare causes including fibrocartilaginous embolism and vascular malformations. Diagnosis is clinical combined with restriction on diffusion-weighted MRI. There are no specific treatment guidelines. The evidence for hyperbaric oxygen therapy (HBOT) in acute spinal infarction is mixed. This case series describes ten cases of acute spinal infarction at Fiona Stanley Hospital (FSH), half of which received HBOT.

Methods Data for all MRI-proven spinal cord infarctions at FSH between 2014 and 2018 were reviewed.

Results Ten patients, median age 55years (31–74), 60% male. Aetiologies: three fibrocartilaginous emboli, five likely atheroembolic disease, one antiphospholipid syndrome, one cryptogenic. Sixty percent presented with flaccid paraplegia ranging from levels C4 to T11. Five patients received HBOT within a median time of 31 hours from symptom onset, with average 15 treatments. Three patients received triple therapy of HBOT, pentoxifyline and high-volume lumbar-puncture

drainages and had median MRC muscle power of 5- on discharge, compared with 2+ power in those who did not. Nine required inpatient rehabilitation, of average eight weeks' duration, with median disability at discharge of 3 on the Modified Rankin Scale.

Conclusion Spinal infarctions can be severely disabling. Fibrocartilaginous embolism may be more common than previously thought. More research is needed on the role of acute triple therapy with pentoxifyline, spinal drains and HBOT given the marked difference in this case series.

074 EARLY AUSTRALIAN EXPERIENCE WITH ERENUMAB FOR CHRONIC MIGRAINE

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Introduction Erenumab is available in Australia since October 2018. We review the effectiveness and safety in 2 Australian headache centres.

Methods Erenumab (70 mg or 140 mg) was prescribed. We monitored headache days, migraine days, analgesic use, adverse reactions, Headache Impact Test-6 (HIT6) score and Migraine Disability Assessment (MiDAS) score, at baseline and at 3 months. Primary outcomes were reduction in headache and migraine days, and adverse effects. Secondary outcomes were improvement in functional scores and analgesic use.

Results 65 patients (ages 18–73; mean 44 years) commencing Erenumab were assessed before and after 3 monthly treatments. The duration of chronic migraine (CM) ranged from 1 to 40 years, with 3 to 16 previous failed prophylactic treatments.

There was a >50% response in overall headache days and migraine days in 29% (19/65) and 46% (27/59), respectively. There was a modest (10–49%) response in overall headache days and migraine days in 29% (19/65) and 27% (18/59), respectively. There was no improvement in headache days and migraines in 42% (27/65) and 27% (14/59), respectively.

At onset, the mean HIT-6 and MiDAS scores were 66 and 65, decreasing after 3 treatments to 59 and 32, respectively. The mean monthly days taking triptan and codeine medications reduced from 9 and 6 days, to 5 and 3 days, respectively.

There were few reported side effects.

Conclusion This Australian cohort in tertiary referral refractory migraine patients achieved a significant rate of reduced headache and migraine days with good safety and tolerability.

075 A CASE OF SPONTANEOUS CSF RHINORRHEA IN A PATIENT WITH MARFAN SYNDROME

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Introduction Spontaneous cerebrospinal fluid leak is uncommon condition and frequently associated with hereditary

disorders of connective tissues. Nasal CSF leakage is extremely rare.¹

Methods and results We present the case of a 40-year-old woman presented to hospital for few days history of postural headache associated with clear intermittent discharge from right nostril without any signs of meningism. There was no history of trauma. She has a background history of Marfan syndrome with associated complications of ASD repair at age 2, mechanical Aortic and Mitral valve replacement, aortic root repair, previous ST elevation MI with LV dysfunction, automated implantable cardioverter-defibrillator in situ, atrial fibrillation, and Hashimoto's thyroiditis. Her regular medications are warfarin, bisoprolol and thyroxine. The clear nasal discharge was positive for β -2 transferrin confirming cerebrospinal fluid. Her CT Brain did not reveal any clear site of CSF leak. She had a flexible nasendoscopy which showed normal nasal passageway, no defect in nasal mucosa and no active CSF leakage. She was managed conservatively with strict bed rest and advised to minimise strenuous activity and heavy lifting.

Conclusion Spontaneous cerebrospinal fluid leak is uncommon condition and frequently associated with hereditary disorders of connective tissues. Nasal CSF leakage is extremely rare.¹ Testing β -2 transferrin has high sensitivity and specificity.² Initial treatment may include bed rest, oral or intravenous hydration, oral caffeine or corticosteroids.³⁻⁴ If conservative therapy fails, surgical repair with nasal endoscopic approach is recommended.^{2,5}

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A RARE CASE OF ANTI-GLUTAMATE DECARBOXYLASE ANTIBODY SYNDROME PRESENTING WITH RECURRENT VOMITING

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Introduction Anti-glutamate decarboxylase antibody (anti-GAD) has been linked with various neurological syndromes including stiff-person syndrome, limbic encephalopathy, cerebellar ataxia, eye movement disorders and epilepsy (collectively known as 'anti-GAD positive neurological syndromes').¹ We describe a very atypical phenotypic presentation of anti-GAD syndrome with unexplained vomiting and weight loss.

Case A 46 years old lady with no past medical or family history of note, presented with 6 months history of severe headaches and recurrent attacks of episodic vomiting (4–6 episodes of multiple vomiting daily) with no identified precipitant and complete normality in between the episodes with no other associated symptoms. She reported 15 kg of unintentional

weight loss. Neurological examination and investigations including MRI brain, CT angiogram and liver enzymes, immunoglobulins, thyroid function, vasculitic screen were normal. Upper GI endoscopy, gastric emptying studies, CT imaging of chest, abdomen and pelvis and whole body PET scan were unremarkable. Serum autoimmune antibody screen was positive with high titre of anti-GAD antibody (1200 kU/liter). The cerebrospinal fluid anti-GAD antibody titre was raised at 103.7 kU/liter with otherwise normal parameters including negative oligoclonal bands. The nerve conduction studies did not show continuous motor activity or spasmodic reflex myoclonus (seen in stiff-person syndrome).² A therapeutic trial of immunosuppression was introduced with moderate improvement in symptoms.

Conclusion Anti-GAD neurological syndromes are rare and this is a unique presentation of the same. It is not completely understood why the presence of one antibody causes varied syndromes. The hypothesis is that the recurrent vomiting is possibly due to diaphragmatic spasms.

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AN UNUSUAL PRESENTATION OF ANTIBODY NEGATIVE AUTOIMMUNE ENCEPHALITIS: A CASE REPORT

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Introduction Immune-mediated encephalitis can occur in the absence of measurable pathogenic antibodies in CSF or serum.

Methods and results We present the case of a 29-year-old Sudanese woman presented with four days history of meningism. She is three months post-partum with her fifth child and does not have significant past medical history. She is a current smoker with history of methamphetamine and heroin use until 5 years ago. There were no focal neurological deficits on examination. CT brain was normal and her CSF had $420 \times 10^6/L$ leucocytes (Polymorphs 18, Mononuclears 402). Three days later, she deteriorated clinically with GCS 11 and developed left sided hemiplegia requiring intubation and ventilation in intensive care.

The CSF culture along with autoimmune encephalitis panel, anti-neuronal antibodies, HSV, VZV, Enterovirus, cryptococcal antigen, TB quantiferon, HIV, Malaria were negative. Her immunology tests were unremarkable including rheumatoid factor, complements, anti-nuclear antibodies, dsDNA, PR3, MPO, RNP, Smith, Ro, La, Scleroderma 70 and Jo-1 antibodies. The MRI Brain showed persistent changes in right hemispheric cortical and subcortical gyriiform FLAIR hyperintensities which were also present in DWI images. The EEG showed right temporo-occipital region 7–8 Hz rhythmic discharges with generalised diffuse slowing. She had right parietal lobe brain biopsy which shows perivascular and intramural inflammation by mononuclear cells almost comprised of CD3 T-cells consistent with encephalitis associated with secondary lymphocytic vasculitis. She was treated with intravenous pulsed methylprednisolone and IVIg