cessation of seizures. He was discharged on weaning dose of prednisolone, maintenance IVIg, mycophenolate, and ASM. Ten months after cessation of immunotherapy, patient's mesothelioma continued to be stable.

Conclusion This case highlights the important role of immunotherapy in treating focal seizure secondary to ICI-induced AE. Interestingly, cessation of ICI therapy did not lead to tumor progression at 1 year follow-up in this case.

2758 A CASE LIKE NO UDDER

[1]Mina MB Botrous*, [2]Sumana SG gopinath. [1]*St George Hospital, Carlton, NSW, Australia*; [2]*Neurology, Campbelltown Hospital, campbelltown, NSW, Australia*

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A young patient with acute behavioural disturbance due to meningo-encephalitis of unclear cause. 38 year old butcher returned from work feeling generally lethargic and unwell. He woke up in the night pacing aimlessly around the house and developed non-specific tingling in the right leg and presented to the emergency department . A stroke code was initiated for focal deficits. The acute investigations were unremarkable including CT Brain and Angiogram. He was discharged home but represented 2 days later when his wife found him urinating on the bed at midnight without recollection of the events. No focal neurological findings on examination.MRI brain demonstrated leptomeningeal enhancement supratentorially bilaterally. Initial lumbar puncture demonstrated WCC >300 with predominately mononuclear cells. No organisms grown and CSF viral panel, fungal culture, AFB all negative. EEG demonstrated excessive slowing without any epileptiform discharges.Empiric cover for meningo-encephalitis commenced and IV Methylprednisolone for 5 days with an oral taper The limbic encephalitis panel was negative. Brucella IgM was low positive on serology and Flavivirus IgM positive but this was thought to be a possible false positive. CSF PCR for Brucella and Japenese encephalitis were negative. Completed 3 weeks of antimicrobials and a course of steroids with good clinical improvement.Notable features are the clinical/radiological findings compatible with meningo-encephalitis of unclear cause. The patient's occupation presented challenges due to the potential exposure to atypical organisms without clear diagnostic results.We discuss the importance of occupational exposure to potential CNS infections, and need to include uncommon pathogens in our diagnostic armamentarium.

2759 ADULT-ONSET ACUTE NECROTISING ENCEPHALITIS WITH BILATERAL HOMONYMOUS INCONGRUENT QUADRUPLE SECTORANOPIA

Jessica Redmond, Robert Hurford, Anthony Fok. *Department of Neurology, Royal Melbourne Hospital, Melbourne, VIC, Australia*

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Introduction Acute necrotising encephalopathy (ANE1) is a genetic predisposition related encephalopathy that can occur following influenza type A, parainfluenza or Human Herpes Virus 6 infection. It is commonly seen in children and associated with the autosomal dominant missense mutation in the RAN Binding Protein 2 (RANBP2) gene.[1–3]

Case A 45-year-old female with known RANBP2 mutation presented with coryzal symptoms, headache and blurred vision and tested positive to influenza A infection. RANBP2 was previously screened in our patient as her daughter developed ANE1 in childhood. Formal visual fields showed bilateral homonymous incongruent quadruple sectoranopia. Magnetic Resonance Imaging (MRI) Brain demonstrated T2 FLAIR bilateral lateral geniculate nucleus hyperintensities with foci of low susceptibility and diffusion restriction and was treated with intravenous methylprednisolone and plasma exchange. Follow-up showed MRI hyperintensities had reduced and the patient's visual fields improved. Optical coherence tomography (OCT) of the retinal nerve fibre layer (RNFL) and ganglion cell layer demonstrated corresponding thinning in the pattern of field defects consistent with retrograde degeneration.

Discussion We describe novel visual field defects secondary to bilateral thalamic lesions from influenzae A triggered ANE1. The wedge-shaped visual field defects are explained by the vascular topography of the thalamus and the incongruency is related to the anterior location of the visual pathway lesions.

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2760 MANAGING HYPOKALAEMIC PERIODIC PARALYSIS DURING PREGNANCY AND LABOUR: A CASE REPORT

Namrata D Sobarun*, Emily Watson*. *Princess Alexandra, Woolloongabba, QLD, Australia*

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Case Report Hypokalaemic periodic paralysis (HypoPP) is a rare inherited neuromuscular disorder characterized by intermittent episodes of focal or generalized weakness of skeletal muscle which can last hours to days, with concomitant hypokalaemia.[1] Pregnancy has been previously reported to exacerbate symptoms, however ideal management during pregnancy and labour is not well documented.[2]

We present a case of a 34-year-old woman with confirmed HypoPP secondary to a pathological variant in the SCN4A gene who surprisingly reported improvement in her symptoms during pregnancy, requiring minimal oral potassium supplementation. Significant care was taken to avoid known triggers, with a heavy emphasis on dietary modification. The patient went on to have a vaginal delivery at term, utilising spinal anaesthesia without complication.

This case highlights the importance of an individualized and multi-disciplinary approach when managing patients with rare neurological conditions such as HypoPP in the obstetric setting.

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2763 VARICELLA EFFECTING THE BACK OF THE VENTRICLE: A CASE OF AREA POSTREMA HICCUPS CAUSED BY VARICELLA ZOSTER VIRUS

Serge Geara*, Chathupa Wickremaarachchi. *Gosford Hospital, Wyoming, NSW, Australia*

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We present a case of an immunocompetent 54 year old male who presented with a 6 day history of headache, photophobia, nausea and fevers with no other signs of meningism. He also developed intractable hiccups interrupting sleep. On examination the patient was found to have Left sided T4 dermatomal distribution of a Zoster Rash. Cranial nerve examination was normal.

Cerebrospinal fluid showed normal glucose and protein with a white cell count of 158, all monocytes. The sample was polymerase chain reaction positive for Varicella Zoster Virus (VZV).MRI brain showed bilateral area postrema T2/FLAIR hyperintensity. The patient responded well to 3 days of intravenous methylprednisolone (IVMP) and aciclovir, followed by oral valaciclovir, in addition to gabapentin and metoclopramide. VZV reactivation is a rare but well recognised cause of intractable or perisstent hiccups and nausea (IHN). In a series by Hayashi et al,[1] it is clinically distinct from neuromyelitis-optica spectrum disorder (NMOSD) associated with IHN and responds well to IVMP and aciclovir. VZV also affects older males and is usually unilateral compared to NMOSD.

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2768 CARNITINE DEFICIENCY: A REVERSIBLE CAUSE OF PROXIMAL MUSCLE WEAKNESS

[1]Ivonne Lichtenberg*, [2]Nicholas Yeo, [3]Min Xia Wang, [1]Abhay Venkat, [3]Roger Pamphlett. *[1]Department of Neurology, Wollongong Hospital, Wollongong, NSW, Australia*; *[2]Wollongong Hospital, Wollongong, NSW, Australia*; *[3]Department of Neuopathology, Royal Prince Alfred Hospital, Sydney, NSW, Australia*

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Objective Carnitine is necessary for β-oxidation of fatty acids within the mitochondria. Carnitine deficiency often affects the skeletal and cardiac muscle. Secondary carnitine deficiency in adults can result from inadequate dietary intake, poor absorption, renal tubular dysfunction or medication side effects. Here we report a 72 year-old woman who presented with proximal lower limb weakness secondary to carnitine deficiency.

Case A 72 year-old woman presented with worsening mobility with increasing falls. She had a history of sensorineural hearing loss, chronic hydrocephalus and epilepsy on a background of childhood meningitis. She was on long-term phenytoin, primidone and sulthiamine. She had a normal diet and no significant family history. Examination revealed a symmetrical proximal lower limb myopathy. Magnetic resonance imaging (MRI) of the neural axis was non contributary, nerve conduction studies were unremarkable but electromyography was consistent with a myopathic process. MRI of the thighs showed patchy low-grade oedema suggestive of a myositis but her serum myositis autoantibody panel was negative and her serum creatinine kinase levels were normal. A muscle biopsy revealed multiple small and large vacuoles within myofibres on hematoxylin/eosin staining, which on oil red O staining were seen to contain lipid, consistent with a carnitine deficient myopathy. A serum acylcarnitine panel was consistent with secondary carnitine deficiency. The patient has made a dramatic improvement following oral carnitine replacement and adjustment of her antiepileptic medications.

Conclusion When identified, carnitine deficient myopathy is an easily treatable condition in which symptoms are largely reversible.

2770 CLINICAL AND RADIOLOGICAL CHARACTERISTICS AND OUTCOMES OF PATIENTS WITH RECURRENT OR RELAPSING TUMEFACTIVE DEMYELINATION

Mst Irin Pervin*. *Concord Repatriation General Hospital, Glenwood, NSW, Australia*

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Objective We studied the clinical course, neuroimaging, CSF, and treatment of patients with recurrent tumefactive demyelinating lesions (TDLs).

Methods We used PubMed to identify published reports of recurrent TDLs and included 1 unpublished patient from the Brain and Mind Centre, Sydney. The expanded disability status scale (EDSS) scores for the patients were estimated when not provided.

Result We identified 19 cases (11F, 8M) with recurrent TDLs. The median age at onset of the index TDL was 37 years (range 12–72) and most were solitary lesions 68% (13/19). A gadolinium-enhancing TDL was seen in 86% (12/14) and 50% (6/12) of those showed incomplete ring enhancement. Twenty-six percent (5/19) were diagnosed previously with MS and 3 fulfilled 2017 McDonald criteria.

CSF-restricted oligoclonal bands (OCBs) were detected in 32% (5/17). Only one of those tested (n=13) was positive for AQP4-IgG and was diagnosed with NMOSD. No patient tested (n=6) was MOG-IgG positive.

A moderate-to-marked response to treatment (high dose corticosteroid +/- disease modifying agents/plasmapheresis) was seen in 82% (14/17). The estimated median EDSS at presentation was 3 (range 1–7) and EDSS at the median follow-up of 36 months (range 6–144) was 2 (range 1–10). Most remained ambulatory (EDSS <4 in 14/19), 2 patients died.

Conclusion The profile of patients with relapsing TDLs is similar to that of typical MS, but there are differences including a lower female:male sex ratio, large lesions and comparative lack of CSF-restricted OCBs. Outcomes vary among this group of patients ranging from minimal disability through to death.