had a waddling gait. Clinical picture was consistent with a clinical diagnosis of LEMS rather than myositis, which was confirmed by elevated anti-VGCC antibodies and response to Acetylcholinesterase inhibitors.

Results Anti-VGCC antibodies elevated at 119pM(<30).

Transiently elevated CK, negative myositis autoantibodies, negative anti-MuSK antibodies, negative AChR antibodies.

Although repetitive nerve stimulation did not show increment in the right ulnar CMAP after isometric muscle activation, the clinical picture was consistent with LEMS.

Marked improvement to treatment with oral prednisone and pyridostigmine. Due to side effects, pyridostigmine was changed to 3,4-Diaminopyridine therapy with excellent response.

Steroids were weaned off and the patient is adequately controlled on 3,4-Diaminopyridine.

Conclusion Our case report shows that LEMS can arise as a result of an immune-related adverse event (irAE) to pembrolizumab; an Anti-PD-1 Monoclonal Antibody. The immune response persists after cessation of this checkpoint inhibitor medication. It is important to recognise and treat this condition early.

063 'HAVE I GONE MAD?': A CASE OF ALICE IN WONDERLAND SYNDROME

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Case Report

Introduction Alice in Wonderland Syndrome (AIWS) is a rare neurological disorder that is characterised by unusual distortions to perception. The aetiology of AIWS is unclear, though it has been reported in a number of neurological conditions including infection (esp. EBV), CNS lesions, migraines and as a side effect to medications.

This report outlines the case of a 72 year old gentleman who presented to hospital due to visual changes which he described as dysmetropsia (objects appeared distorted - thinner) and episodic chromatopsia. His chromatopsia was described as his entire visual field coloured with an orange hue, though this colour changed throughout the course of his admission. Movements appeared to occur in slow motion and movements of humans had a robotic appearance. Neurological examination was otherwise normal.

These symptoms resolved after 2 days, and on subsequent examination his neurological and ophthalmological examinations were unremarkable.

Results MRI brain revealed a right occipito-temporal T2 hyperintense lesion, initially interpreted as a subacute ischaemic infarct. Repeat MRI with contrast revealed a stable appearance of the lesion with areas of subtle contrast enhancement. PET scan showed reduced metabolic activity within the lesion with reduced FDG accumulation.

Biopsy of the lesion identified features of a diffuse astrocytoma.

Conclusions AIWS is a poorly recognised syndrome. Symptoms are not typical for an ischaemic event and alternative diagnosis should be investigated as an explanation for the cause of visual distortions.

064 FALSE POSITIVE RT-QUIC TEST FOR CREUTZFELDT JAKOB DISEASE IN DEMENTIA WITH STATUS EPILEPTICUS

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We present the case of a 72-year-old woman with likely false positive tests for Creutzfeldt Jakob disease (CJD). She had a background of focal epilepsy and dementia, related to previous alcohol abuse and head trauma. On her initial presentation in October 2019, she was drowsy with continuous left sided focal motor seizures. An EEG demonstrated continuous periodic lateralised epileptiform discharges (PLEDs) arising from the right temporal region, consistent with status epilepticus. She was commenced on levetiracetam and subsequently lacosamide, with seizure resolution over 12 days. Despite control of her seizures, she remained significantly cognitively impaired. A cerebral MRI demonstrated asymmetric cortical and thalamic diffusion restriction and the possibility of sporadic CJD was raised. A lumbar puncture revealed a normal total protein (0.4 g/L), a positive 14-3-3 protein but an undetectable tau protein. She was eventually discharged to a residential aged care facility, however returned to a different hospital in January 2020, obtunded with recurrent generalized seizures. Further results of her previous CSF examination were now available, revealing a positive RT-QuIC assay. However, a repeat MRI brain demonstrated resolution of the previous regions of diffusion restriction. Serial EEGs demonstrated continuous right temporal PLEDs which improved after the addition of sodium valproate. Repeat cognitive screening was markedly improved, however not quite reaching her 2019 baseline. We suggest that a false positive RT-QuIC test probably arose from status epilepticus in the context of significant pre-existing cerebral pathology. Excluding a pre-mortem diagnosis of CJD may be challenging in the setting of recurrent seizures.

065 EBV AND NMDA RECEPTOR ANTIBODY POSITIVE OPSOCLONUS-MYOCLONUS SYNDROME IN AN IMMUNOCOMPROMISED PATIENT WITH RENAL CLEAR CELL CARCINOMA: A CASE REPORT

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Introduction Rare cases of double positive Epstein-Barr virus (EBV) and anti-N-methyl-D-aspartate receptor (anti-NMDAR) antibody causing encephalitis have been described associated with solid organ transplant.[1–4] Opsoclonus-myoclonus syndrome is often a paraneoplastic or parainfectious phenomenon, but has only rarely been described associated with anti-NMDAR and usually with additional neuropsychiatric symptoms or encephalopathy.[5–6] We describe the rare case of a renal transplant patient with opsoclonus-myoclonus syndrome

associated with renal cell carcinoma and anti-NMDAR and EBV DNA detected in cerebrospinal fluid (CSF).

Methods This was a personal case of the authors, with documentation and investigations reviewed from patient medical records at Sir Charles Gairdner Hospital.

Results A 55 years old immunocompromised female patient presented with two weeks of increasing ataxia, oscillopsia and tremor, preceded by 3 weeks of headache, nausea and vomiting. This was on a background of three failed renal transplants for IgA focal segmental glomerulosclerosis and recent resection of stage I renal clear cell carcinoma. Examination was consistent with opsoclonus-myoclonus-ataxia syndrome. There were no features on encephalopathy apart from mild emotional lability.. CSF results: lymphocytosis with an elevated protein, positive EBV using polymerase chain reaction and positive anti-NMDAR. Treatment: Plasma exchange, Rituximab, intravenous acyclovir for two weeks followed by oral acyclovir for 3 months. Improvement was marked, although with residual myoclonus on 2 month follow-up.

Conclusions This rare case of paraneoplastic double-positive EBV and anti-NMDAR opsoclonus-myoclonus syndrome in an immunocompromised patient demonstrates the broadening clinical phenotype of anti-NMDAR and highlights the contentious issue of EBV pathogenicity and treatment in an immunocompromised patient.

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066 CONTACTIN-1-MEDIATED CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY (CIDP) PRESENTING AS AN ACUTE CASE OF GUILLAIN BARE SYNDROME (GBS)

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New and emerging evidence describing the role of autoantibodies to nodal and paranodal proteins in the pathogenesis of chronic inflammatory demyelinating polyneuropathy (CIDP) has arisen over the past decade, with significant diagnostic, therapeutic and prognostic implications. Although rare, CIDP with anti-contactin-1 (CNTN1) IgG4 antibodies has a distinct pathogenesis and clinical phenotype that differs from both typical CIDP and its other atypical variants. Here, we report the case of a young normally well 49-year-old man from rural Australia with severe refractory anti-CNTN1-mediated CIPD who presented like a case of Guillain-Barré syndrome (GBS) who improved dramatically after chemoimmunotherapy with Rituximab and who also had an unexpected late response to subsequent treatments with intravenous immunoglobulin (IVIg). In reporting this case, we hope to highlight important considerations in the diagnosis and treatment of patients with severe refractory CIDP, and especially those patients with anti-CNTN1 seropositive disease.

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067 NEUROFILAMENT LIGHT CHAIN CONCENTRATION PREDICTS RISK OF RELAPSE IN PARTICIPANTS WITH RELAPSING MULTIPLE SCLEROSIS IN PHASE 3 OZANIMOD TRIALS

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Objectives Plasma neurofilament light chain concentration (pNfL-c) is increased in patients with multiple sclerosis (MS) and may serve as a biomarker for neurologic damage and disease activity in relapsing MS. We analyzed changes in pNfL-c and on-treatment risk of relapse with ozanimod vs interferon β-1a (IFN).

Methods In this post hoc analysis of the phase 3 SUNBEAM (NCT02294058; ≥12 months) and RADIANCE (NCT02047734; 24 months) trials, pNfL-c was measured at baseline and after 12 and 24 months of treatment with oral ozanimod 0.46 or 0.92 mg/d or intramuscular IFN 30 μg/wk. Poisson generalized linear models were used to fit the number of relapses as a function of baseline pNfL-c and treatment group with an offset for duration. Predictive modeling of expected annualized relapse rate (ARR) was calculated using median percentage change in pNfL-c from baseline.

Results At end of treatment, median pNfL-c was reduced from baseline by 20%–23% ($P<0.01$) and 23%–27% ($P\leq0.0001$) with ozanimod 0.46 and 0.92 mg, respectively, and by 13%–15% with IFN. Higher baseline pNfL-c was associated with more relapses ($P<0.0001$), and greater median reductions in pNfL-c from baseline were associated with lower ARR.