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

¹Jasmine Ashhurst, ²Rami Haddad, ³Matthew Tait. ¹St George Hospital, Sydney, NSW, Australia; ²Neurology, Orange Health Service, Orange, NSW, Australia; ³Neurosurgery, Nepean Hospital, Sydney, NSW, Australia

10.1136/bmjno-2021-ANZAN.63

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

¹Rajiv Wijesinghe, ²Craig Anderson, ²Miriam Welgampola, ³Christine Stehmann, ³Steven Collins, ¹Penelope Spring. ¹Department of Neurology, Concord Repatriation General Hospital, Concord, NSW, Australia; ²Department of Neurology, Royal Prince Alfred Hospital, Camperdown, NSW, Australia; ³Australian National CJD Diagnostic Service, Florey Institute for Neuroscience and Mental Health, Parkville, VIC, Australia

10.1136/bmjno-2021-ANZAN.64

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

¹Catherine Ashton, ²Janavi Dunuville. ¹Neurology Department, Fiona Stanley Hospital, Murdoch, WA, Australia; ²Department of neurology, Sir Charles Gairdner Hospital, Nedlands, WA, Australia

10.1136/bmjno-2021-ANZAN.65

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