did not return to pre-morbid levels. The MRI lesions resolved and did not recur.

Conclusion Focal vasculitis is rare but may result in neuronal loss and specific cortical damage and atrophy, in this case leading to embouchure dystonia.

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102 LAMBERT EATON MYAESTHENIC SYNDROME IN THE ABSENCE OF MALIGNANCY

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10.1136/bmjno-2021-ANZAN.102

Introduction We report a case of Lambert Eaton Myaesthenic Syndrome (LEMS) in an 85-year-old gentleman with no active malignancy.

Case An 85-year-old gentleman presented with a 3-month history of proximal weakness, confusion, nausea and vomiting. His medical history included gastric adenocarcinoma with curative resection 21-years ago and a 2-year history of a stable sensorimotor peripheral neuropathy. During his admission he experienced an episode of new onset fluctuating diplopia. Neurological examination demonstrated mild upper and lower limb non-fatiguable weakness. There was no detectable cranial nerve palsy.

A myasthenia antibody panel was ordered. Voltage-gated-calcium-channel-antibodies were positive (47pM) (normal range < 30pM). Repetitive nerve stimulation demonstrated an increment in compound muscle action potential of the right nasalis and right abductor digiti minimi following exercise and high-rate stimulation consistent with the clinical diagnosis of LEMS. Investigation for malignancy including tumour markers, CT chest, abdomen and pelvis, MRI-pancreas and whole body PET scan were unremarkable.

The patient underwent a 1-month period of inpatient rehabilitation and was discharged home. At 6 months, he remains well with no further episodes of diplopia or weakness. To date, no malignancy has been identified.

Conclusion LEMS in absence of an identified malignancy is an uncommon diagnosis. Those cases that have been documented are also more likely to occur in younger patients. The case we present here highlights a constellation of vague seemingly discordant symptoms with a unifying diagnosis and offers the patient a chance to be actively monitored for the development of malignancy in the future.

103 AN UNDIFFERENTIATED AUTOIMMUNE NEUROINFLAMMATORY ILLNESS ASSOCIATED WITH LOW CSF HYPOCRETIN & CENTRAL HYPOTHALAMIC DYSREGULATION

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10.1136/bmjno-2021-ANZAN.103

Introduction This case report explores a possible undifferentiated autoimmune neuroinflammatory illness presenting with recurrent fevers, abdominal pain, hypersomnolence and sleep attacks with low cerebrospinal fluid (CSF) hypocretin, and a partial response to anakinra, a human interleukin 1 receptor antagonist.

Case Presentation A 19 year old female presented with 5 years of abdominal pain and fatigue with no clear aetiology identified following extensive investigation. She subsequently was found to have recurrent fevers to 38°C, an intermittent fine, macular rash and sudden sleeping at inappropriate times. Her brain MRI was normal and (CSF) showed normal protein and no white cells, but a low hypocretin level (<200 units). Further investigations including whole exome sequencing, gastrointestinal, autoimmune and metabolic assessments, yielded limited findings. Previous therapy with colchicine had been ineffective.

Management and Outcome For a presumptive diagnosis of an undifferentiated autoinflammatory disorder, she was received prednisolone 10 mg daily for 4 weeks with no benefit. She then initiated anakinra, which improved in rash and sleep attacks. Despite initially controlling her recurrent fevers for a period of four weeks, this symptom ultimately recurred, with ongoing abdominal pain.

Discussion Low levels of hypocretin in the CSF has been associated with narcolepsy type 1 and has thought to been associated with an undefined autoimmune mechanism. It is hypothesised that her hypothalamic orexin has been altered due to these inflammatory changes leading to body temperature dysregulation and sleep disorder. Interestingly the hypersomnolence appear to have improved with anakinra, a therapy not typically used in narcolepsy.

104 OVERLAPPING AUTOIMMUNITY: A CASE OF CONCOMITANT AQUAPORIN-4 AND MYELIN OLIGODENDROCYTE GLYCOPROTEIN (MOG) ANTIBODY POSITIVITY IN NEUROMYELITIS OPTICA SPECTRUM DISORDER

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10.1136/bmjno-2021-ANZAN.104

Objectives To describe a rare case of double antibody positive Neuromyelitis Optica Spectrum Disorder (NMOSD) with both Aquaporin-4 and MOG antibodies, occurring following a Pertussis infection in a patient with a history of auto-immunity.

Methods Retrospective review of clinical records.

Results A 41-year-old Chinese woman with a history of Systemic Lupus Erythematosus presented with a sub-acute onset of progressive gait ataxia and urinary retention occurring seven days after a confirmed Bordetella pertussis infection. Magnetic resonance imaging revealed extensive subcortical and thalamic T2/FLAIR hyperintensities with subtle enhancement, and a longitudinally extensive non-enhancing spinal cord lesion (T1-T7), without optic nerve involvement. Cerebrospinal fluid protein was raised (0.55 g/L) with 7 mononuclear cells and matched oligoclonal bands. Viral PCRs were negative including JC virus and Pertussis. Established live cell-based immunoassays revealed positivity for both Aquaporin-4 antibodies (in CSF and serum) and MOG antibodies in high

titres. Our patient meets the 2015 Consensus Diagnostic Criteria for NMOSD.[1] Treatment with high dose corticosteroids and rituximab lead to clinical and radiological improvement, but she had a clinical relapse 10 months later with new LETM (T3-T7), necessitating increased immunosuppression with more rigorous rituximab dosing of 1000mg every 6 months.

Conclusions Double positivity for both Aquaporin-4 and MOG antibodies in NMOSD is rare. We describe a case of double-positive NMOSD occurring following an infective illness. This case demonstrates that NMOSD may occasionally masquerade as post-infectious Acute Disseminated Encephalomyelitis and highlights the importance of checking antibodies in these patients, given the treatment strategies and risk of relapse differs considerably.

105 ANTI-LEUCINE-RICH GLIOMA INACTIVATED 1 (LGI1) ENCEPHALITIS ASSOCIATED WITH HIGH GRADE PAPILLARY UROTHELIAL CARCINOMA

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10.1136/bmjno-2021-ANZAN.105

Introduction LGI1 encephalitis is a rare form of limbic encephalitis, that was first recognised as a primary autoimmune phenomenon, and subsequently described in association with a limited number of malignancies.[1] We report a novel case of LGI-1 encephalitis occurring concurrent to a high-grade papillary urothelial carcinoma.

Case Presentation A previously well 72-year-old male presented to a rural hospital with a first episode generalised tonic-clonic seizure, confusion and progressive behavioural change. He was diagnosed with LGI-1 encephalitis, with positive CSF antibodies, and mesial temporal T2 hyperintensity on MRI brain. There was no response to first line treatment with steroids, intravenous immunoglobulin, and mycophenolate. Malignancy screening revealed a lesion within the upper pole of the left kidney, favoured to represent a transitional cell carcinoma. Biopsy demonstrated a low grade papillary urothelial carcinoma. The patient's encephalopathy continued to worsen over a period of months, despite ongoing immunosuppression. He underwent a left nephrouretectomy, and histology demonstrated a high-grade papillary urothelial carcinoma. Subsequent to this, there was improvement in cognition and behaviour. Psychotropic and immunosuppressive medications were slowly weaned. At 9-month follow-up, the patient has returned close to baseline function, and has been clinically stable off all immunosuppressive treatment.

Conclusions LGI-1 encephalitis has previously been described in association with thymoma, lymphoma, teratoma, and more recently with lung and prostate cancer.[2 3] We believe our case is the first report of association between LGI-1 encephalitis and high grade papillary urothelial carcinoma.

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106 CONTRAST-INDUCED ENCEPHALOPATHY AFTER CARBON DIOXIDE ANGIOGRAPHY IN THE UPPER EXTREMITY AND IODINATED CONTRAST – A CASE REPORT

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10.1136/bmjno-2021-ANZAN.106

Objective Carbon dioxide (CO_2) is used as an alternative contrast agent in angiography for patients with iodinated contrast allergy or impaired renal function. CO_2 angiography is contraindicated in cerebral circulation based on demonstrated neurotoxicity in animals.[1] [2] We present a case of reversible neurological complications post CO_2 angiography and iodinated contrast.

Methods and Results A 65-year-old man presented with an ischaemic finger from steal syndrome post-arteriovenous fistula ligation, on a background of end-stage renal disease and type 2 diabetes. He underwent CO_2 angiography for evaluation of right upper limb circulation. Immediately after the first CO_2 injection into the right brachial artery, he became unresponsive and spontaneously recovered after 1-minute. Due to poor distal opacification with CO_2, 15ml of iodinated contrast was administered. At 3-hour post-procedure, he developed left facial droop, left hemiparesis and left visual neglect. CT brain, angiogram and perfusion study at 5-hour post-procedure showed no acute changes. Overnight, he worsened to dense left hemiplegia. Non-contrast CT brain at 11-hour post-procedure showed oedema and hyperdensity in the right hemisphere. He had a seizure on day 1 post-procedure. MRI brain performed 24-hour post-procedure showed dramatic resolution of right hemispheric cerebral oedema with no diffusion restriction. All neurological deficits completely resolved 7-day post-procedure.

The CO_2 which refluxed into the cerebral circulation from the brachial artery, caused the breakdown of blood-brain barrier, allowing penetration of iodinated contrast and subsequent right hemispheric cerebral oedema.

Conclusions This case highlights the risk of air embolism and neurotoxicity of CO_2 angiography and the rare occurrence of contrast-induced encephalopathy.

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107 A TAIL OF TWO NMDA RECEPTOR ANTIBODY ENCEPHALITIDIES: AGGRESSIVE TREATMENT, DIVERGENT OUTCOMES

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10.1136/bmjno-2021-ANZAN.107