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

Jessica Stabler, David Williams. *John Hunter Hospital, Newcastle West, NSW, Australia*

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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

[1,2]Hue Mun Au Yong, [3]Matthew K Ligtermoet, [3]Douglas Crompton. *[1]Department of Neurology, Alfred Health, Melbourne, VIC, Australia*; *[2]Department of Neurology, Royal Melbourne Hospital, Parkville, VIC, Australia*; *[3]Department of Neurology, Northern Health, Epping, VIC, Australia*

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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

[1]Christopher Blair, [2,3]Nicolas Urriola, [1]Leon Edwards, [1]Patrick Aouad. *[1]Department of Neurology and Neurophysiology, Liverpool Hospital, Liverpool, NSW, Australia*; *[2]Department of Immunology, Liverpool Hospital, Liverpool, NSW, Australia*; *[3]Department of Immunology, Royal Prince Alfred Hospital, Camperdown, NSW, Australia*

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