demonstrated thickening and FLAIR signal hyperintensity of the Corpus Callosum. Spinal imaging showed a large longitudinal intradural extramedullary lesion posteriorly at C2 and a smaller lesion anteriorly around the C1 region. Lymph node biopsy revealed numerous non-caseating epithelioid cell granulomas, with no evidence of mycobacterium suggestive of neurosarcoidosis.

Neurosarcoidosis should be considered in patients who present with symptoms suggestive of demyelinating or a stroke like episode(s). Early treatment is crucial for preventing disease-related morbidity and highlights the importance of clinician awareness of neurosarcoidosis and its diversity in presentation.

2704 DEMYELINATING DUO: TWO CASES DEMONSTRATING THE SPECTRUM OF TNF-α INHIBITOR-RELATED DEMYELINATION

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Objectives Tumour necrosis factor (TNF)-α inhibitors are immunosuppressants used in autoimmune conditions. Rarely, they may be implicated in central and peripheral demyelination. We present two cases highlighting this spectrum.
Methods Case report.
Results The first case is of a 48-year-old man who presented with three months of weakness in his upper and then lower limbs, occurring on a background of ulcerative colitis on infliximab. Nerve conduction studies revealed reduced compound muscle action potentials across multiple nerves in the upper and lower limbs, including proximally, in-keeping with multiple foci of conduction block. GM1 antibodies were negative. With an impression of multifocal motor neuropathy with conduction block, he was commenced on intravenous immunoglobulin and made a marked clinical recovery. Infliximab was changed to vedolizumab for his ulcerative colitis.

The second case is of a 45-year-old female who presented with a relapsing syndrome of diplopia, dysarthria, and left facial and right hemi-body sensory changes, occurring on a background of rheumatoid arthritis on adalimumab. MRI showed enhancing and non-enhancing demyelinating lesions in the supratentorium, brainstem and spinal cord. With an impression of multiple sclerosis-like central demyelination, she was given pulsed intravenous methylprednisolone before rituximab was commenced as dual treatment for this and her rheumatoid arthritis, with eventual marked clinical improvement.
Conclusions TNF-α inhibitors are used widely for a range of autoimmune conditions but can rarely be implicated in demyelinating disease.[1] Our cases highlight the spectrum of central and peripheral demyelination that can occur with this therapy, and how management can be safely navigated.

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2705 CHARACTERISING THE USE OF OFATUMUMAB- A SECONDARY USE OF DATA STUDY CHARACTERISING OFATUMUMAB UTILISATION IN RELAPSING MULTIPLE SCLEROSIS PATIENTS (EAFTOS)

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Objectives Ofatumumab is approved in Australia for the treatment of adults with relapsing forms of multiple sclerosis (RMS). The objective of this study is to characterise the onboarding experience and utilisation of ofatumumab in RMS patients in Australia.
Methods Patients commencing ofatumumab and registered in the digital patient support program, MSGo, were given the opportunity to enroll in the retrospective, longitudinal secondary use of data analysis. The primary endpoint is the proportion of doses not completed within three days of the expected date during initiation and +/- 14 days during the first three months of maintenance. Key secondary endpoints assess the profile of the patients initiating ofatumumab and factors influencing compliance to treatment.
Results The interim analysis (Data-cut: 29th Jun 2022) included 235 de-identified patients. The baseline characteristics showed 23% of registered patients were treatment naive. The majority of patients completed initiation dose 2 and 3 within 7 days ±3 days from the previous dose within the expected timeframe (proportion 0.985, 95% CI 0.96–0.997). The proportion of adherent doses completed during maintenance doses 2 and 3 within 28 days ±14 days from the previous dose, was 0.977 (CI 0.94- 0.994) and 0.981 (CI 0.95–0.996) respectively.
Conclusions This study provides insight into the characteristics of patients initiating ofatumumab in Australia. These data show high rates of adherence during initiation and the first 3 months of maintenance. Further data from the EAFToS study will aid in the understanding of the real-world experience in Australia for ofatumumab patients.

2709 CASE REPORT: IDIOPATHIC BILATERAL PHRENIC NEUROPATHY CAUSING BILATERAL DIAPHRAGMATIC PARALYSIS

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Background Idiopathic bilateral phrenic neuropathy leading to bilateral diaphragmatic paralysis is a rare cause of respiratory failure. Patients with this condition often present with orthopnoea, dyspnoea on exertion, and sleep disturbance. Prognosis is usually poor.
Case Description A 69-year-old Caucasian female presented with acute onset of chest pain and dyspnoea. Initial investigations ruled out acute myocardial infarction or pulmonary embolus. Three days later, patient was found obtunded with severe type 2 respiratory failure, which required urgent

intubation. She was extubated on the same day and transitioned to non-invasive ventilation (NIV). Day 3 post extubation, her sniff test revealed no movement of the bilateral hemidiaphragms. She was urgently re-intubated. There was no history of pain, prodromal illness, trauma, COVID infection or vaccinations. Her past medical history is significant for mild COPD and hypertension. Her neurological examination was unremarkable. Her CSF studies were unremarkable, and so was her MRI Brain and cervical spine result. Nerve conduction studies had shown absent phrenic motor response and normal upper and lower limb motor and sensory study. Patient received 5 days of plasma exchange. She was successfully extubated and transitioned to NIV afterward. Nine days later, she also had right-sided diaphragmatic plication. Patient had excellent clinical response and she was weaned off NIV gradually.

Conclusion There is no established guideline for treatment of idiopathic bilateral phrenic neuropathy. Supportive therapy with NIV is the mainstay of treatment. This case highlights plasma exchange in conjunction to unilateral diaphragmatic plication as an effective treatment for idiopathic bilateral phrenic neuropathy.

2710 EFFECTS OF PURINERGIC P2X RECEPTOR 7 (P2X7R) INHIBITION IN GLIOBLASTOMA

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Objectives Glioblastoma is the most common and aggressive form of primary brain cancer with a median survival of 15 months from diagnosis. The purinergic receptor P2X7 (P2X7R) is a regulator of several cell signalling pathways, and its expression is upregulated in glioblastoma. This study examined the expression and function of P2X7R in a human glioblastoma cell line, U251 and utilised a pharmacological antagonist of P2X7R, AZ10606120, to inhibit receptor function and delineate downstream consequences of receptor inhibition.

Methods P2X7R expression and function in the U251 cell line was determined using immunocytochemistry, live cell Ca^{2+} imaging and live cell dye uptake assays. Effect of AZ10606120 on cell viability was investigated via cell counts and LDH assay. The mode of tumour cell death was subsequently investigated via annexin V and cleaved caspase-3 staining, and multiplex RNA analysis.

Results P2X7R was expressed and functional in U251 cells. AZ10606120 treatment72 significantly decreased tumour cell number ($p<0.0001$), and significantly increased tumour cell death, as evidenced by increased LDH release ($p<0.001$). This was concentration-dependent, modelled by a least squares linear regression ($R^2 = 0.8221$). No difference was observed in annexin V or cleaved caspase 3 staining, indicating minimal apoptosis occurring upon AZ10606120 treatment. Multiplex mRNA analysis demonstrated downregulation of genes associated with apoptosis, pyroptosis and necroptosis. Inhibition of P2X7R using small interfering RNA significantly decreased cell number and increased extracellular LDH.

Conclusions This study describes findings of significant translational potential in neurooncology. It highlights AZ10606120 related P2X7R inhibition as a novel therapeutic target in glioblastoma.

2714 CASE REPORT: A FIRST PRESENTATION OF MITOCHONDRIAL ENCEPHALOPATHY, LACTIC ACIDOSIS, AND STROKE-LIKE EPISODES (MELAS) MIMICKING ACUTE ENCEPHALITIS

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Case A 51-year-old woman presented with focal motor seizures and confusion. Relevant history included diabetes mellitus and sensorineural hearing loss. She was given intravenous levetiracetam, and acyclovir for suspected encephalitis. She required admission to ICU for agitation. Serum lactate was 7.2 mmol/L, attributed to seizures. Lumbar puncture was acellular with raised protein (0.64 g/L) and glucose (6.3 mmol/L). Gram stain and culture showed no organisms, whilst multiplex PCR and limbic encephalitis/antineuronal antibodies were negative. EEG showed right lateralised periodic discharges. MRI brain showed FLAIR hyperintensity in the right temporal lobe.

A maternal family history of dementia, diabetes, and hearing loss was elicited. On serial CSF, lactate was raised at 5.1 mmol/L. Mild proximal weakness was detected, with normal creatinine kinase. MR spectroscopy showed a parietal lobe lactate peak. Serum biomarkers of mitochondrial disease showed elevated GDF15 (3180 ng/L; normal<1470 ng/L) with normal FGF21 (238 ng/L; normal<360 ng/L. The m.3243A>G mutation was detected, confirming a diagnosis of MELAS, with heteroplasmy levels of 14% in blood and 63% in urine.

Focal seizures were controlled with levetiracetam, with confusion resolving within 24 hours of seizure control. She has remained well on levetiracetam & coenzyme Q10 with no further neurological events for 12 months.

Conclusion MELAS is likely underdiagnosed, with the m.3243A>G variant found in ~1 in 400 people.[1] It can present later in life, including mimicking acute encephalitis. A personal or family history of diabetes, hearing loss, or myopathy should alert clinicians to the possibility of MELAS, especially when other encephalitides are excluded.

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2715 SURVEY OF HEADACHE EDUCATION AND TRAINING EXPOSURE AMONGST NEUROLOGY ADVANCED TRAINEES IN NEW SOUTH WALES AND VICTORIA

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