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.

REFERENCE

1. Manwaring N, Jones MM, Wang JJ, Rochtchina E, Howard C, Mitchell P, Sue CM. Population prevalence of the MELAS A3243G mutation. *Mitochondrion* 2007;**7**:230–233. doi:10.1016/j.mito.2006.12.004

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

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Background Headache remains a major public health concern, with patients experiencing difficulty accessing optimal, evidence-based care.
Aim To evaluate gaps in education and training in headache amongst Neurology Advanced Trainees in New South Wales and Victoria.
Methods An eighteen-question survey was created using REDCap software. The survey was distributed to New South Wales and Victorian Neurology Core Trainees with a participation information sheet explaining the aims of the study, that participation was voluntary and that respondents would remain anonymous. Data were collected from Sept-October 2022.
Results The response rate was 30% (n = 22/77). Respondents comprised 50% first core year (AT1) and 50% second core year trainees (AT2). Amongst the respondents, 59% had less than 2 hours of exposure to headache education throughout their university degree and 69% had no exposure to headache clinics, and only 14% felt adequately prepared to manage headache disorders in either the outpatient or inpatient setting.
Conclusions Few neurology advanced trainees who responded to the survey felt adequately prepared to manage headache disorders in the inpatient or outpatient setting. Gaps in education and training were identified from medical school through to advanced training. Addressing these gaps is an avenue to optimize the management of headache disorders in Australia.

2716 A COMMON VISUAL COMPLAINT HERALDING UNDERLING MALIGNANCY

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Introduction Neurological complications of haematological malignancies are commonly reported. However, they are less frequently the presenting complaint. Neurological presentations of haematological disease are often difficult to localise and signs are complicated by treatments to treat possible differential diagnoses (McKee & Li, 2022). The Neurologist needs to be aware of these when faced with the complex undifferentiated patient.
Case Summary This report documents the diagnostic dilemma of a 78 year old male presenting with an atypical history of painful diplopia, headache and recurrent intermittent episodes of acute, severe pain affecting all four limbs proximally. Past medical history was significant for ischaemic heart disease and previous prostate cancer cured with radical prostatectomy. Investigations showed mildly raised ESR at 45mm/hr and neutropaenia (Neutrophils: 0.6 $x10^9$). Prostate Specific Antigen (PSA) was < 0.01ug/L. Due to temporal headache, visual symptoms and pain, treatment for temporal arteritis was initiated with prednisolone 1mg/kg/day. Temporal artery biopsy was normal and subsequent radiological investigations revealed a pituitary mass, widespread sclerotic bone lesions, and diffuse bone marrow uptake on nuclear scintigraphy. With a normal PSA, haematological processes were considered. While initial comprehensive computerised tomography was normal, following steroid cessation FDG-PET demonstrated widespread subcutaneous lymphadenopathy. Bone marrow biopsy confirmed diffuse large B cell lymphoma.
Conclusion This case raises the difficulty in neurological presentations of haematological disease and illustrates the dichotomy of symptomatic treatment and diagnostic accuracy. While neurologists are experienced in therapeutics for known malignant disease atypical, difficult to localise symptoms should necessitate sufficient investigation and consideration of systemic disease.

REFERENCE

1. McKee Z, Li Y. Unusual neurological presentations resulting in diagnosis of lymphoma in three patients. *RRNMF Neuromuscular Journal*, 2022;**3**(3):25–28, viewed 10 February 2023 https://journals.ku.edu/rrnmf/article/view/17930/16630

2717 HEADACHE WITH CONCURRENT EAGLE'S 'JUGULAR' SYNDROME, CONTRASTING OUTCOMES TO VASCULAR INTERVENTIONAL TREATMENT: A CASE SERIES

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Objective Eagle's 'jugular' syndrome is a rare cause of headache, related to an elongated styloid process impinging the internal jugular vein (IVJ) and altering dynamic intracranial pressure. This case series reports two cases of Eagle's 'jugular' syndrome and the use of a minimally invasive, vascular surgical technique as management.
Case The first case is of a 78 year old male with a two week history of bilateral retro-orbital pain that radiated into the neck and shoulders and was increased by turning the head. CT venogram and MRI venogram demonstrated bilateral IVJ stenosis with no other significant pathology. A staged bilateral IVJ stent was inserted and there was complete resolution of the pain. The second case is of a 49 year old with a 3 month history of bifrontal pain with temporal radiation. Photophobia, phonophobia and left facial numbness were also reported. No papilloedema was present but lumbar puncture opening pressure was 28 cm of water, with acellular CSF. CT venogram and MRI venogram demonstrated bilateral IVJ stenosis with no other significant pathology. A right IVJ stent was inserted. Whilst a post-procedure LP demonstrated a lower opening pressure of 20 cm, the patient reported no improvement in headache symptoms.
Conclusion With contrasting outcomes, these cases provide valuable insight into the criteria for diagnosis and treatment for patients suffering from headache due to Eagle's 'jugular' syndrome.

2720 TIA IN THE EMERGENCY DEPARTMENT: RATE OF SUBSEQUENT STROKE AND DIAGNOSTIC CORRELATION WITH NEUROLOGY OUTPATIENT CLINIC

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Background Data on the risk of stroke after a TIA is mostly derived before the era of routine upfront CT angiography and improved pharmacotherapies. Differences between emergency