**Presentation and Clinical Findings** A 67 year old male presented to the emergency department with a 24 month history of progressively worsening, severe neuropathic pain in the left lower limb, weakness of ipsilateral ankle dorsiflexion, and associated gait disturbance.
**Diagnosis and Intervention** Initial serum biochemistry was unremarkable. A paraneoplastic screen, lymphocyte surface markers, and tumour markers were negative. Cerebrospinal fluid analysis demonstrated raised proteins of 0.69 g/L without neoplastic cells visualised on cytology. Gadolinium enhanced magnetic resonance imaging (MRI) demonstrated thickening and enhancement of the cauda equina, L5, S1 and S2 nerve roots. Marked hypermetabolism within the same nerve root distribution was observed in $^{18}$fluorodeoxyglucose ($^{18}$FDG) positron emission tomography (PET). A biopsy of the L5 nerve root was performed histopathology revealed lymphocytic infiltrate. Immunohistochemistry of the specimen was positive for B-lymphocyte antigen CD20, B-cell lymphoma 2 (Bcl-2) and multiple myeloma 1 (MUM1). These features were consistent with diffuse large B-cell lymphoma (DLBCL).
**Outcomes** The patient subsequently underwent chemotherapy with R-CHOP, and went into remission following one cycle.
**Conclusion** Primary neurolymphomatosis presents a diagnostic challenge and as such formal diagnosis is often delayed. Whilst biopsy is the gold standard for diagnosis, gadolinium enhanced MRI and $^{18}$FDG-PET are useful in characterising lesions and determining feasibility of biopsy.

## 2681 TRIGEMINAL NEURITIS DUE TO EMTRICITABINE/ TENOFOVIR FOR HIV PRE-EXPOSURE PROPHYLAXIS

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Toxic peripheral neuropathies have been described, and are typically cumulative in their pathophysiology. Cranial neuropathies secondary to medication toxicity are extremely rare. Use of emtricitabine/tenofovir as pre-exposure prophylaxis (PrEP) is recommended as standard of care for people at risk of HIV infection. Cranial neuropathies as an adverse effect of this drug have only been described once in the literature (Van Slyke, 2018). We present the case of a 22-year-old information technology worker who developed acute right trigeminal neuritis within 24 hours of initiating emtricitabine/tenofovir. MRI with gadolinium contrast demonstrated abnormal T2 signal hyperintensity and enhancement affecting the maxillary and mandibular divisions of the right trigeminal nerve, with the ophthalmic division involved to a lesser degree. Symptoms resolved within 6 weeks following medication cessation and repeat MRI imaging showed near resolution of enhancement. Despite advice, the patient rechallenged the medication and within 24 hours his symptoms recurred. The proposed mechanism of trigeminal neuropathy is a toxic neuritis due to tenofovir, with some studies showing modulation of mitochondrial biogenesis and inflammatory pathways (Fields, 2019).

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## 2683 A PERFECT MINI-STORM: UNCOMMON AETIOLOGY FOR A COMMON PRESENTATION OF BELL'S PALSY

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**Objective** To discuss a rare presentation of bilateral facial nerve palsy in a 32 year old Australian female with Lyme neuroborreliosis, SARS-CoV-2 and positive Epstein-Barr virus (EBV) serology.
**Case** A 32 year old female presented to the emergency department with a right sided, lower motor neuron (LMN) facial palsy in the setting of a recent viral upper respiratory tract infection. Magnetic resonance imaging of the brain showed enhancement of the right facial nerve. She was diagnosed with Bell's palsy and given a short course of oral prednisone. She re-presented 11 days later having developed a left sided LMN facial palsy. Serum EBV viral capsid antigen (VCA) IgM was equivocal in the setting of both VCA and nuclear antigen IgG positivity.

Further history revealed a recent SARS-CoV-2 infection and travel to the USA and Canada. Cerebrospinal fluid (CSF) analysis showed a lymphocytosis but negative EBV polymerase chain reaction. She was treated with further steroids and antiviral therapy. Her travel to Borrelia endemic areas prompted empiric treatment with doxycycline and testing which confirmed a diagnosis of Lyme neuroborreliosis with positive Borrelia IgG and IgM. Immunoblot was positive in both serum and CSF. The patient has made a near-complete recovery.
**Conclusion** Bilateral Bell's palsy has been reported with Lyme neuroborreliosis, SARS-CoV-2 and EBV infection previously, but this is the first case to report co-infection. This case highlights the importance of tailoring investigations to the clinical context and serves to remind clinicians of the value of a travel history.

## 2684 TWO FORMS OF NEUROPATHY ASSOCIATED WITH IMATINIB THERAPY: A CASE REPORT

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**Introduction** Imatinib is a tyrosine kinase inhibitor (TKI) widely used in the treatment of chronic myeloid leukaemia and other malignancies in which tyrosine kinases are over expressed. The first case of neuropathy associated with imatinib was reported in 2011.[1] A distal mixed axonal neuropathy is now recognised as an uncommon late adverse effect of imatinib,[2] however other types of neuropathy have not been described previously. We report a case in which both a mixed axonal neuropathy and an acute, relapsing, steroid responsive neuroplexopathy occurred.

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**Case description** A 73-year-old man with CML and JAK2 positive myeloproliferative neoplasm was treated with imatinib and pegylated interferon. In 2020 he developed an acute neuropathy affecting the left leg. MRI showed abnormal signal and enhancement in the sciatic trunk and related nerves. There was no improvement following withdrawal of interferon but rapid improvement following methylprednisolone. A mild distal mixed axonal neuropathy was present at that time. In October 2022 he developed an acute common peroneal neuropathy in the other leg and nerve conduction studies showed marked worsening of the generalised axonal neuropathy. As no other cause was found imatinib was ceased. Marked symptomatic improvement occurred within 6 weeks.

**Discussion** This is the first report of a relapsing steroid responsive neuroplexopathy associated with TKI therapy. It suggests that TKIs may rarely be associated with an inflammatory neuropathy, as well as with a generalised mixed axonal neuropathy. As TKIs are widely used in haematological and other malignancies, awareness of this treatable complication is important.

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## 2685 DEVELOPMENT OF THE MSBASE IMAGING REPOSITORY (MSBIR)

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**Objectives** To create and integrate a dedicated MSBase Imaging Repository (MSBIR) with the MSBase registry. To recruit MSBase sites to contribute to MSBIR. To facilitate quantitative analysis of brain MRI scans uploaded to MSBIR using an automated AI-based software platform and transmit these metrics into MSBase.

**Methods** Following stake-holder consultation, technical work commenced on the MSBIR build by Radiologics (Extensible Neuroimaging Archive Toolkit [XNAT] experts), Sydney Neuroimaging Analysis Centre (SNAC) & University of Sydney (USYD). Multiple sites were contacted regarding contribution to MSBIR. Automated, AI-based imaging pipelines measuring cross-sectional and longitudinal brain lesion and volume metrics from compatible clinically-acquired multiple sclerosis (MS) MRI scans were developed and refined by SNAC.

**Results** The customised MSBIR-XNAT production release was deployed on USYD hosted Amazon Web Services servers. SNAC developed products support the platform: (i)TORANA™, medical image gateway service that de-identifies images over secure/encrypted protocols, (ii)COEUS™, advanced-search web portal, allows data retrieval for MSBase projects. Currently 7 sites have contributed 16235 MRI scans to MSBIR. Fully-automated quantitative analysis pipelines have been developed and implemented. Quantitative MRI brain lesion and volume metrics are available for compatible scans. All metrics are: (i)Stored in MSBIR/MSBase (de-identified data), (ii)Displayed in corresponding MSBase patient records.

**Conclusions** MSBIR-XNAT has been deployed and integrated with MSBase. Imaging data ingestion and further site/subject recruitment is ongoing. Compatible MRI brain scans entering MSBIR are automatically analysed by imaging pipelines and quantitative data stored in MSBIR and displayed in MSBase patient records. Clinical-imaging MS research collaborations utilising MSBIR are underway.

demonstrates a rare case of intramedullary spinal cord metastasis from melanoma with an acute thoracic cord syndrome.

## 2689 HYPERTROPHIC PACHYMENINGITIS IN SETTING OF RELAPSING POLYCHONDRONDITIS – A MANAGEMENT ISSUE

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**Introduction** We present a patient with an aggressive, relapsing course of hypertrophic pachymeningitis on a background of relapsing polychondritis. He is currently stable on high dose corticosteroids and cyclophosphamide This case adds to literature about this uncommon co-occurrence of hypertrophic pachymeningitis in patients with relapsing polychondritis and its management difficulty.

**Case report** A 53-year-old-male presented to us with left upper limb weakness, headaches and confusion. This was on a background of relapsing polychondritis, obesity, obstructive sleep apnoea and type 2 diabetes mellitus. For his relapsing polychondritis, he had tried multiple immunosuppressants in the past, and was on prednisolone at the time of our initial review. On examination he had bilateral papilloedema. MRI brain showed hypertrophic pachymeningitis. Serial lumbar punctures showed markedly elevated opening pressures, protein levels and aseptic pleocytosis. Extensive work up of bloods, CSF analysis, imaging and pachymeningeal biopsy excluded secondary causes such as malignancy, IgG4 disease, ANCA-associated vasculitis, VEXAS syndrome and infection. Clinical exacerbations have responded to high-dose corticosteroids but continued to occur whilst on tocilizumab and adalimumab. He is currently stable on a combination of oral prednisolone and cyclophosphamide.

**Conclusion** Non-IgG4 and non-ANCA-associated idiopathic hypertrophic pachymeningitis can be difficult to diagnose and manage. Relapsing polychondritis has been associated with aseptic meningitis, often with markedly elevated CSF protein, as in our case. Hypertrophic pachymeningitis is rarely described with relapsing polychondritis and can be challenging to manage needing aggressive immunomodulatory therapy.

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