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.