Background Syphilis is increasing in prevalence in the community.[1] [2] Neurosyphilis has protean manifestations making recognition, diagnosis and early initiation of treatment challenging.

Methods/Results We present a case of early syphilitic meningitis in a 37-year-old female presenting with multiple cranial neuropathies (V, VI, VII, VIII and XII) developing over the course of two weeks. This began with a sensation of disequilibrium and unsteady gait, and progressed to difficulty closing both eyes, right lip numbness, bilateral hearing impairment (right, followed by left), and dysarthria. She did not report headache, meningism, features of primary syphilis infection or risk factors for sexually transmitted infections (STIs). Examination confirmed the presence of right-sided trigeminal, bilateral abducens, facial, vestibulocochlear and hypoglossal nerve palsies. Cerebrospinal fluid (CSF) examination was inflammatory (protein 1.28 g/L, glucose 3.8mmol/L) with predominant lymphocytosis (76%, WCC 441 x 106/L). Magnetic resonance imaging (MRI) demonstrated post-contrast enhancement of the trigeminal nerve at the pons, as well as facial and vestibulocochlear nerves at the geniculate ganglion with no leptomeningeal enhancement. Our patient was diagnosed with neurosyphilis on serum and CSF serological testing (Serum Treponema pallidum particle agglutination assay (TPPA) positive, chemiluminescent microparticle immunoassay (CMIA) IgG and IgM positive, rapid plasma reagen 1:32. CSF TPPA positive, Venereal Disease Research Laboratory test titre of 1:8). She was treated with intravenous benzylpenicillin with rapid improvement in her cranial neuropathies.

Conclusions This is the most extensive cranial neuropathy reported with syphilitic infections to date. Neurosyphilis should be considered as a differential in patients presenting with multiple cranial neuropathies.

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046 MR-BASED INTRAMUSCULAR FAT FRACTION ASSESSMENT IN HEREDITARY SENSORY NEUROPATHY TYPE 1

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Objectives Hereditary sensory neuropathy type 1 (HSN1) is a rare progressive neuropathy characterised by profound sensory dysfunction, often accompanied by significant weakness. Muscle magnetic resonance (MR) imaging with determination of intramuscular fat accumulation has been proposed as a marker of progression in this condition and we aimed to investigate this further.

Methods Calf-level muscle MR images were acquired longitudinally over three years in patients with genetically confirmed HSN1 due to *SPTLC1* and *SPTLC2* mutations. These patients were part of a larger trial of L-serine supplementation as a candidate therapy and MR images were acquired at baseline and while on treatment. Individual muscles and muscle groups were manually segmented at two cross-sectional levels in the proximal calf. Intramuscular fat accumulation and muscle atrophy were assessed.

Results Detailed MR analysis was performed in a preliminary series of three patients. We demonstrated an average annual change in MR-based intramuscular calf fat fraction of 3.2%, 1.0% and 3.7% at a cross-sectional level 130mm below the tibial plateau and 2.0%, 0.6% and 1.2% at a cross-sectional level 25% of the tibial plateau-medial malleolus distance. The degree of muscle atrophy did not significantly change. There appeared to be ongoing progression of disease during this short duration of L-serine supplementation.

Conclusion MR-based intramuscular calf fat fraction can be used to monitor progression in HSN1 and has potential utility in clinical trials. Technical limitations to this technique may be overcome using volumetric imaging with automated muscle segmentation in the future. Further investigation of L-serine supplementation is required.

047 RARE LATE ONSET NEUTROPENIA IN A PATIENT WITH MULTIPLE SCLEROSIS TREATED WITH OCRELIZUMAB AND REVIEW OF LITERATURE

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Introduction Ocrelizumab is a recombinant humanised monoclonal antibody that selectively depletes CD20 expressing B lymphocytes. Ocrelizumab was approved in Australia for use in relapsing-remitting and primary-progressive multiple sclerosis in July 2017. Rare incidences of late onset neutropenia with ocrelizumab have been reported (3 cases worldwide, none in Australia).

Case A 40-year-old man of Lebanese descent with multiple sclerosis, diagnosed 11 years ago and stabilized with 600 mg 6-monthly ocrelizumab since March 2018, presented in November 2019 with lethargy and myalgia, two weeks after his fourth cycle of ocrelizumab. Clinical examination was unremarkable. Full blood count showed white cell count of 2.35x109/L and absolute neutrophil count of 0.1x109/L with normal lymphocyte count, peripheral blood film, haemoglobin and platelet count. Serum iron studies, B12, folate, C-reactive protein, thyroid function were normal and so was the chest x-ray. The urine microscopy showed sterile pyuria. Electrolytes, liver function and renal function were normal. The nasopharyngeal viral swabs were negative. MRI brain-cervical spine showed stable old plaques and no enhancement with contrast. He had no other co-morbid condition and was not taking any other prescribed or over-the-counter medications. The neutrophil count improved to 7.68x109/L in 48 hours after Filgrastim 400 mcg subcutaneously. Ocrelizumab was ceased.

Conclusion Late onset severe neutropenia is rarely reported with ocrelizumab. In our case, the neutropenia occurred after 1.5 years of ocrelizumab use and the mechanism remains unclear. The case raises the issue of ocrelizumab re-exposure vs cessation in these patients and highlights the importance of monitoring of serial blood count.