microvascular change in 15%; mood disturbance in 15%; medication in 15%; alcohol in 4% and AD in 4%. Compared with the Epilepsy group, the AD group had a lower Addenbrookes Cognitive Examination III (ACE-III) score (79.3 ± 10.8 versus 87.5 ± 6.5 , $p=0.01$); specifically in the attention, memory and visuospatial subdomains ($p=0.004$, $p=0.002$ and $p=0.02$) but not fluency and language subdomains ($p>0.05$); and lower scores on additional assessments of naming, visuospatial and executive function ($p \leq 0.001$). The AD group had more abnormal metabolism in the temporal, parietal and occipital lobes than the Epilepsy group ($p=0.02$, $p=0.006$ and $p=0.005$).

Conclusion Patients with late onset epilepsy and cognitive complaints rarely have dementia diagnosed at their first neuropsychological assessment and tend to have milder cognitive impairment than patients with AD. The two groups can be differentiated by their neuropsychological and FDG-PET profiles.

REFERENCE

1. Sen A, Capelli V, Husain M. Cognition and dementia in older patients with epilepsy. *Brain* 2018;**141**(6):1592–1608.

Poster abstracts

042 A CASE OF ISOLATED MUSCULOCUTANEOUS NERVE INJURY FOLLOWING SKYDIVING SIMULATION

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Background Isolated musculocutaneous nerve injuries are rare, and mostly iatrogenic or traumatic.

Case Presentation We present a case of isolated musculocutaneous neurapraxia in an otherwise well young woman following uncomplicated simulated skydiving.

Management and Outcome While the injury was quite debilitating, complete neurological recovery occurred within two months without any intervention.

Discussion This case illustrates a rare pattern of neurological injury, caused by a recreational activity growing in popularity. The pattern of injury mimics that of an upper trunk brachial plexopathy or C5/6 radiculopathy. Increased awareness of the injury avoids misdiagnosis and affords the opportunity for prevention.

043 NEUROLOGICAL MANIFESTATIONS IN RHEUMATOLOGICAL DISEASE: A CASE SERIES I

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Objective Rheumatology encompasses a broad range of multi-systemic, autoimmune and inflammatory disorders. Neurological manifestations of these diseases are not uncommon. Neurological findings may predate rheumatological findings or may emerge months to years post initial diagnosis. Rheumatological diseases presenting as neurological syndromes can cause diagnostic challenges.

Methods/Results We present a range of rheumatological cases with unusual neurological presentations that demonstrate this point including; C2-C3 facet arthropathy in Diffuse Scleroderma, Granulomatosis with polyangiitis manifesting with craniofacial involvement, pseudo vasculitis associated cerebrovascular events, SAPHO syndrome with a thoracic syrinx, Neuro- Bechet's vasculitis with tumour-like CNS lesions, Platybasia in Paget's disease and others.

Conclusions Familiarity with the neurological manifestations of rheumatologic diseases is important for both rapid diagnosis and appropriate intervention.

044 METABOLIC SYNDROME IN A NEW ZEALAND GLIOBLASTOMA COHORT 2005–2020: A RETROSPECTIVE ANALYSIS AND REVIEW OF THE LITERATURE

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Background Glioblastoma (GBM) is an aggressive form of glioma. Even with standard-of-care Stupp protocol (surgery, radiotherapy, and temozolomide), median overall survival is only 10-12 months in population-based studies. Metabolic reprogramming is a hallmark of glioblastoma, with energy metabolism aberrantly geared towards aerobic fermentation. The prevalence of metabolic syndrome is 16% in the general NZ population and 32% in the Maori population.

Objectives We aimed to determine 1) if metabolic syndrome was more prevalent in our GBM cohort compared to general NZ population 2) if metabolic syndrome was associated with worse overall survival in GBM 3) if ethnicity influenced survival outcomes.

Methods We performed a retrospective analysis of 170 patients diagnosed and treated for GBM between 2005–2020 in one institution. Clinical and biochemical data relating to metabolic syndrome were collected. Overall survival was determined from the date of initial a surgical diagnosis to the date of death or data acquisition.

Results 18.2% of GBM patients met the criteria for metabolic syndrome, 27.7% of Maori and 16.1% of European New Zealanders. Patients with metabolic syndrome had statistically significant worse overall survival compared to those patients without metabolic syndrome regardless of treatment [mean 9.7 vs 18.4 months] $p=0.016$ ($p \leq 0.05$). Power was too low to comment on the prevalence of metabolic syndrome or ethnicity.

Conclusion Our study demonstrates that metabolic syndrome is associated with statistically significant poorer outcome in GBM patients. Consequently, this data will provide a control group for our current prospective study investigating the anti-neoplastic effects of metabolic therapies in GBM.

045 MULTIPLE CRANIAL NEUROPATHIES IN A PATIENT WITH SYPHILITIC MENINGITIS

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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 reagent 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.

REFERENCES

1. Kojima N, Klausner J. An update on the global epidemiology of syphilis. *Curr Epidemiol Rep* 2018;5(1):24–38.
2. Ghanem K, Ram S, Rice P. The modern epidemic of syphilis. *N Engl J Med* 2020;382(24):2379–80.

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 $2.35 \times 10^9/L$ and absolute neutrophil count of $0.1 \times 10^9/L$ 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 $7.68 \times 10^9/L$ 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.