followed by plasma exchange. Then given high dose oral corticosteroids with slow tapering and intravenous Cyclophosphamide four weekly. Significant neurological improvement noted over next 8 weeks with patient being alert and participating in ongoing multidisciplinary rehabilitation for hemiplegia.

Conclusion A case of cerebral biopsy confirmed, CSF and serum antibody-negative encephalitis is presented.

078 SENSORY NERVE ABNORMALITIES IN MOTOR NEURON DISEASE

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Introduction Electrodiagnostic evaluation is crucial in establishing the diagnosis of motor neuron disease (MND) and excluding other pathologies. It is recommended that sensory nerve conduction studies (NCS) include the ulnar and sural nerves, and generally accepted that sensory nerves are normal in MND. There are however previous reports in the literature documenting variable sensory abnormalities in patients with MND. We sought to determine the frequency of unexplained sensory abnormalities seen on NCS in patients with MND.

Methods Medical records of patients attending our tertiary MND clinic over a 2 year period were reviewed. We identified 92 patients with a clinical diagnosis of MND for whom electrodiagnostic studies were available to review. Sensory abnormalities in patients without a clear underlying aetiology (eg. compressive neuropathies, diabetes) were considered unexplained.

Results Unexplained sensory abnormalities were detected in at least one nerve in 18/92 (20%) patients. In 17 of those 18 patients, the ulnar sensory response was abnormal. 12 of 18 patients demonstrated abnormalities in 2 or more sensory nerves. Sensory abnormalities were present in 4 of 37 (10.8%) patients with bulbar onset MND and 14 of 55 (25.4%) patients with limb onset MND. Sensory symptoms were infrequently reported and did not correlate with abnormalities found on NCS.

Conclusions Unexplained sensory nerve action potential abnormalities are not uncommon in MND, with ulnar sensory responses the most frequently affected. These findings raise the possibility of sensory nerve pathology in patients with MND and suggest that the presence of unexplained sensory abnormalities should not exclude a diagnosis of MND.

079 GLIAL FIBRILLARY ACIDIC PROTEIN (GFAP) ASTROCYTOPATHY ASSOCIATED WITH CEREBRAL MICRO-INFARCTION AND POOR THERAPEUTIC RESPONSE

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Introduction Glial fibrillary acidic protein (GFAP) astrocytopathy is a lesser recognised immune-mediated meningo-encephalomyelitis, which is steroid responsive in the majority of cases. Neuroimaging is unique with a distinctive symmetric white matter perivascular linear and punctate enhancement pattern. We present a case with classical phenotype but delayed clinical response, and highlight the importance of early recognition and treatment.

Case A 59-year-old Caucasian female presented with a two month history of headache, gait disturbance, insomnia, agitation, disorientation and reduced oral intake. Examination revealed a high frequency upper limb tremor, hypertonicity and pathologically brisk reflexes with impaired cognitive function. MRI brain and spinal cord demonstrated high T2 signal and striking perivascular and punctate enhancement in supratentorial white matter, cervical and upper thoracic cord. CSF examination revealed lymphocytic pleocytosis and elevated protein. Brain biopsy demonstrated reduced GFAP expression, perivascular T-lymphocytic infiltrate, and recent white matter microinfarction. CSF and serum GFAP antibodies were positive.

Motor deterioration accompanied progression to a stuporous state. High dose corticosteroids were commenced, followed by intravenous immunoglobulin and mycophenolate. While there was marked improvement of perivascular contrast enhancement on imaging, the patient continued to demonstrate prominent tremor, gait disturbance and behavioural issues 9 months following symptom onset.

Conclusions The persistence of disability in this case is likely the result of axonal loss from the initial insult, reflected by the biopsy evidence of microinfarction. Awareness of the unique pattern on MRI and the clinical phenotype will aid in early recognition and prompt treatment of this condition, thus preventing the potential long term morbidity.

080 THE INCIDENCE, DIAGNOSIS AND OUTCOMES OF IDIOPATHIC INTRACRANIAL HYPERTENSION IN THE SOUTHERN TASMANIAN CATCHMENT

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Introduction This study aimed to identify the incidence of idiopathic intracranial hypertension (IIH) in Southern Tasmania, Australia. Secondary aims were to elucidate demographics, current approaches to investigation, treatment and outcomes. To our knowledge, similar regional studies have not been performed.

Methods The study was approved by the University of Tasmania Human Ethics Committee. Patients presented between June 2016-June 2018 to Royal Hobart Hospital, the single tertiary Neurology service in Tasmania. Cases were identified by screening lumbar punctures (LP) performed by Neurology services (inpatient, outpatient, Radiologically-assisted) and by surveying all regional Neurologists. Medical records were used to corroborate LP results and determine whether patients met Modified Dandy Criteria (MDC) (used to define IIH in current literature). Regional population statistics were obtained from the Australian Bureau of Statistics (ABS). Duplicate records were excluded. Exclusion criteria included age <18 at