post thrombolysis a non contrast CT brain was performed and was reported negative for haemorrhage. The patient reported altered vision in her left eye, worsening over the 24 hours since thombolysis. A further non contrast CT brain demonstrated an acute posterior haemorrhage within the left globe that had been present but not noted on previous imaging. Formal ophthalmology review and B scan ultrasonography confirmed subhyaloid haemorrhage with further vitreous haemorrhage. The patient proceeded to undergo vitrectomy under sedation with mild improvement in visual acuity at 3 months post discharge.

2815 PHENOTYPING VARIANTS OF TUMEFACTIVE DEMYELINATING LESIONS ACCORDING TO CLINICAL AND RADIOLOGICAL FEATURES – A CASE SERIES

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Objectives Apart from the rare demyelinating variants of Marburg's acute MS, Schilder's Disease, and Balo's concentric sclerosis, there are no detailed data to phenotype tumefactive demyelinating lesions (TDLs), aside from being greater than 2cm on MRI. We identified similar clinical and radiological features of 4 patients with TDLs that may represent a distinct phenotype. Our primary objective was to describe the details of these patients, with secondary objectives of a literature review and treatment recommendations.

Methods We performed a retrospective cases series review of 4 patients with very large TDLs (greater than 4cm). We reviewed the clinical features of each patient including EDSS scores at multiple time points. Results of investigations including blood tests, CSF analysis and radiological features, as well as treatments were described. We also summarized relevant literature via database searches including PubMed.

Results All patients presented with hemiplegia and apraxia. The mean age at onset was 37 years with an equal sex distribution. All patients were diagnosed with TDLs based on MRI and CSF analysis, precluding the need for brain biopsy. All responded to potent immunotherapy (including high dose corticosteroids, plasma exchange, rituximab and/or cyclophosphamide). The mean lag from diagnosis to treatment was 1 day. The median EDSS at presentation was 6 and recovery to a median EDSS of 2 occurred over 6 months.

Conclusions We propose that TDLs greater than 4cm are termed giant demyelinating lesions (GDLs). We suggest using this criterion, with clinical and laboratory data, to lead to rapid diagnosis and treatment.

2816 DIAGNOSTIC UTILITY OF LONG-TERM AMBULATORY VIDEO ELECTROENCEPHALOGRAPHY MONITORING

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Objectives Ambulatory video electroencephalography (A-VEEG) represents a low-cost, convenient and accessible alternative to inpatient VEEG monitoring, however few studies have examined their diagnostic yield. In this large-scale Australian study, we evaluated the efficacy of long-term A-VEEG recordings in capturing diagnostic events and addressing the referring question(s).

Methods Adult and paediatric A-VEEG reports from January 2020 to June 2021 were reviewed retrospectively. Diagnostic utility was explored by examining i) time of first diagnostic event, and ii) ability to address the referring question(s) – seizure *localisation*, *quantification*, *classification*, and/or *differentiation* (differentiating epileptic from non-epileptic events).

Results 600 reports were analysed, ranging 1–10 days duration (mean=5.7). At least one event was captured in 46% of recordings. 13% captured epileptic events and 36% captured events without electrographic changes. Unrecognised events were captured in 52 recordings, and were mostly (81%) epileptic events. 9% of events were not classified due to absence of clinical, video or electrographic data. 234 recordings (39%) captured a diagnostic event, of which 96% were first captured within the initial five days of recording. 85% of reports with at least one event (and 52% of all reports) captured diagnostic events and/or electrographic changes which unequivocally addressed the referrer's question(s). Specifically, this represented 75% of reports (27/36) regarding *classification* of seizures, and 46% of reports (235/515) regarding *differentiation* of events. 45% of studies captured interictal abnormalities; in their absence, almost all seizure-like events (96%) were non-epileptic in nature.

Conclusions A-VEEG recordings were of high quality and diagnostic value in capturing clinically relevant events.

2817 LATE ONSET SELENON-RELATED MYOPATHY PRESENTING WITH SEVERE RESPIRATORY FAILURE

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Multiminicore disease is the second commonest form of congenital core myopathy, caused by autosomal recessive gene mutations in SELENON, MYH2, TTN, CCDC78 or MYH7.[1] Though there is some clinical heterogeneity, the classical form is associated with predominant axial weakness and usually presents as a neonate or in the first year of life.

Case A 39-year-old man presented in severe hypercapnic respiratory failure. History was significant for scoliosis since childhood and pulmonary hypertension. Initial hospital admission required intubation and ventilation, he was treated for pneumonia and discharged home with non-invasive ventilation. Neurology review found a Trendelenberg gait, symmetrical proximal weakness, and positive Gower's sign; there were no sensory deficits. Serological testing demonstrated a normal creatine kinase level with negative myositis antibodies. Electrophysiology was normal, EMG disclosed myopathic units proximally. A muscle biopsy showed non-specific myopathic features. A subsequent neuromuscular genetic panel demonstrated two pathogenic variants in the Selenon gene (c.943G>A (p.Gly315Ser)) associated with multiminicore myopathy. Reassessment of the muscle biopsy, including with electron microscopy, did not find definite cores. The patient was