neuropathies is essential as recent treatment trials show a remission rate of up to 40%.

Aims Compare retrospective data on clinical, investigational and treatment factors in patients who have ceased IVIg with patients who have failed a cessation trial.

Methods 15 patients who successfully suspended IVIg infusions were compared with 15 in whom decreasing or stopping IVIg was unsuccessful.

Results 30 patients (12 with CIDP and 3 with MMN in both groups) were diagnosed 39.5 months from onset of symptoms in the successful group vs. 40.7 months in the unsuccessful group ($p=0.953$). There was a significant difference in the summed upper limb sensory amplitudes on electrophysiology prior to starting IVIg between the patients with CIDP (17.4 mV vs. 9.8mV $p=0.007$). There was no difference in the average doses between the groups. A successful cessation trial was attempted at a mean of 60.5 months post starting treatment, compared with 60 months in the unsuccessful patients.

Conclusion There is a need for objective biomarker to measure disease activity because other than one neurophysiology marker, other factors did not help predict a successful cessation trial of IVIg.

087 ENDOVASCULAR CLOT RETRIEVAL (ECR) IN THE ELDERLY. FOR BETTER OR WORSE IN THE REAL WORLD?

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Introduction

Across multicentre trials ECR is safe and effective in octogenarians. Despite RCT evidence elderly patients may be denied ECR due to perceived poor risk-benefit. We examine impact of age on ECR outcomes and outcomes in transcatheter aortic valve implantation (TAVI) cases (where stroke risk is high), in a real world setting.

Methods We analysed 311 consecutive ECR cases between 2016 and 2019 in 10 year age bands for ECR outcomes including 90 day mRS and mortality. Impact of premorbid function (mRS), NIHSS, recorded co-morbidities, and aetiology was assessed. TAVI case outcomes were examined.

Results Thirty one percent of ECR outcome cases were over 79 years of age; 90 day mortality was 34%; 25% had a 90 day mRS 0–2. Early NIHSS improvement was 5. Ninety-day mortality and mRS 0–2 for 10–19 ($n=3$), 20–29 ($n=2$), 30–39 ($n=4$), 40–49 ($n=23$), 50–59 ($n=27$), 60–69 ($n=69$), 70–79 ($n=84$), 80–89 ($n=62$) and 90–99 ($n=11$) years were 0 and 100%, 0 and 100%, 33 and 67%, 4 and 78%, 15 and 52%, 13 and 49%, 17 and 33%, 24 and 2% and 55 and 18%, respectively. There was 9% lost to follow-up.

Six TAVI cases had a NIHSS of 8–20 and pre-morbid mRS<3, four with mRS 0. Mean 24 hour NIHSS improvement was 8.

Conclusion Without age exclusions older patients had worse unadjusted outcomes. However, patients over 79 years had clinically important early improvement in NIHSS score and

ninety day outcomes were comparable to favourable RCT data and TAVI patients also had early improvement.

088 CLADRIBINE TABLETS WERE ASSOCIATED WITH RAPID ONSET OF IMPROVEMENTS IN MRI OUTCOMES IN THE ORACLE-MS TRIAL

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Introduction In ORACLE-MS (616 subjects with a first demyelinating event at high risk of converting to multiple sclerosis), cladribine tablets (CT) 10 mg (3.5 mg/kg or 5.25 mg/kg cumulative dose over 2 years) significantly delayed the time-to-conversion to clinically definite multiple sclerosis (CDMS), and reduced new/persisting T1 gadolinium-enhancing (T1 Gd+), new/enlarged or active T2 and combined unique active (CUA) lesion number. Here, the timing of CT effect is evaluated.

Methods MRI scans were performed at screening and every 12 weeks, for non-converting CDMS subjects. MRI-based endpoints were analyzed using analysis of covariance (ANCOVA) and negative binomial models. The temporal effects of the first yearly treatment course of CT and placebo on T1 Gd+, active T2, and CUA lesions were evaluated.

Results 96 weeks: the reduction in mean T1 Gd+, active T2, or CUA lesion number per patient per scan was nominally significantly greater for CT versus placebo ($p<0.0001$). Early change in Gd+ lesion volume (at Week 13) from baseline was CT, -155.73 mm³; placebo, -14.76 mm³. Comparatively larger reductions in mean active T2 and CUA lesion numbers with CT at Week 13 versus placebo were observed (active T2: CT, -1.25; placebo, -1.43; CUA: CT, -1.56; placebo, -2.41). The mean number of T1 Gd+ lesions at 13 weeks following CT was 0.37 versus 1.0 with placebo.

Conclusions MRI data from ORACLE-MS subjects suggest the first yearly treatment course of CT has a rapid onset of action, with beneficial treatment effects on active lesion number and volume evident by Week 13.

089 BREAKING THE CYCLE OF CHRONIC DAILY HEADACHE WITH A LOW-DOSE SUBCUTANEOUS LIGNOCAINE AND KETAMINE INFUSION

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Introduction Management of chronic migraine includes correcting analgesic rebound headache and implementing suitable medication for prevention and acute episodes. However, in many cases this management paradigm oversimplifies the complexity of chronic migraine, particularly the entrenched central pathways that perpetuate chronic migraine. Intravenous

lignocaine can curtail chronic migraine and analgesic rebound headache (1). Further, ketamine provides short-term analgesia and enables reduction in central sensitisation of pain pathways, particularly in the setting of codeine/opioid overuse (2). This paper describes use of subcutaneous lignocaine and ketamine infusion in chronic migraine.

Methods A prospective observational cohort study was undertaken in patients with chronic migraine. Patients received a prolonged subcutaneous lignocaine and ketamine infusion (mean duration 11 days) and underwent evaluation at four-time points over six months. The effects on the excitability of motor axons in the median nerve were documented using standard procedures.

Results Fourteen patients were recruited. The infusion was well tolerated; no major side effects were seen. There were no significant long-term changes in the excitability of motor axons. At six months, 13/14 patients had sustained benefit. Three of 4 patients remained free of analgesic rebound headache. One patient remained headache-free. Conversion to episodic migraine occurred in 6/14. Improvement in chronic migraine was reported by 6/14. Three of six were able to return to work, with 1 returning to studies. Benefit was greater in those with depression and history of opioid/codeine use.

Conclusion Subcutaneous lignocaine and ketamine can help break entrenchment in chronic migraine as part of a structured management plan.

090 THE EFFECT OF CLADRIBINE TABLETS ON DELAYING THE TIME TO CONVERSION TO CLINICALLY DEFINITE MULTIPLE SCLEROSIS (MS) OR MCDONALD MS IS CONSISTENT ACROSS SUBGROUPS IN THE ORACLE-MS STUDY

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Introduction In the Phase 3 ORACLE-MS trial in 616 subjects with a first demyelinating event at high risk of converting to multiple sclerosis (MS), treatment with cladribine tablets 10 mg (3.5 mg/kg or 5.25 mg/kg cumulative dose over 2 years [CT3.5 and CT5.25, respectively]) significantly delayed time to conversion to clinically definite multiple sclerosis (CDMS) according to Poser criteria (67% or 62% risk reduction [RR], respectively) and time to conversion to 2005 McDonald MS (50% or 57% RR, respectively), versus placebo. The objective was to analyze the effect of cladribine tablets vs placebo on conversion to CDMS and McDonald MS across ORACLE-MS patient subgroups based on baseline characteristics.

Methods In this post-hoc analysis, time-to-conversion to CDMS or McDonald MS over the double-blind period was analyzed for patients treated with CT5.25 (N=204), CT3.5 (N=206) or placebo (N=206) across different subgroups. Subgroups were defined by baseline characteristics which have been investigated as potential predictors of CDMS conversion (age [<30 or ≥ 30 years], gender, first classification demyelinating event [monofocal or multifocal], presence of T1 Gd+ lesions and number of T2 lesions [<9 or ≥ 9]).

Results Treatment with CT3.5 or CT5.25 was consistently efficacious across the subgroups examined on conversion to

CDMS versus placebo for most comparisons (RR range: CT3.5, 39%–72%; CT5.25, 36%–79%). Similarly, treatment effect of both doses on conversion to 2005 McDonald MS was consistent across subgroups (CT3.5, 40%–59%; CT5.25, 42%–79%).

Conclusions The effect of cladribine tablets on delaying the time-to-conversion to CDMS, or to McDonald MS, is consistent across subgroups.

091 CEREBELLAR OEDEMA IN FULMINANT ADULT LEIGH SYNDROME

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Introduction We report a case of adult Leigh syndrome resulting in rapidly fatal cerebellar oedema.

Case A 19-year-old female presented with a five-week history of hyperventilation, generalised weakness, dysarthria and bilateral ptosis. Brain Magnetic resonance imaging (MRI) findings and the presence of a mitochondrial mutation (NC_012920.1 (MT-ATP6):m.9176T>C) in blood and urine with approximately 97% heteroplasmy, confirmed a diagnosis of Leigh syndrome.

Two-days after a normal lumbar puncture, opening pressure 8cm water, her conscious level rapidly declined. CT revealed marked cerebellar oedema with brainstem compression. Despite immediate decompression, she did not recover consciousness and died six-weeks after symptom onset.

Conclusion Adult Leigh syndrome is a progressive untreatable inherited mitochondrial disorder typically of infants and children. Adult cases are rare and described mostly in single case reports. There is marked phenotypic and genotypic variability. Over 83% of Leigh's syndrome is identified by the age of 2 years, however, there have been cases reported in patients up to 74 years old. There are over 60 mutations described in Leigh syndrome, which are identified in only half of reported cases. Classic MRI changes include bilateral symmetric T2 hyper-intensities in the basal ganglia and brainstem. To our knowledge, this is the first reported case resulting in fulminant cerebellar oedema. A challenge of diagnosis remains the marked heterogeneity in presenting symptoms including cognitive decline, behavioural change and ophthalmoparesis. Typically, this syndrome has been confirmed by histopathology at autopsy. Advances in genetics and imaging have allowed earlier accurate diagnosis, potentially paving the way for improved therapeutics.

092 MOTOR NEURON DISEASE WITH MALIGNANCY: CLINICAL AND PATHOPHYSIOLOGICAL INSIGHTS

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Introduction While some regard an association between motor neuron disease (MND) and malignancy as co-incidental, others