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