2589 SUDDEN UNILATERAL AUDIOVESTIBULAR LOSS DUE TO ACUTE LABYRINTHINE HAEMORRHAGE CAN BE MISSED ON MRI BRAIN SEQUENCES

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A 45-year-old woman presented with sudden onset vertigo, left hypoacusis, and tinnitus, consistent with acute unilateral audiovestibular loss. Bedside examination revealed a left sided sensorineural hearing loss and leftward falling gait. Left peripheral vestibular hypofunction was confirmed acutely on video head impulse testing characterised by reduced vestibular-ocular reflex gains with overt catch-up saccades in the left horizontal and posterior semicircular canals. Acoustic reflexes were absent on the left side. Pure tone audiometry confirmed a profound left sensorineural hearing loss, with no measurable hearing threshold in the left ear. An MRI brain with diffusion weighted imaging performed after 24 hours was unremarkable. Repeat MRI of the internal acoustic canal (IAC) on day 7 demonstrated increased T1 and FLAIR signal throughout the left cochlear and semicircular canals, without contrast enhancement. A diagnosis of labyrinthine haemorrhage was made. Extensive testing for coagulopathies and vasculitides was negative. On long-term follow-up the patient remained profoundly deaf, however balance and vestibular symptoms improved with early vestibular physical rehabilitation. This case highlights the importance of an inner ear MRI in presentations of acute audiovestibular loss, as well as the utility of ancillary audiovestibular testing. Labyrinthine haemorrhage may be missed on stroke-specific sequences

2637 ELECTROENCEPHALOGRAPHIC CHANGES ASSOCIATED WITH PROPOFOL RELATED INFUSION SYNDROME

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Propofol Related Infusion Syndrome (PRIS) is a life-threatening complication of propofol use. EEG changes associated with PRIS have not been described.

A 21-year-old man with recent traumatic brain injury requiring craniectomy re-presented with recurrent tonic-clonic seizures requiring intubation. After convulsive seizures were terminated, with midazolam, levetiracetam, phenytoin and propofol; continuous EEG monitoring (cEEG) demonstrated focal non-convulsive status epilepticus. Propofol was increased with clonazepam and valproate added. On day 4 the patient developed acute cardiac dysfunction with metabolic acidosis, hyperkalaemia, rhabdomyolysis and hyperlipaemia (all consistent with PRIS). Propofol was ceased. The patient received haemofiltration and aggressive supportive care and survived.

Initial cEEG demonstrated continuous 1-Hz left hemispheric lateralised periodic discharges (LPDs) with associated fast activity (LPDs+F) and recurrent left hemispheric electrographic seizures without clinical accompaniment. The right hemisphere demonstrated changes consistent with an anaesthetic effect. Electrographic seizures improved on Day 3 though LPDs+F persisted. On Day 4, prior to any overt clinical features of PRIS, there was progressive dysfunction initially of the right hemisphere, followed by prolonged periods of generalised suppression followed by burst suppression, with ongoing left LPDs+F. Following propofol cessation these changes persisted for 48 hours and by day 10 the right hemispheric changes had resolved with persistent focal slowing and frequent epileptiform discharges seen on the left.

The EEG changes seen, in the absence of hypotension or hypoxia, likely reflect PRIS-related cerebral mitochrondrial dysfunction. As cEEG is being increasingly utilised recognition of the spectrum of EEG changes of PRIS may allow early identification and immediate cessation of propofol.

2755 LIMBIC-PREDOMINANT AGE-RELATED TDP-43 ENCEPHALOPATHY – A MIMIC FOR AMYOTROPHIC LATERAL SCLEROSIS

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Background Limbic-predominant age-related TDP-43 encephalopathy (LATE) is a TDP-43 proteinopathy, typically diagnosed in elderly patients. At present, a definitive diagnosis of LATE is made from neuropathological changes at autopsy. Neuropathological changes are characterised by the anatomical distribution of TDP-43 in the amygdala, hippocampus and middle frontal gyrus. Separately, LATE is most commonly associated with cognitive impairment, typically dementia of the Alzheimer's type.

In terms of causation, TDP-43 is a protein encoded by the TARDBP gene. Of relevance to motor function, mutations involving the gene encoding TARDBP are causative for amyotrophic lateral sclerosis (ALS) in <5% of familial cases of ALS. Separately, TDP-43 protein is the hallmark ALS pathology, present at autopsy in >90% of patients diagnosed with ALS.

Cases The present case series describes two patients managed for ALS, who were subsequently diagnosed with LATE following post-mortem examination.

Case 1 (76M) initially presented with upper limb wasting and predominantly lower motor neurone pattern weakness, progressing to generalised disease with weakness over a 12 year period. Case 2 (79F) presented with disease-onset in the distal left upper limb, progressing to a left sided spastic hemiparesis and spastic dysarthria. She was diagnosed with PLS (likely Mills' variant) and died 7 years after symptom onset.

Discussion Unifying phenotypic features of an ALS-presentation of LATE include limb-onset disease, slower disease progression

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