patients. Multiple domains of autonomic nervous system may be affected. A more comprehensive, accurate yet accessible test battery is required to better evaluate autonomic impairment in iRBD patients.

REFERENCE

1. Ferini-Strambi L, *et al*. Autonomic symptoms in idiopathic REM behavior disorder: a multicentre case-control study. *J Neurol*, 2014;**261**:1112–8

2437 INTRALABYRINTHINE HAEMORRHAGE: AN IMPORTANT CAUSE OF ACUTE VERTIGO PRESENTING SIMULTANEOUSLY WITH SUDDEN HEARING LOSS

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10.1136/bmjno-2022-ANZAN.164

Introduction Intralabyrinthine haemorrhage is a rare cause of sudden sensorineural hearing loss (SNHL) associated with an acute vestibular syndrome (AVS).
Case Report A 45-year-old female with a history of stable migraine with aura, presented with an AVS with simultaneous sudden SNHL and tinnitus without preceding viral symptoms. Examination revealed a positive left bedside head impulse test and left SNHL. Pure tone audiometry (PTA) revealed profound left SNHL. Video head impulse testing revealed significantly reduced vestibular-ocular reflex gain with catch-up saccades of the left posterior semicircular canal. Comprehensive vestibular testing revealed widespread left vestibular failure. Blood evaluation revealed mild lymphopenia but normal coagulation studies, inflammatory markers, vasculitic, autoimmune and pro-thrombotic testing. Computed tomography angiogram of head and neck and magnetic resonance imaging (MRI) brain with diffusion-weighted imaging (DWI) performed within 24-hours of symptom onset were normal. MRI of the internal auditory meatus (IAM) on day 7 revealed increased T1 and fluid-attenuated inversion recovery (FLAIR) signal in the left cochlea and semicircular canals, with no post-contrast enhancement. She was initially treated with oral prednisolone and valciclovir. Intratympanic steroids were given at 2, 3 and 4 weeks. Vestibular symptoms had improved by day 7 but at her 3-months follow-up she remained profoundly deaf in that ear and will be evaluated for cochlear implant candidacy.
Conclusion Identifying patients having an intralabyrinthine haemorrhage in the context of an AVS is important as it carries a poor prognosis. All patients with acute vertigo with sudden SNHL should be considered for urgent MRI of the labyrinth.

2438 THE VALUE OF CONCURRENT ELECTROCARDIOGRAPHY WHEN PERFORMING AN ELECTROENCEPHALOGRAPH

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10.1136/bmjno-2022-ANZAN.165

Introduction The use of concurrent, single lead electrocardiograph (ECG) recording, when performing a routine electroencephalograph (EEG), has been standard practice for many years. The diagnostic utility of a concurrent ECG is relatively unknown. Previous studies have reported its usefulness in syncope and the detection of newly identified cardiac dysrhythmia but have relied on specialist cardiologist interpretation of the ECG trace. This study expands the understanding of concurrent ECG and provides demographic information regarding the incidence, nature and diagnostic utility of ECG interpretation, during routine EEGs, as evaluated by neurologists.
Methods A single center, retrospective study of routine concurrent EEG and ECG recordings was performed. All routine EEGs, performed over one year, were analysed. Demographic data, underlying comorbidities, reasons for referral and ECG changes were assessed.
Results ECG abnormalities were identified in 147 (13.5%) of concurrent ECG/EEG routine recordings. The presence of ECG abnormalities was significantly associated with the reason for referral, namely being assessed for the evaluation of seizure activity and with increasing patient age. Thirty-eight patients (3.5%) had newly identified ECG abnormalities, of which atrial fibrillation (AF) (12 patients) and sinus bradycardia (9 patients) were the most common. Five patients (0.5%) had a change in their management consequent to the identified ECG changes.
Conclusions These findings support the value of neurologists' interpretation and need for ongoing concurrent ECGs, during routine EEG recording. , The study raises concern about the requesting clinician's response to the dentification of newly diagnosed cardiac dysrhythmias.

2439 VARICELLA ZOSTER VIRUS RHOMBENCEPHALOMYELITIS: A CASE REPORT

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10.1136/bmjno-2022-ANZAN.166

Objectives Varicella Zoster Virus (VZV) can cause a spectrum of neurological presentations.
Methods Case report.
Results We describe the case of a 66 year old immunocompetent male who presented with a 2 day history diplopia and left sided ataxia on the background of a recent left facial VZV infection, presenting with vesicular rash and dysesthesia in the left trigeminal V2 and V3 distributions. Examination revealed a left internuclear ophthalmoplegia with skew deviation, left hemifacial numbness and tandem gait instability. CSF protein was elevated. Facial swab was VZV PCR positive. CT Angiogram revealed no intracranial stenosis suggestive of a vasculopathy. 1.5 T MRI Brain and Spine revealed high T2 signal in the dorso-lateral pons, medulla and adjacent upper cervical cord in the trigeminal nuclei. Smaller foci were also present in the left facial colliculus and in the right inferior cerebellar peduncle reflective of a rhombencephalomyelitis. Antiviral therapy was administered for 2 weeks. Follow up 3 T MRI at 5 weeks post discharge additionally revealed high T2 signal along the course of the left trigeminal nerve in the brainstem as well as high T2 signal and enhancement in the cisternal segment of the left trigeminal nerve. There was clinical improvement in all neurological symptoms over a 3 week period.
Conclusion This case demonstrates that VZV can result in a broad inflammatory process across the neuraxis and cause a

rhombencephalomyelitis in immunocompetent adults. MR imaging can confirm brainstem involvement.

2440 SACUBITIRIL/VALSARTAN INDUCED SYMPTOMATIC MYOCLONUS: A CASE REPORT

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10.1136/bmjno-2022-ANZAN.167

Objective Drug induced myoclonus has been identified in number of medication classes. We describe a case of drug induced myoclonus from a newly marketed novel combination medication angiotensin receptor-neprilysin inhibitor Sacubitril/Valsartan (Entresto®), indicated for heart failure.
Methods A case report.
Results A 75 year old independent male presented with 11 hours of head and upper limb sudden onset myoclonic activity on the background of known history of heart failure having commenced Sacubitril/Valsartan one month prior and recently dose escalated. There was no inter-current illnesses and no other drug changes. Examination revealed an involuntary, irregular intermittent myoclonic activity in bilateral arms and head. Vital signs were normal. There was no acute abnormality on CT Brain or baseline blood tests. EEG showed marginal slowing in the right central region and no electrical correlate for the myoclonus. Following cessation of the Sacubitril/Valsartan the patient's myclonus abated within 36 hours and the patient was discharged home with no further complication.
Conclusion Myoclonus and other involuntary movements have been reported with the use of Sacubitril/Valsartan with onset ranging from hours to days in patients with similar medical profiles.[1] Similar side effects have been seen in rodent models but not in primates, and no clear pharmacological mechanism exists at this stage. We present this case to inform other clinicians of the potential serious neurological side effect of this increasingly used medication.

REFERENCE

1. Bejan-Angoulvant T, Genet T, Vrignaud L, Angoulvant D, Fauchier L. Three case reports of involuntary muscular movements as adverse reactions to sacubitril/valsartan. *Br J Clin Pharmacol*. 2018;**84**(5):1072–1074.

2441 MRI TRACTOGRAPHY IN MILLS SYNDROME: A CASE REPORT

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10.1136/bmjno-2022-ANZAN.168

Objectives Mills syndrome is a rare isolated and asymmetrical upper motor neuron (UMN) syndrome.[1]
Methods A case report.
Results A 51-year-old presenting with a 5-year history of progressive right sided pyramidal weakness and spasticity. Although no cognitive symptoms were reported, the patient exhibited executive dysfunction on formal testing. Examination revealed right sided facial asymmetry as well mild pyramidal weakness in the upper and lower limbs but associated with prominent UMN features including spasticity, brisk reflexes and a positive right sided Hoffman's sign. Sensory examination was normal and there were no definite extrapyramidal signs or apraxia. Nerve conduction studies and electromyography demonstrated absence of denervation and large fibre neuropathy in clinically affected right sided muscles. Transcranial magnetic stimulation demonstrated an inexcitable left sided motor cortex. MRI Brain showed global atrophy with asymmetry in the left frontotemporal region. Cerebral PET revealed diffuse reduction in metabolism in the frontal cortex, more marked on the left and to a lesser degree in the parietal and temporal lobes bilaterally. CSF studies were bland. T-tau, P-tau and Abeta1–42 were not elevated. Genetic testing was negative for c9Orf72. 3T-MRI Tractography of the brain demonstrated asymmetry in cortical spinal tract fibres with significant reduction in left white matter fibre density. A clinical diagnosis of Mills syndrome was made. The patient received treatment with muscle relaxants and physical rehabilitation.
Conclusion Mills syndrome is a diagnosis of exclusion requiring multimodal investigations. Novel structural imaging such as the use of MRI Tractography can aid in the diagnosis of Mills syndrome.

REFERENCE

1. William Huynh, Sicong Tu, Colin J. Mahoney, Raymond Schwartz, Matthew C. Kiernan. *Neurology* Apr 2021;**96**(14):677–678.

2442 CENTRAL PONTINE MYELINOLYSIS WITHOUT HYPONATREMIA IN LATE-STAGE PREGNANCY

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10.1136/bmjno-2022-ANZAN.169

Introduction Central Pontine Myelinolysis (CPM) is a demyelinating disorder affecting the central pons linked to osmotic stress and hyponatremia. Cases in pregnancy have been associated with hyperemesis gravidarum, but also with other fluctuations in electrolyte and metabolic balance.[1]
Case A 22-year-old lady presented at 34 weeks (G2P0) with severe headache. Pregnancy to that date had been unremarkable and a detailed neurological examination was unremarkable. A MRI was performed revealing a central pontine lesion demonstrated by hyper-intensity on T2 and FLAIR imaging with hypo-intensity on T1 sequences, associated with significant diffusion restriction consistent with osmotic demyelination. Serum sodium level was normal at 136 mmol/L and remained so throughout her pregnancy with no other electrolyte imbalance noted.

A differential diagnosis of inflammatory, infective and ischaemic insult amongst others was considered. A spinal MRI was unremarkable and a lumbar puncture revealed a normal cell count, protein and glucose, with no oligoclonal bands. Blood pressure was controlled throughout pregnancy, and serological testing of serum FLT1/PIGF ratio did not reveal any early pre-eclampsia. The patient subsequently had a successful vaginal delivery at 36+1/40 weeks after induction of labour.

A repeat MRI performed 3 months after delivery demonstrated almost complete resolution of the pontine signal intensity with no further diffusion restriction.
Conclusion This case highlights a unique presentation of a central pontine myelinolysis in late-stage pregnancy without