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

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## 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

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## 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

any identifiable secondary cause. The resolution of imaging changes post-delivery suggests that the pregnancy itself may have been a triggering or associated factor in the development of the disorder.

## REFERENCE

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## 2443 THE SPECTRUM OF SEROPOSITIVE IMMUNE-MEDIATED ENCEPHALITIS IN WESTERN AUSTRALIA

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10.1136/bmjno-2022-ANZAN.170

**Objectives** To analyse the clinical features of patients with seropositive immune-mediated encephalitis in Western Australia.

**Methods** Patients with a clinical syndrome consistent with immune-mediated encephalitis and positive anti-neuronal antibody in Western Australia were retrospectively identified between 2005 and 2021. The clinical features including investigations, treatment and outcome were analysed.

**Results** 58 patients met the inclusion criteria (35 (60%) patients female, median age at symptom onset 58 years (range 16–84 years). The most common antibody identified was had anti-NMDA-R (57%) followed by VGKC-complex (17%), anti-Hu (ANNA-1) (9%) and anti-CV2/CRMP5 (5%), with $GABA_B$, PCA-Tr, GAD-65, Ri (ANNA-2), Ma-2, Amphyphysin and GFAP seen in the remaining 12% of patients. All patients presented with one of encephalopathy (65.5%), neuropsychiatric symptoms (62.1%) or seizures (53.4%).29% of patients had prodromal flu-like symptoms. 3 patients developed anti-NMDA-R encephalitis post-partum Only 50% of patients had an associated malignancy, most commonly ovarian teratoma ) in patients with anti NMDA-R Ab and non-small cell lung cancer in patients with other . Of the 43 (74%) patients with 33 (57%) patients had MRI abnormalities. 86% of patients received , with initial treatment most commonly being high dose steroids (82%) and/or IVIG (68%). Median length of stay was 45 days. Although 70% had a favourable MRS (0–2) at last follow up, 11 patients (19%) died.

**Conclusion** The findings demonstrate the spectrum of immune-mediated encephalitis in Western Australia with anti-NMDA-R Ab encephalitis most frequently seen.

## 2445 A RARE CASE OF ERDHEIM-CHESTER DISEASE ASSOCIATED WITH ANTI-GABA-B ANTIBODIES

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10.1136/bmjno-2022-ANZAN.171

**Introduction** Encephalitis associated with anti-GABA-B antibodies is a rare phenomenon which has been typically shown to manifest as a paraneoplastic process, usually associated with small-cell lung cancer. The present case is an unusual presentation of cognitive decline in the context of GABA-B antibody positivity in association with Erdheim-Chester disease (ECD).

**Case** A 71 year-old previously high-functioning female presented to the Emergency Department with an abnormal outpatient MRI brain. On further history, there had been a subtle decline in memory and disinhibited behaviour in a premorbidly highly intelligent woman. After extensive investigation, she was found to have positive serum GABA-B antibodies. The search for underlying malignancy was inconclusive. It was not until the orthopaedics team requested a plain X-ray of the femurs that the classic radiological features of Erdheim-Chester disease were revealed, which was confirmed on a bone biopsy. She was diagnosed with Erdheim-Chester disease associated with anti-GABA-B antibodies. Her BRAF v600E mutation was negative, and hence she was commenced on treatment with cobimetinib. Therapy initially produced minor improvement in simple attention and processing speed on neuropsychological assessment, but there continued to be a marked decline in orientation, learning and memory abilities. Unfortunately, the patient continued to suffer from marked cognitive and behavioural disturbance six months after receiving treatment.

**Conclusion** This is an unusual case of cognitive decline secondary to a paraneoplastic autoimmune encephalitis associated with ECD. The case serves as a reminder that ECD should be considered as a differential for patients who present with neurological symptoms.

## 2446 RECURRENT GUILLAIN-BARRE SYNDROME FOLLOWING COVID-19 VACCINATION AND SUBSEQUENT INFECTION

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10.1136/bmjno-2022-ANZAN.172

We report the case of a 41-year-old previously well male who developed recurrent Guillain-Barre syndrome (GBS) following sequential COVID-19 vaccination and again after subsequent acute COVID-19 infection. While recurrent GBS is reported, monophasic courses predominate the literature with temporal associations to new viral illnesses or vaccinations. Notably with the world-wide viral and vaccine crisis, atypical forms of GBS have been reported particularly in association to COVID-19 vaccination. In this case, our patient had an onset of distal parasthaesias at three weeks following COVID-19 vaccination progressing proximally over the next two weeks. These symptoms eased and he proceeded with his second dose at the planned time interval. Unfortunately, one week following this vaccination he developed a recurrence of symptoms so severe they prompted presentation to an emergency department. At this time, he was diagnosed with GBS due to the clinical picture of distally progressive parasthaesias and hyporeflexia with cerebrospinal fluid albuminocytologic dissociation. He subsequently rapidly responded to a standard course of intravenous immunoglobulin. Following this initial treatment course, he suffered a recrudescence of symptoms following acute COVID-19 infection. These symptoms remained mild and have abated with now only residual intermittent tingling parasthaesias which no longer interferes with his daily function. There is a lack of evidence to support a causal relationship between COVID-19 vaccination and recurrent GBS however, this case raises