PLR and NLR for a negative HIT in stroke was 5.85 (95% CI 3.07 – 10.6) and 0.17 (95% CI 0.08 – 0.30) respectively. PLR and NLR for peripheral HINT pattern for PV was 17.3 (95% CI 8.38 – 32.1) and 0.15 (95% CI 0.07 – 0.26) respectively. PLR and NLR for central HINT pattern for stroke was 5.61 (95% CI 4.19 – 7.7) and 0.06 (95% CI 0.03 – 0.12) respectively.

Conclusion The HIT and HINT exams appear moderately good discriminators of central and peripheral vertigo. However, these results may not apply in the ED setting as most papers evaluated these tests beyond 24 hours.

2386 DYSTONIC STRIDOR AND LARYNGEAL MYOCLONUS IN A MEDULLOBLASTOMA SURVIVOR PRECIPITATED BY REDUCTION IN INTRACRANIAL PRESSURE

[1]Cathy K Cui*, [2]Nicholas Jufas, [3]Dajina Dragicevich, [4]Roger Harris, [1]Karl Ng*. *[1]Department of Neurology, Royal North Shore Hospital, Sydney, NSW, Australia*; *[2]Department of Ear, Nose and Throat Surgery, Royal North Shore Hospital, Sydney, NSW, Australia*; *[3]Department of Speech Pathology, Royal North Shore Hospital, Sydney, NSW, Australia*; *[4]Intensive Care Medicine, Royal North Shore Hospital, Sydney, NSW, Australia*

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Background Whilst stridor related to bilateral vocal cord paralysis (BVCP) is a recognised complication of increased intracranial pressure, it typically resolves with ventricular-peritoneal (VP) shunting. We present a case where tracheostomy was needed after successful VP shunting.

Case A 33-year-old-man presented with headache, slurred speech and immobility. His background included resected medulloblastoma with head-neck radiotherapy (aged 8), long-term VP shunt, and post-treatment ataxia which had progressed over 2 years with development of intermittent nocturnal stridor. He underwent emergent shunt revision for a distally blocked shunt, with resolution of hydrocephalus. Two days afterwards, he developed hiccups and worsened stridor which progressed to respiratory obstruction over 24 hours. Laryngoscopy showed tightly adducted midline vocal folds with coarse pharyngeal, palatal, and tongue myorhythmia (3 Hz). There was no response to benzodiazepines and no epileptiform activity on EEG. He had loss of gag reflex on neurological examination with normal eye movements. A cerebral and neck MRI showed resolution of hydrocephalus and post-treatment changes related to previous medulloblastoma. There has unfortunately been no improvement in vocal fold movement over 3 weeks.

Discussion Subacute stridor may be due to pathological upper motor neurone activation of the branchial motor component of the vagal nerves with this person's background brain injury.[1] Tight acute midline cord positioning and myoclonus may be unusually caused by bilateral recurrent laryngeal nerve lesions secondary to changes in intracranial pressure,[2 3] but is not the favoured sole mechanism here. Botulinum toxin therapy could provide benefit but may compromise a future safe swallow.

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2388 DIAGNOSTIC CHALLENGES ASSOCIATED WITH GRANULOMATOUS DISEASES: AN ILLUSTRATIVE CASE REPORT

[1]Anivartha Herur*, [1,2]Michael H Barnett, [1]Andie S Lee, [1,3]Benjamin Jonker, [1]Lauren Troy, [3]Yael Barnett, [1,2]Heidi N Beadnall. *[1]Royal Prince Alfred Hospital, Sydney, NSW, Australia*; *[2]The Brain and Mind Centre, The University of Sydney, Camperdown, NSW, Australia*; *[3]St Vincent's Hospital, Sydney, NSW, Australia*

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Case Report A 29-year-old female presented with a 4-month history of right-sided hemifacial sensory disturbance. Examination revealed reduced pain and temperature sensation in the right V1, V2 and V3 regions and a diminished right corneal reflex. Past history included intermittent herpes labialis and travel to India twice in the prior 5 years.

Enhanced brain and skull-base magnetic resonance imaging (MRI) revealed enhancement at the cisternal segment of the right trigeminal nerve and right infraorbital nerve T2-hyperintensity. Diffuse extra-axial enhancement was noted in right Meckel's cave, extending into the posterolateral cavernous sinus. Positron emission tomography (PET) imaging demonstrated markedly increased metabolism in the right Meckel's cave and metabolism in the hilar and mediastinal lymph nodes consistent with sarcoidosis. Lymph node biopsies were inconclusive but revealed a possible granuloma. Right trigeminal nerve biopsy was subsequently performed. Histopathology revealed non-caseating granulomas with few acid-fast bacilli present. Primary neuritic leprosy was diagnosed and a 12-month treatment course of rifampicin, dapsone and clofazimine was commenced.

Repeat MRI 3-months post-treatment demonstrated reduced enhancement in the right cavernous sinus. Clinically her symptoms improved with antibacterial and steroid therapy. Repeat PET imaging revealed resolution of the Meckel's cave changes but increased metabolism in thoracic and abdominal lymph nodes, raising the possibility of an alternative diagnosis (i.e. sarcoidosis) or dual pathology. Pregnancy halted further diagnostic work up.

This case highlights the complexities associated with diagnosing granulomatous disease even with access to multimodal imaging and biopsy sites, and the need to revisit the diagnosis if there are atypical features.

2389 THE YIELD OF EEG AND OTHER NEUROLOGICAL INVESTIGATIONS IN PATIENTS WITH NEW ONSET OF PSYCHIATRIC SYMPTOMS

[1,2]Fu-Chu FC Chiang*, [1]Robert RMT Thomas, [1]Meng MT Tan, [1]Gaurav GS Singh*. *[1]Neurology, Gold Coast University Hospital, Gold Coast, QLD, Australia*; *[2]Queensland Health, Robertson, QLD, Australia*

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Objectives To establish the diagnostic yield of electroencephalogram (EEG) and other investigations for evaluation of limbic encephalitis in patients with new onset psychiatric symptoms.

Methods A retrospective audit was conducted of all EEGs performed between July 2020 to August 2021 for workup of new-onset psychiatric symptoms at a tertiary neurology referral centre. Data was obtained from electronic medical records including patient history, neurological examination findings, results of investigations including EEG, brain magnetic

resonance imaging (MRI), cerebrospinal fluid (CSF), serum autoantibodies and final clinical diagnosis.

Results 82 patient requests were identified, 9 outpatients and 73 inpatients. 8 patients (10%) had an abnormal EEG: 1 showed epileptiform abnormalities (patient already known to have epilepsy) and 7 showed non-specific slowing (generalised in 3 and focal in 4)n. 5 of 38 patients (13%) with brain MRI had structural abnormalities demonstrated. 2 out of 46 patients (4%) tested for anti-neuronal antibodies had low-titre VGKC autoantibodies, both determined to be false positive results. No patients had abnormal neurological signs apart from confusion or drowsiness. No patients had a final diagnosis of limbic encephalitis.

Conclusions While evaluation for epilepsy or limbic encephalitis is important in patients with new onset psychiatric symptoms this audit demonstrates that yield of neurological investigations for evaluation of limbic encephalitis in general psychiatric presentations is low. Careful patient selection using clinical criteria such as described by Graus, *et al*. may increase the yield and reduce the burden of investigations on the public health system.

2393 A RARE CASE OF RECURRENT MENINGOENCEPHALITIS WITH HEARING LOSS AND DORSAL COLUMN MYELITIS

Shuo Xi*, Jessica Qiu, Jessica Stabler, Sai Nagaratnam, Jon Iredell, Neil Mahant. *Department of Neurology, Westmead Hospital, Westmead, NSW, Australia*

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Introduction Brucellosis is the most common zoonosis worldwide with a wide spectrum of clinical presentations. We describe a case of neurobrucellosis manifesting as meningoencephalitis, hearing loss, and dorsal column myelitis.

Case Presentation A 36-year-old Egyptian male developed insidious onset of headache, malaise, and fever, progressed to confusion and episodic aggression with gait ataxia. One year prior, a similar episode spontaneously resolved but was followed by subacute sensorineural hearing loss.

Examination demonstrated bidirectional gaze-evoked nystagmus, severe bilateral sensorineural hearing loss, with upper limb pseudoathetosis and proprioceptive loss.

Neuroimaging revealed bilateral leptomeningeal enhancement involving the central sulcus on T2/FLAIR, and cervical predominant longitudinally extensive T2 hyperintensity of the dorsal columns without contrast enhancement.

CSF studies showed hypoglycorrachia of 1.7 mmol/L, elevated protein of 3.9 g/L, with lymphocytic pleocytosis (235/μL mononuclears and 90/μL polymorphs). B12, homocysteine, and copper levels were normal. Tuberculosis testing was negative. Brucella IgG and IgM were both detected on serum. CSF genus-specific brucella PCR assay confirmed the diagnosis of neurobrucellosis.

He was initially commenced on empiric high dose corticosteroids and antituberculosis therapy, and following positive brucella testing changed to doxycycline, rifampicin, and ceftriaxone. There was marked improvement in confusion, sensory ataxia, and hearing. Treatment will continue for 3–6 months.

Conclusion Neurobrucellosis is an important differential for acute and chronic lymphocytic meningoencephalitis. Cranial neuropathies and neuropsychiatric changes are common, and should raise the index of suspicion. This is the first report of longitudinally extensive dorsal columns myelitis with neurobrucellosis. Early recognition and treatment are crucial in limiting complications.

2395 CASE REPORT: NEUROSARCOIDOSIS PRESENTING WITH BIZARRE GAIT AND EXTENSIVE PULMONARY EMBOLI

[1]Marita Smith, [1]Tom Trevis, [1,2]Laurie McLaughlin, [1]Melisa Hamidon, [1,3,4]Andrew Swayne*, [1,3,4]Reuben Beer, [1,3,4]Cullen O'Gorman. *[1]Department of Neurology, Mater Centre for Neurosciences, Mater Hospital, Brisbane, QLD, Australia; [2]School of Medicine, Griffith University, Gold Coast, QLD, Australia; [3]Department of Neurology, Princess Alexandra Hospital, Brisbane, QLD, Australia; [4]Faculty of Medicine, The University of Queensland, Brisbane, QLD, Australia*

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Objectives & Methods Neurosarcoidosis accounts only for 5–10% of Sarcoidosis cases,[1] neurological complaints however maybe the first prompt for medical review.

This case report from a tertiary neurology centre illustrates that Neurosarcoidosis may present in an advanced state with a slow progressive course and neurological findings inconsistent with a single lesion location.

Results We present a 55-year-old female, referred to Neurology clinic with a six-month history of progressive neurological symptoms including transient diplopia, gait disturbance and reduced balance with left lowerlimb weakness, ataxia, altered sensation and proprioception.

MRI of the brain and spine demonstrated diffuse enhancing nodular lesions along the dura and leptomeninges in the brain, cervical and thoracic spine including the pontine and interpeduncular cisterns, medulla and cervicomedullary junction.

Further investigations with CT and PET revealing extensive pulmonary thromboembolic disease and multiple prominent, FDG avid lymph nodes. Serum and CSF ACE were significantly elevated with samples demonstrating no evidence of malignancy or infection.

Biopsy of the supraclavicular node demonstrated granulomatous lymphadenitis where well-formed granulomas replaced nodal parenchyma. These demonstrated chronic features having undergone regression and fibrosis. Findings were consistent with a diagnosis of Sarcoidosis.

The patient was initially commenced on short course of intravenous corticosteroids, with Infliximab[2] added early due to the extensive disease burden.

Conclusions This case highlights the potential varied clinical presentation of Neurosarcoidosis even in the context of diffuse and longstanding disease burden. With cranial nerve neuropathies being the most common presentation of Neurosarcoidosis[3]; this bizarre presentation emphasises the need for targeted work-up in atypical cases.

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