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

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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

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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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