048 THE INDICATIONS AND UTILITY OF ELECTROENCEPHALOGRAM, AN AUDIT OF HOSPITAL PRACTICES

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Objectives The MBS taskforce recommends discouraging use of electroencephalogram (EEG) investigations for low yield indications without neurological specialty input. This study aimed to examine the indications and utility of electroencephalogram within a hospital setting.

Methods A retrospective audit was undertaken over a 3 month period across two hospitals of adult inpatient EEGs ordered by services other than neurology.

Results Data was collected on 236 EEG encounters. 11% of EEGs performed had a definitive diagnosis of seizures on discharge, of these EEGs the report documented; a normal EEG in 7%, epileptiform activity in 19%, and non-specific slowing in 74%. 17% of Adult EEGs were performed for low yield indications. None of these EEGs resulted in change of management nor a diagnosis of seizures. An additional 14% were performed as part of a 'falls work up,' none of the EEGs for this indication resulted in a diagnosis of seizure on the discharge summary.

Conclusion This audit supports previous findings that EEGs have a low sensitivity and can not be exclusively used to attain a diagnosis. Low yield indications were common within this audit and the EEG was not clinically significant in this group. EEG should not be used to rule out seizures when the clinical suspicion for seizures is near zero and this audit identified an additional low yield category within the hospital setting as part of a 'falls work up.' This study supports the conservative use of EEG in line with the MBS funding taskforce protocol.

049 UNDER PRESSURE – AN UNUSUAL TRIGGER OF POSTERIOR REVERSIBLE ENCEPHALOPATHY

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Background A 68-year-old female, and experienced recreational diver, presented via a regional hospital to our neurology service with acute onset visual disturbance following a 30-metre open water dive.

She had no significant past medical history, specifically no history of hypertension and was on no regular medications. She was an ex-smother with a 20 pack year history

On arrival she was hypertensive to 190/100mmHg but otherwise systemically stable. She had intact visual acuity and visual fields but had difficulty distinguishing objects from complex backgrounds and described vivid visual distortions.

MRI performed acutely demonstrated multifocal areas of T2 hyperintensity within her posterior parietal and occipital lobes and a provisional diagnosis of posterior reversible encephalopathy syndrome (PRES) was made.

Further questioning revealed that the patient was diving with a 70/30% nitrogen/oxygen mixture for which she had not received appropriate training, and was diving at depths close to the recommended limits for this mixture. Diving mixtures containing higher concentrations of oxygen are used to reduce the risk of nitrogen narcosis and decompression sickness but can be associated with CNS toxicity thought to be due to significantly increased PaO2 and associated cerebral vasoconstriction.

The patient was managed with intensive blood pressure control and made a significant recovery within seven days.

Conclusions We postulate that her PRES may have been triggered by excessive cerebral vasoconstriction from hyperoxaemia in a patient with impaired vasoregulatory reserve.

REFERENCES

1. Wingelaar TT, van Ooij P-JAM, van Hulst RA. Oxygen toxicity and special operations forces diving: hidden and dangerous. *Front Psychol* 2017;**8**:1263. doi:10.3389/fpsyg.2017.01263
2. Matsuo R, Kamouchi M, Arakawa S, Furuta Y, Kanazawa Y, Kitazono T. Magnetic resonance imaging in breath-hold divers with cerebral decompression sickness. Case Rep Neurol 2014;6(1):23–27. Published 2014 Jan 24. doi:10.1159/000357169

050 PRESENCE OF VGCC ANTIBODIES WITH POSSIBLE LATE ONSET MULTIPLE ACYL-COA DEHYDROGENASE DEFICIENCY

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Introduction Voltage-gated calcium channel (VGCC) antibodies are considered specific for Lambert-Eaton myasthenic syndrome (LEMS). However, VGCC antibodies have been reported in other groups of patients without LEMS.

Multiple acyl-CoA dehydrogenase deficiency (MADD) is a type of mitochondrial myopathy which can present with a late onset form.

We report a case of a patient with positive VGCC antibodies concurrently with a diagnosis of possible late onset MADD.

Methods Clinical information and results of investigations were obtained.

Results A 75 year old woman presented with a gradual onset of proximal weakness associated with dyspnoea.

There was no relevant past medical history and family history was unremarkable.

Apart from mild proximal weakness, rest of the neurological examination was unremarkable. Deep tendon reflexes were normal.

Acylcarnitine profile on multiple occasions showed a pattern consistent with MADD. Muscle biopsy showed mild mitochondrial changes.

VGCC antibodies were detected on 2 separate occasions (86 and 100 pM; Ref Range <30pM). CMAP amplitudes and repetitive stimulation was normal with no facilitation nor decrement found. Position emission tomography was unremarkable.

The patient's symptoms were thought to be secondary to MADD, therefore was treated with riboflavin, Q10 and carnitine and described significantly improved proximal strength and function.

Conclusion It has been generally considered that the VGCC Ab has a high sensitivity and specificity for LEMS. However,

in a study of 100 neuromuscular patients with elevated VGCC antibodies, only 6 patients were diagnosed with LEMS. This case illustrates the importance of applying appropriate clinical judgement with results of investigations.

051 ISCHAEMIC STROKE AS THE ONLY MANIFESTATION OF ANTI-NEUTROPHILIC CYTOPLASMIC AUTOANTIBODY ASSOCIATED VASCULITIS

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Background The diagnosis of anti-neutrophilic cytoplasmic autoantibodies (ANCA) associated vasculitis (AAV) in first episode strokes is more challenging compared with consecutive strokes, especially in patients lacking other clinical features of AAV.

Methods/Results Here, we present the case of a 71-year-old female with positive myeloperoxidase (MPO) ANCA and negative proteinase 3 (PR3) ANCA. Our patient presented with a one-week history of pyramidal weakness in both upper and lower limbs, hyperreflexia and clonus. Brain MRI demonstrated widespread bi-hemispheric cortical and deep white matter acute infarcts. Investigations revealed eosinophilia on full blood examination and positive MPO-ANCA antibody. Consistent with features of stroke secondary to AAV, the deep penetrating vessels were predominantly affected resulting in a multifocal distribution of infarcts in the white matter. MPO-ANCA positive vasculitis diseases are more commonly associated with renal, pulmonary and cutaneous manifestations, however our patient did not have other systemic manifestations of AAV, and her presentation was solely limited to the CNS.

Conclusions This case highlights the challenges of diagnosing primary CNS vasculitis, especially an atypical MPO-ANCA positive disease that fails to have the classical clinical signs and course.

052 PREDICTIVE VALUE OF SIGNS AND SYMPTOMS IN CODE STROKES FOR DIAGNOSIS OF ISCHAEMIC STROKE OR TIA

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Objectives This study aimed to determine the history features, signs and symptoms identified during a code stroke that correlate with the diagnosis of ischemic stroke or transient ischemic attack (TIA). We investigated the rate of stroke mimics and aimed to develop a clinical prediction model.

Methods Consecutive code stroke presentations to a primary stroke centre were recruited. Patient characteristics, medical history, signs or symptoms on activation of code stroke were collected from the medical record. Diagnosis of ischemic stroke was determined by radiographic evidence of infarction. Univariate analysis and multivariable logistic regression analysis were used to determine the features that predict ischemic stroke/TIA versus mimic.

Results Among 493 code strokes, 64.5% were mimics. The most commonly diagnosed mimics were migraine, peripheral vertigo and seizure. Upper limb sensory change (OR 3.27 [95% CI, 1.75-6.11]), hemiplegia (OR 2.70 [95% CI, 1.65-4.43]), dysphasia (OR 2.62 [95% CI, 1.56-4.40]) and history of atrial fibrillation (OR 2.01 [95% CI, 1.14-3.54]) or hypertension (OR 1.77 [95% CI, 1.10-2.83]) are highly predictive of stroke/TIA. Headache (OR 0.40 [95% CI, 0.23-0.69]) is predictive of a mimic. Dizziness and vertigo were more common in stroke mimics. C-statistic for the study models ranged from 0.70 to 0.76.

Conclusion Objective signs such as unilateral motor weakness and dysphasia are highly predictive of ischemic stroke/TIA whereas symptoms of headache and dizziness are suggestive of stroke mimic. Stroke mimic rate is influenced by local prevalence and threshold for code stroke activation. Incorporating positive and negative predictive features may improve future stroke prediction tools.

053 EOSINOPHILIC GRANULOMATOSIS WITH POLYANGIITIS PRESENTING WITH SIMULTANEOUS CENTRAL AND PERIPHERAL NERVOUS SYSTEM INVOLVEMENT

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Objective Peripheral nervous system involvement in eosinophilic granulomatosis with polyangiitis (EGPA) is well described. However, central nervous system involvement is uncommon. We describe a case of simultaneous central and peripheral nervous system involvement in EGPA. The diagnosis was confirmed on histopathology. A high index of suspicion is needed to initiate prompt treatment for this potentially life-threatening condition.

Report 85-year-old male presented with subacute generalised lower limb weakness on the background of known eosinophilic asthma treated with interleukin-5 inhibitor (mepolizumab) and prednisolone. During his admission, he developed transient aphasia, followed by mononeuritis multiplex involving the left median and femoral nerves and purpuric rash over few days. Laboratory investigations showed no peripheral eosinophilia. ANCA revealed a borderline elevated myeloperoxidase antigen (MPO) of 21 U/mL. MRI brain revealed multiple small foci of diffusion restriction within the basal ganglia bilaterally, as well as paranasal sinusitis. MRA/CT cerebral angiogram was unremarkable. Prolonged telemetry and TOE did not show any central embolic cause. Left lateral gastrocnemius muscle biopsy revealed fibrinoid necrosis associated with adjacent eosinophils. Induction with intravenous cyclophosphamide was commenced along with high dose corticosteroids. He has been neurologically stable since.

Conclusion Simultaneous peripheral nervous system involvement with multiterritory stroke should heighten the suspicion for systemic vasculitis. Cerebral arterial imaging may be normal in small to medium vessel vasculitis such as EGPA. .Pre-existing mepolizumab therapy may make diagnosis more challenging by normalising pathology results. Histopathology can be of value to confirm diagnosis.

REFERENCES

1. Keogh KA, Specks U. Churg-Strauss syndrome: update on clinical, laboratory and therapeutic aspects. *Sarcoidosis Vasc Diffuse Lung Dis* 2006 Mar;**23**(1):3–12.