Letters to the editor

OVERVIEW

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Posterior reversible encephalopathy syndrome

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Editor – Tan and Tan reported a case of severe hypertension in which the patient had mild symptoms and normal neurological examination.[1] Although interesting, we are concerned about the diagnosis of such a case.

Posterior reversible encephalopathy syndrome (PRES), also termed reversible posterior leukoencephalopathy syndrome, is a clinico-radiological diagnosis.[2] The occurrence of PRES is related to autoregulation failure of cerebral blood circulation and/or endothelial dysfunction.[2] Vasogenic oedema revealed by apparent diffusion coefficient (ADC) maps as increased signal intensity, preferably involving the posterior white matter, and reversible clinical manifestations like seizures, altogether contribute to the diagnosis of PRES.[3] As mentioned in their abstract, 'hypertensive encephalopathy (HE) is a subset of posterior reversible encephalopathy syndrome'.[1] This appears problematic, as the latter should be a subset of the former. HE may occur with or without abnormal neuroimaging findings, the former of which may be diagnosed as PRES.

The 52-year-old man complained of worsening occipital headache and giddiness and denied weakness, blurring of vision or altered sensation. Neurological examination yielded no positive findings. In this regard, the diagnosis of encephalopathy is only supported by headache, giddiness and abnormal computed tomography (CT) findings. However, according to the National Institute of Neurological Diseases and Stroke, encephalopathy is a term for any diffuse disease of the brain that alters brain function or structure; the hallmark of encephalopathy is an altered mental state; depending on the type and severity of encephalopathy, common neurological symptoms are progressive loss of memory and cognitive ability, subtle personality changes, inability to concentrate, lethargy and progressive loss of consciousness.[4] Global brain dysfunction is also referred to by the Nature Publishing Group.

Taken together, this is a case of severe hypertension with unidentified hypodense appearance in CT. The diagnosis of encephalopathy is not supported by the clinical manifestations. Magnetic resonance imaging is necessary for a reliable diagnosis. ■

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Cardiac investigations in acute ischaemic stroke

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Editor – I read with interest the retrospective study from Bahl *et al* highlighting cardiac aetiologies (up to 24%) in an unselected young population with acute ischaemic stroke (n=167).[1]

Atrial fibrillation (AF) is responsible for up to one-third of acute ischaemic strokes and may be the index presentation of AF.[2] With an established efficacy of oral anticoagulation in the prevention of stroke associated with thromboembolic events and AF, thorough cardiac investigations are warranted to reduce morbidity and mortality, particularly in a young patent population.

Bahl *et al* investigated patients for AF with ambulatory electrocardiography (ECG) monitoring and a mean duration of 68.4 hours (2.9 days), however detection of AF was low (1.8%; 2/109). The authors acknowledged the need for prolonged ambulatory monitoring and the AF-SCREEN collaboration has endorsed handheld patient activated ECG devices as a preferred screening tool.[3] The National Institute for Health and Care Excellence (NICE) has appraised similar technology (AliveCor®), reported to be cost-effective and have both a high sensitivity and specificity in the detection and interpretation of AF.[4] Furthermore, EMBRACE demonstrated that AF lasting 30 seconds was detected in 16.1% of cryptogenic stroke patients with use of a 30-day event triggered recorder compared with 3.2% with a 24-hour monitor,

and this led to anticoagulation in nearly double the number of patients in the intervention group.[5]

Interestingly, 9% (15/167) of patients had an intracardiac source of embolus ('heart failure and thrombus' and 'valvular heart disease') and 25.1% (42/167) of patients had no underlying cause. Despite this, only 4.2% (7/167) of patients had transoesophageal echocardiography (TOE). A thrombus located in the left atrium or, more precisely, the left atrial appendage (LAA) is the most prevalent source of intracardiac emboli and is typically associated with AF. TOE is the imaging modality of choice for the evaluation of LAA.[6,7] Furthermore, in the absence of diagnosed AF, left atrial or LAA abnormalities may be a compelling indication for prolonged ECG monitoring.

In summary, investigation for aetiology of stroke in young patients should involve scrupulous cardiac investigations identifying those patients who would benefit from prolonged ambulatory ECG monitoring and increased utilisation of TOE. ■

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Procalcitonin for patient stratification and identification of bacterial co-infection in COVID-19

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Editor – an abundance of biomarkers has been measured in hospitalised patients with COVID-19. Initial reports from China have shown that most patients with COVID-19 did not have elevated procalcitonin (>0.5 μg/L).[1,2] However, elevated levels were found more frequently in severe cases and in patients who died.[2–4]

Variance in procalcitonin levels have previously been proposed to differentiate systemic inflammation of bacterial origin from viral origin in community acquired pneumonia and sepsis, with a significant rise indicating bacterial infection.[5,6] The lack of a procalcitonin rise in viral infections may be due to virus-stimulated production of interferon-γ by macrophages, which inhibits TNF-α in the immune response.[5] The presence of lower procalcitonin levels has been shown to have a 94% negative predictive value for bacterial co-infection in intensive care unit patients with confirmed influenza A(H1N1)pdm09.[7] Therefore, we suggest that raised procalcitonin observed in COVID-19 could be due either to bacterial co-infection, which is itself causing increased severity and driving systemic sepsis, or as a direct marker of a more severe or widespread viral infection.

As such, procalcitonin measurement on admission may be a useful marker to firstly predict patient deterioration in hospital and secondly, non-elevated procalcitonin on admission may be a good predictor of the absence of bacterial co-infection and allow the more targeted use of antimicrobials thus promoting antibiotic stewardship. Further work is needed to correlate the presence of raised procalcitonin and the presence of bacterial co-infection in COVID-19 patients. ■

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