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Ultrasound optic nerve sheath diameter measurement in optic neuritis

Dear Editor,

We read with great interest the case report written by Yee et al. regarding four cases in which they demonstrated the usefulness of transbulbar ultrasonography in evaluating the optic nerve to identify the presence of optic neuritis [1].

We congratulate the authors for their significant article because it really emphasizes the effectiveness of this technique in diagnosing orbital pathologies, but we would like to comment several aspects concerning the measurement of the optic nerve sheath diameter (ONSD).

In their case series, Yee et al. measured ONSD with B scan ultrasound technique, that is generally used to diagnose ocular diseases [2,3] but unluckily is less sensitive in measuring the orbital structures, because it is affected by the blooming effect [4-9]. This is related to the absence of a standard sensitivity setting in performing B scan and it means that, in case of this ultrasound method, if we measure ONSD with a lower sensitivity setting, this will give bigger dimensions compared to the ones obtained with an increased sensitivity setting. This effect could be less significant when we deal with large lesions, but it could be misleading if we suppose a difference inferior to 0.5 mm, as in case of ONSD appraisal.

For this reason, in case of future studies, we would like to suggest utilizing the Standardized A Scan technique: this examination makes these measurements objective and exacter, because it shows easily discernible high reflective spikes from the interface between arachnoid and subarachnoidal fluid, and it is blooming effect free too. In addition, it also permits more accurate reference range values, that can be used worldwide [10,11].

Furthermore, we would like to highlight that it is possible to distinguish between an increase in ONSD related to the presence of an optic neuritis or an optic nerve meningioma and that one caused by intracranial hypertension, thanks to "30 degree test" performed with a scan ultrasound examination [12-15].

Lastly, we would like to put attention on the relevance of learning satisfactorily how performing ultrasonography, avoiding errors and obtaining more trustworthy data. In fact, in literature, there are some papers where the measurements were performed in a wrong way [16], due to the blooming effect and to the difficulties in the exact placement of the probe and markers; moreover, skill to get reproducible measurements and a very good knowledge of orbital and ocular anatomy are necessarily required [17].

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Which one is more important in traumatic brain injury: Hypotension or hypoxia?

I have greatly enjoyed reading the recently published article by Seo et al. [1]. In this study, the authors examined the association between hypoxia level and outcomes according to shock status in traumatic brain injury (TBI) patients. They found that the mortality rates were 49.4% in severe hypoxia, 30.7% in mild hypoxia, 18.5% in normoxia. Mortality rates were 47.1% in TBI patients with shock status and 20.5% in non-shock TBI patients. There was a trend toward worsened outcomes with mild and severe hypoxia in patient with and without shock, however, the only met statistical significance for patients with both severe hypoxia and non-shock status. These results suggested that, in patients already suffering hypotension, hypoxia did not add any significant effect.

Previous studies have revealed that hypotension (systolic blood pressure < 90 mmHg) and hypoxemia (P_aO_2 < 60 mmHg) are important prognostic factors and should be avoided in patients with TBI [2]. However, there are no data in the literature which one is the more important factor (hypoxia or hypotension) influencing the prognosis. The study by Seo et al. gives rise to thought that hypotension is more important than hypoxia. However, in another study, Spaite and colleagues showed that had mortality rate was 20.7% in hypotensive patients, and 28.1% in hypoxic patients suggesting hypoxia as more important risk factor for TBI patients [3]. Therefore I think that further studies are needed to reveal whether hypotension or hypoxia is more important in patients with TBI.

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Diagnostic considerations in detecting apical hypertrophic cardiomyopathy while utilizing point-of-care ultrasound

The emergency department (ED) clinical approach to patients with unexplained syncope has ushered in an era of advances in the point-of-care ultrasound (POCUS) practice. Cardiac ultrasound (US) is a key application that is often utilized in young patients with syncope when screening for structural abnormalities such as hypertrophic cardiomyopathy (HCM). The application of the cardiac US for this screening, however, can be hindered by phenotypic variability of hereditary HCM. The apical variant of HCM constitutes a minority of all cases (<3%) and is generally associated with a benign prognosis. We describe a 28-year-old woman and former collegiate middle-distance/endurance runner who presented to the emergency department (ED) with syncope. Earlier in the day, she was one mile into a planned ten-mile run, when she reached an intersection requiring her to stop. Upon stopping abruptly, she experienced a feeling of warmth, followed by shortness of breath, blurred vision and subsequent loss of consciousness. The patient had syncopized several times before in her life—all in the setting of abruptly stopping after a warm-up exercise. She denied any other complaints and had no other relevant past medical history. Family history was notable for myocardial infarction (MI) in the patient's father while he was in his 50s, as well as sudden cardiac death of her paternal grandfather while he was also in his 50s. Upon arrival to the ED, blood pressure was 93/59 mm Hg; heart rate, 59 beats per minute; oxygen saturation, 99% on room air, and temperature 98.4 F. On examination, the patient was well appearing, alert, oriented and in no acute distress. Orthostasis was not present. Breath sounds were clear and equal bilaterally. Cardiac examination demonstrated regular rhythm without murmur, rub or gallop. Distal pulses were intact and jugular venous distension was absent.

Electrocardiogram (ECG) was notable for sinus bradycardia with deep and symmetric T-wave inversions in leads I, II, III, aVF, V3-6, as well as ST depressions in leads V3-6. The R waves were also very prominent in leads II, III, aVF, V3-6. These findings were consistent with left ventricular hypertrophy (LVH) with strain pattern. Point-of-care cardiac ultrasound (US) demonstrated no pericardial effusion and grossly normal ejection fraction without obvious evidence of segmental subaortic septal hypertrophy or LV outflow obstruction.

Inpatient workup included a comprehensive echocardiogram, which demonstrated near-obliteration of the left ventricular apical cavity at end systole and prominent LVH in the apex measuring 11–13 mm, with a "spade" shaped left ventricle, without evidence of outflow obstruction (Fig. 1). The patient underwent stress testing with an exercise capacity of 17 metabolic equivalents of task, with an ECG that was non-diagnostic for ischemia secondary to a baseline LVH, but showing no arrhythmias or ectopic beats during exercise or recovery. The patient also underwent cardiac magnetic resonance imaging at rest, including conventional structural and functional imaging, which showed concentric thickening of the apical segments of the left ventricle (LV) with maximal wall thickness of 17 mm and associated mid-myocardial late gadolinium enhancement involving 6% of the left ventricular myocardium—establishing the diagnosis of apical HCM (AHCM) (Fig. 2).

Patients with HCM exhibit a variable phenotype with LV hypertrophy being the main manifestation, and diastolic dysfunction and dynamic LV outflow tract obstruction as important pathophysiologic features. The diagnosis is confirmed when thickening ≥15 mm is noted anywhere on the LV wall during end diastole [1].Wall thickening is frequently asymmetric, and most commonly involves the basal septum, just below the aortic valve, leading to LVOT obstruction. It is important to note, however, that our patient exhibits AHCM, a rare variant of HCM (<3%) involving solely the apex of the LV [2,3]. In AHCM, transthoracic echocardiography (TTE) will demonstrate hypertrophy of the LV apex and a spade-like left ventricular cavity during systole when aided by intravenous echo-contrast material [2,4]. Cardiac US that focuses solely on LV outflow and basal septal thickening conveys a possibility of false negative diagnosis. Careful assessment of the entire LV including the apex may preclude this pitfall.

Studies have generally indicated a benign prognosis for individuals with AHCM. Nevertheless, there have been case reports of patients with AHCM developing potentially serious arrhythmias including atrial fibrillation, supraventricular tachycardia and ventricular tachycardia [1-5]. Notably, all of these reports have been made in individuals over the age of 50. In the case of our relatively young and exceptionally healthy patient, it is unlikely syncope was secondary to outflow obstruction,