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Letter to the Editor

Right Hemispheric Hypoperfusion Resulting from Cannulation for ECMO should be Confirmed Using Multimodal MRI Rather than qEEG

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Letter to the Editor

We read with interest the article by Pierson *et al*. regarding a two-year-old boy with Shiga toxin-associated hemolytic uremic syndrome who required mechanical ventilation starting on the ninth day of hospitalization due to pulmonary hemorrhage and biventricular heart failure [1]. Despite these measures, he developed asystole on hospital day 12 and was emergently placed on extracorporeal membrane oxygenation (ECMO), with cannulation performed via the right common carotid artery (RCCA) and the internal jugular vein [1]. Neurological examination following the cessation of ECMO treatment, while the patient was sedated, revealed mutism, ocular divergence, and left-sided hemisyndrome. These findings were attributed to an ischemic stroke in the left pons and a narrowing of the right common carotid artery [1]. A retrospective quantitative electroencephalography (qEEG) analysis showed a relative attenuation of alpha power and an increase in delta power in the right hemisphere, with the total amplitude being higher on the right than on the left [1]. The study is insightful, yet several points warrant discussion.

A first point is that ischemic stroke cannot be diagnosed via EEG but only through cerebral imaging. EEG changes associated with ischemic stroke are nonspecific and include focal slowing, intermittent rhythmic activity, and spikes or sharp waves, occasionally accompanied by periodic lateralized epileptiform discharges (PLEDs) [2]. Classically, EEG recordings in ischemic stroke demonstrate focal slowing, predominantly in the delta and theta frequency bands, as well as an attenuation of hemispheric background activity (reduced amplitude). These changes often occur within seconds to minutes following a reduction in blood flow [2]. In addition, patterns such as frontal intermittent rhythmic delta activity (FIRDA) may occur, which can indicate deep-seated cortical or subcortical lesions [2]. qEEG can provide metrics such as the delta-alpha ratio (DAR) or the Brain Symmetry Index (BSI); these enable the identification of cerebral asymmetries and the monitoring of regional blood flow [3].

A second point is that, in sedated, intubated, and ventilated patients, hypoperfusion of a cerebral vascular territory can be most reliably assessed using multimodal MRI. Perfusion-weighted imaging (PWI) is particularly useful in this regard, as it demonstrates a hypointense signal in hypoperfused vascular territories [4]. Diffusion-weighted imaging (DWI) and the apparent diffusion coefficient (ADC) may appear normal in such cases [4]. The most important PWI parameter is "time-to-peak" (Tmax); a delay of 4 to 6 seconds indicates critical tissue hypoperfusion. A prolonged mean transit time (MTT) indicates delayed blood flow through the capillary bed, thereby identifying under-perfused cerebrovascular regions. A drop in cerebral blood flow (CBF) below certain thresholds can also indicate active hypoperfusion. Cerebral blood volume (CBV) is typically normal but may be elevated due to compensatory mechanisms and reduced in the core areas of an infarct [4].

A third point is that the narrowing of the right common carotid artery (RCCA) was not adequately explained [1]. Since the caliber of the RCCA normalized two weeks after the initial TOF angiography, vasospasm cannot be ruled out. It is also conceivable that a carotid artery dissection occurred during cannulation for ECMO. To rule out dissection, CT angiography, digital subtraction angiography, and axial T1-weighted images with fat saturation would have been helpful, as this modality can directly visualize a mural hematoma [5].

Overall, multimodal MRI is preferable to qEEG for detecting hemispheric hypoperfusion resulting from cannulation of the RCCA. Hypoperfusion via the cannulated RCCA is typically compensated for by cross-flow from the contralateral left carotid artery.

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