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Letter to the Editor

Whether Migraine in Mitochondrial Disorders is Indeed Associated with Stroke-like Lesions must be confirmed by means of an MRI Scan

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Letter to the Editor

We read with interest the article by Yan *et al*., which presents a multicenter cross-sectional study on the clinical presentation of migraine in a cohort of 48 patients with mitochondrial disorders (MIDs) manifesting as encephalopathy or myopathy [1]. The study also aimed to investigate a potential link between migraine and mitochondrial dysfunction [1]. Half of the MID patients developed a migraine within a one-year observation period [1]. Epilepsy and lactic acidosis were found to be independently associated with migraine [1]. Of 29 patients with MELAS syndrome, 12 reported acute headaches that either accompanied or preceded focal neurological deficits or seizures; five additional MELAS patients, who did not exhibit this specific type of headache, were diagnosed with migraine [1]. The authors concluded that the prevalence of migraine in patients with MIDs is five times higher than in the general population and that epilepsy and elevated serum lactate levels are associated with migraine [1]. While the study is noteworthy, several aspects warrant further discussion.

A first point is that no systematic cerebral angiography was performed on the patients included in the study [1]. Given that migraine is associated with abnormalities in the reactivity of cerebral arteries [2], it is crucial to assess the state of the cerebral vasculature - for instance, through magnetic resonance angiography, CT angiography, or digital subtraction angiography. It would be particularly informative to know whether the reactivity of the cerebral vessels to vasoconstrictors or vasodilators was impaired.

The second point is that lactate was measured only in the serum, not in the cerebrospinal fluid (CSF) [1]. To determine whether lactic acidosis is associated with the occurrence of migraine, measuring lactate in the CSF is essential. This can be done either through CSF analysis following a lumbar puncture or by using magnetic resonance spectroscopy (MRS). Although MRS was performed on 32 patients, it remains unclear what is meant by "lactate peaks at the lesion." Are the authors referring to a stroke-like lesion (SLL), an ischemic lesion, vasogenic oedema following a seizure, or a hypoxic lesion? Understanding the nature and underlying pathophysiology of the lesions detected is crucial for assessing its potential link to migraine.

The third point is that the medications regularly taken by the patients in the study cohort were not included in the analysis [1]. Since numerous drugs can trigger migraine attacks [3], it would have been imperative to exclude patients taking such migraine-inducing substances from the study. Medications known to potentially trigger migraine attacks include, among others, nitroglycerin, sildenafil, tadalafil, estrogens, serotonin reuptake inhibitors, bupropion, proton pump inhibitors, excessive use of ibuprofen or naproxen, decongestant nasal sprays, cocaine, and amphetamines. As long as patients taking these substances were not excluded from the study, the results and their interpretation remain unreliable.

The fourth point is that it was not reported whether the 29 patients with acute headache preceding or associated with focal neurological deficits and/or seizures (AHF) did, in fact, exhibit a stroke-like lesion (SLL) - the morphological equivalent of a stroke-like episode (SLE) [4]. In how many of the 17 AHF patients did a migraine occur either before or concurrently with a SLE? Was the migraine associated with other focal neurological deficits that could not be attributed to a SLE? What did the MRI findings look like in these patients? Was cerebral imaging actually performed for all the patients included in the study? How many of the 29 patients had a seizure without a SLL?

The fifth point is that it is unclear why patients for whom mitochondrial myopathy was the sole manifestation of their MID were included in the study. In patients with mitochondrial myopathy, additional cerebral involvement is rather unlikely. What was the rationale for including patients with mitochondrial myopathy in a study on migraine?

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