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Letter to Editor

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Migraine in mitochondrial disorders

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Letter to the Editor

In a recently published cross-sectional cohort-study of 93 consecutive patients with a mitochondrial disorder (MID) Vollono et al. found that 33 patients (35.5%) had migraine in the absence of a stroke-like episode (SLE) [1]. The article raises a number of comments and concerns.

Among the 33 patients with migraine, 6 had a history of SLEs [1]. Did migraine occur only during the SLEs or between the SLEs? Since SLEs may last for weeks we should be informed about the interval between onset of SLEs and onset of migraine to assess if migraine was associated with SLEs which is frequently the case [2].

The study lacks information about the triggers of migraine attacks [1]. Did the authors ask for triggering factors in addition to exercise? Were the triggers of migraine different between specific MIDs included or different compared to a non-mitochondrial cohort?

Did those with migraine more frequently have white matter lesions/cortical atrophy or a history of epilepsy or epileptic activity on EEG than those without migraine? How often did the EEG show epileptic activity in the absence of epilepsy among patients in both groups?

Migraine may not only be a phenotypic manifestation of MELAS, MERRF, CPEO, or MNGIE but also of Leigh-syndrome (LS), Leber's hereditary optic neuropathy (LHON), Alpers-Huttenlocher disease (AHS), and in non-specific mitochondrial multiorgan disorder syndrome (MIMODSs) [Finsterer, submitted] Were any of these other specific MIDs found among the 13 patients with unclassified MID?

How often was the family history positive for migraine among the 33 MID patients with migraine? How often was the family history negative for MID but positive for migraine, suggesting that migraine could have occurred independently of the underlying MID?

Six patients (female, n=4) had migraine with aura of which 4 had epileptic activity on EEG [1]. Three had a history of SLEs [1]. How many of the 6 had optic atrophy or abnormal visually-evoked potentials? How many of those with aura had ophthalmologic involvement? In the patients with migraine with aura did the retinal spreading depression model of migraine with aura apply as an explanation?

In six patients the MID was due to a mutation in a nuclear gene but in 27 patients due to a mtDNA mutation [1]. Among those with a mtDNA mutation, was the frequency and intensity of migraine correlated with the heteroplasmy rate?

How many of the 33 with migraine were under a ketogenic diet, which has been shown beneficial in some MID patients in general and particularly in patients with epilepsy or migraine [3]? There are also indications that NO-precursors, such as L-arginine or L-citrulline may have a beneficial effect on mitochondrial migraine. Were these compounds applied in any of the 33 patients?

Did the authors also measure serum lactate/pyruvate and did patients from the migraine group undergo MR-spectroscopy (MRS)? Did frequency and severity of migraine correlate with serum or CSF lactate?

Overall, this interesting study may profit from provision of additional data about the family history for migraine and MID, the current medication, and triggering factors. Serum and CSF lactate and heteroplasmy rate could be useful parameters in relation to migraine.

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