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Letter to the Editor

Migratory Vasodilation Within Stroke-Like Lesions is a Compensatory Mechanism for the Metabolic Impairment in such Areas

Josef Finsterer
Department of Neurology, Neurology & Neurophysiology Centre, Vienna, Austria

Corresponding Author: **Josef Finsterer**

Letter to the Editor

We read with interest the article by Luo *et al*. on a 24-year-old man with mitochondrial encephalopathy, lactic acidosis, and stroke-like syndrome (MELAS) who experienced migratory vasodilation of the cerebral arteries during three separate stroke-like episodes (SLEs) [1]. The dilation was observed particularly in the posterior and middle cerebral arteries [1]. The study is noteworthy but requires discussion.

The first point is that hyperperfusion of stroke-like lesions (SLLs), the morphological correlate of a SLE on imaging, is a known phenomenon in patients with SLEs [2]. One of the characteristics of SLLs in multimodal MRI is that perfusion-weighted imaging in MRI shows the SLL as hyperintense. The cause of hyperperfusion is unknown, but it can be speculated that hyperperfusion is a compensatory mechanism for the metabolic disturbance within the SLL. With hyperperfusion, the body attempts to transport oxygen and substrates for energy production to the area that is no longer able to produce sufficient energy. Hypometabolism within SLLs can be detected by hypometabolism in fluorodeoxyglucose positron emission tomography (FDG-PET) [3].

The second point is that no heteroplasmy rates for the m.3243A>G variant have been reported [1]. Since the phenotypic expression of the m.3243A>G variant depends heavily on the heteroplasmy rates in the affected tissues, it would be useful to test patients for these determinants of the phenotype. Knowledge of heteroplasmy rates and their tissue distribution is also useful for genetic counseling and for predicting the course of the disease. In addition to heteroplasmy rates, the phenotype is determined by haplotype, mtDNA copy number, tissue distribution, energy requirements, age, lifestyle factors, nuclear-mitochondrial interactions, and polymorphisms in nuclear genes involved in mitochondrial metabolism [4].

The third point is that it was not reported whether the m.3243A>G variant was also detected in the patient's mother [1]. If the mother was also a carrier of the m.3243A>G variant, we should know whether she manifested phenotypically or whether she was asymptomatic. Of particular interest is whether she also exhibited phenotypic features of MELAS, especially SLEs. Since mtDNA mutations are transmitted through the maternal line in 75% of cases, it is very likely that she passed on the pathogenic variant to her son.

The fourth point is that no long-term patient outcomes have been reported [1]. Long-term outcomes are of particular interest because SLLs can take different courses. They can regress and disappear completely without leaving any structural lesions. However, they can also lead to white matter lesions, focal atrophy, laminar cortical necrosis, cysts, or toe sign [2]. SLLs also have a strong tendency to recur, as in the index patient, making it very likely that the patient will develop further SLLs as the disease progresses.

Overall, compensatory hyperperfusion is a typical feature of SLLs and is most likely a compensatory response to impaired focal metabolism.

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