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Cerebral Hyper-perfusion in Stroke-like Lesions may be Secondary and not Pathogenic

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Letter to Editor

In a recent article by Li et al. [1], a study of cerebral perfusion in 20 patients with mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS) by MRI/MRA was presented. We have the following comments/concerns.

We do not agree with the statement that stroke-like lesions (SLLs), the morphological equivalent of stroke-like episode (SLEs), predominantly affect the grey matter. SLLs variably affect both grey and white matter and are not confined to either of them.

Vasodilation before or during the acute stage of a SLE is most likely a secondary phenomenon either due to enhanced demand and consumption of metabolites during seizures frequently associated with the SLEs or due to a compensatory reaction to the impaired utilization of metabolites during the metabolic crisis in the respiratory chain or OXPHOS pathways.
Macroangiopathy in mitochondrial disorders (MIDs) may not only be a primary consequence of the underlying mutation but may be a secondary phenomenon of the risk factor profile in MIDs. MID patients frequently develop diabetes, arterial hypertension, or hyperlipidemia [2] and are thus prone to develop atherosclerosis.

The authors investigated 20 patients with MELAS but they found a causative mtDNA mutation in only 17 patients [1]. Was the diagnosis in the remaining three patients based only on clinical findings? Was the phenotype in these three patients associated with mutations in nDNA-located genes?

It would be also interesting to know the degree of heteroplasmy of the m.3243A>G mutation and in which tissues the heteroplasmy rate was determined. We also should be informed in how many of the patients the family history was positive for the disease and how many of the included patients had previous SLEs.

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In patient 8, who had bilateral SLLs in the parietal lobes, matching dilation of the MCA and dilatation of the right ACA were found [1]. We do not agree that the right ACA matches with a right parietal SLL. The right parietal lobe is predominantly vascularized via the right MCA.

How to explain dilation of the right MCA and the right PCA in asymptomatic patient 12 who did not present with a SLL? Why was the MRI carried out if the patient was asymptomatic?

For none of the 20 patients the regular medication is mentioned. From a number of compounds it is well known that they have a dilating effect on cerebral arteries [3,4], why it is essential to mention the medication these 20 were on at the time of the MRI investigation.

Not only dilation of cerebral arteries has been reported in MELAS patients but also vasospasms, which is why these patients receive NO-precursors (L-arginine, L-citrulline) during the acute stage of the SLE [5]. In how many of the included patients were vasospasms seen and how many of the patients received NO-precursors which could also explain the presented hyperperfusion.

Overall, this interesting study could profit from provision of additional data concerning the individual and family history and the genetic findings, and from provision of the current medication and the therapy these patients received during SLEs. The vascular hypothesis of SLEs currently remains unproven.

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