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LETTER TO THE EDITOR

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Flow-void and hyperintense vessel sign are unsuitable to characterise a non-vascular stroke-like lesion

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Abstract

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Keywords: MELAS, stroke-like episode, stroke-like lesion, MRI, vasculopathy

1 | LETTER TO THE EDITOR

With interest we read the article by Chong et al. about a study of 13 patients with mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS) and 20 patients with acute ischemic stroke (AIS) for the sensitivity/specificity of the flow-void sign (FVS) and hyperintense vessel sign (HVS) on MRI [1]. It was found that MELAS is characterised by the presence of the FVS and absence of the HVS, and vice versa in AIS [1]. The study is appealing but raises the following comments and concerns.

We disagree with the statement that a SLE is due to microangiopathy [1]. Though the pathophysiology of SLLs has been occasionally explained with a vascular compromise, there is increasing evidence that a SLL is due to a focal metabolic problem that is dynamic and spreads from the cortical grey matter to the subcortical white matter and regresses over time [2]. The strongest arguments against the vascular hypothesis are that the extension of a SLL is never confined to a vascular territory and that there is no diffusion weighted imaging (DWI) hyperintensity and apparent diffusion coefficient (ADC) hypointensity.

Though there is primary and secondary mitochondrial vasculopathy [3], it is not responsible for the SLL. SLL's are frequently triggered by emotional or physical stress, drugs, or infections, including seizures [4]. The large intracerebral arteries in MELAS remain patent throughout the duration of a SLL.

Flow-void is a misleading term since it implies absence of flow although the vessel is patent. The phenomenon is related to time-of-flight/spin-phase-effects or in-plane motion [5]. Thus, it is conceivable that the FVS is an artefact. Disadvantage of the HVS is that it may not be present in AIS due to microangiopathy.

Supplementary information The online version of this article (10.15520/jmrhs.v4i6.363) contains supplementary material, which is available to authorized users.

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A limitation of the study is that heteroplasmy rates of the variants m.3243A>G, m.5543T>C, and m.10191T>C were not provided. There is also no mentioning of the mtDNA copy number or haplotypes of the 13 MELAS patients. Since heteroplasmy, mtDNA copy number, or haplotype may influence the phenotype and thus the MRI findings, they should be included in the evaluation.

Overall, the study has limitations which challenge the results and their interpretation. Based on the considerations provided, we propose to diagnose a SLL upon the clinical presentation, and T2-, DWI-, and PWI-hyperintensity, and upon oxygen-extraction fraction (OEF)-hypointensity rather than presence of the FVS and absence of the HVS. Additionally, the EGG can be helpful in delineating between AIS and a SLL.

Declarations

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Conflicts of interest: none

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Informed consent: was obtained

The study was approved by the institutional review board

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