Does 3D pASP truly reflect hyperperfusion in stroke-like lesions?

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Keywords
mitochondrial, mtDNA, phenotype, genotype, myopathy, muscle biopsy

Letter to the Editor

In a recent article, Li et al. reported about the application of 3D pseudocontinuous arterial spin labelling (3D pCASL) to 9 patients with mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS) syndrome within one month after onset of a stroke-like episode (SLE)1. 3D pCASL revealed hyperperfusion during the acute and subacute stage of a SLE in all 9 MELAS patients1. We have the following comments and concerns.

Since stroke-like lesions (SLLs), the morphological equivalent of SLEs, are undergoing dynamic changes with regard to morphology, extension, distribution, and perfusion after onset of the lesion2, it is important to know how many days exactly after onset of a SLE magnetic resonance imaging (MRI) investigations had been carried out. Since MRIs were performed out within 1 month after onset of a SLE, it is conceivable that the results may have been strongly influenced by the heterogeneous pathophysiological conditions and dynamic changes of the SLLs over time. Within one month, a SLL may disappear, may be accompanied by a second one in another location, may turn into a mixture of a vasogenic and a cytotoxic edema, or may become a white matter lesion, a cortical laminar necrosis, focal atrophy, or turn into the toenail sign3. The authors themselves present the data of a 17yo patient in whom follow-up MRI showed hyperperfusion whereas the initial investigation had shown hypoperfusion, suggesting that findings on 3D pCASK are time-dependent1. SLLs may not only be characterised by hyperperfusion but also by hypoperfusion, as has been shown by 99m-Tc-HMPAO SPECT4.

Furthermore, we should be informed how many of the 9 had a cytotoxic edema, a vasogenic edema, or a mixture of both on MRI. According to table 2, only 3 had a vasogenic edema and only one had a cytotoxic edema. In the remaining five patients, hypointense and hypertintense lesions were found simultaneously on ADC maps. Concerning the patient with the cytotoxic edema, was the lesion confined to a vascular territory? How to explain the difference in the cerebral blood flow when comparing the central and peripheral perfusion of a SLL?

Since 9 patients had altogether 16 SLLs it can be assumed that some patients had more than a single SLL at the time of the MRI investigation. Did patients with more than a single SLL also manifest phenotypically with more than a single SLE? Were MRI characteristics different between the multiple SLLs?

Since MELAS may phenotypically mimic other disorders1,3–5, we should be informed how many were truly diagnosed upon a genetic test and how many solely upon muscle biopsy findings?

It is also important to know if patients experiencing a SLE received treatment or not, since drugs may strongly influence the morphological presentation on MRI. We thus should be informed how many of the 9 patients received NO-precursors (L-arginine, L-citrulline) or antiepileptic drugs (AEDs). SLEs are frequently associated with seizures and some of the AEDs may be strongly mitochondrion-toxic, thus influencing the pathophysiological cerebral conditions.

In summary, the study would profit from additional evaluation and provision of supplementary data with regard to time point of MRI, treatment, and dynamics of the MRI lesions described.

References
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