



Peculiarities of stroke-like lesions on MRI

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With interest we read the review article by Koksel et al. about diffusion weighted imaging (DWI) findings in patients with acute encephalopathy, tagged with the acronym “CRUMPLED” [1]. The authors discuss DWI findings in Creutzfeldt Jacob disease (CJD), reversible cerebral vasoconstriction syndrome (RCVS), urea cycle disorders manifesting with hyperammonemia, mitochondrial encephalopathy with stroke-like episodes (SLEs), prolonged seizures, and posterior reversible encephalopathy syndrome (PRES) [1]. We have the following comments and concerns.

We do not agree with the statement that MELAS is the most prevalent mitochondrial disorder (MID) [1]. The most common of the MIDs are the mitochondrial multiorgan disorder syndromes (MIMODS), which do not fit to one of the > 50 mitochondrial syndromes tagged with an acronym. MIDs are generally highly prevalent conditions but frequently they go undetected because they are overlooked. It should be clarified if the authors mean that MELAS is the most prevalent among the MIDs that are recognised in the neuroimaging literature when SLLs are focused.

We also do not agree with the statement that stroke-like lesions (SLLs), the morphological equivalent of a SLE, occurring in about 70% of the patients with MELAS, particularly occur in an occipitotemporal distribution [1]. SLLs occur ubiquitously in the central nervous system including the spinal cord [2], and even the optic nerves [3]. Characteristic of SLLs is, that they are not confined to a vascular territory, that they are dynamic with regard to extension and morphology, that an acute and chronic stage can be delineated, and that the acute stage is characterised by hyperperfusion and by reduced oxygen extraction fraction on the new MR sequence oxygen extraction fraction (OEF) [4].

Concerning the discrepancy between different reports about the presence of hypo- or hyperintense ADC maps in the acute stage of a SLL, it needs to be discussed that there is also mitochondrial vasculopathy affecting large and small arteries [5]. In the large arteries mitochondrial vasculopathy may manifest as atherosclerosis, dissection, ectasia formation, spontaneous rupture; or aneurysm for-

mation. In the small arteries mitochondrial vasculopathy may manifest as leukoencephalopathy, migraine-like headache, peripheral retinopathy in Leber's hereditary optic neuropathy (LHON), or SLEs. The latter can be explained by the fact that the metabolic defect may not only affect neurons or glial cells, but also endothelial cells or vascular smooth muscle cells. Mitochondrial vasculopathy is also evident in skeletal muscles showing up as hyper-reactive succinate dehydrogenase (SDH) positive cells [5]. A similar mechanism could be responsible for the cytotoxic components within SLLs. Thus, two different pathomechanisms, present in the same cerebral area, could be responsible for the mixture of cytotoxic and vasogenic edema.

In summary, it is essential to differentiate between an acute and chronic stage of a SLL and to consider not only a single pathomechanism to explain the imaging phenomena on MRI. Furthermore, additional techniques currently available such as perfusion weighted imaging (PWI), MR OEF, or single photon emission computed tomography (SPECT) might be useful to comprehend intrinsic mechanisms of some SLLs, supporting imaging diagnosis of MIDs in this setting, particularly when MELAS is suspected, and helping us to distinguish them from other encephalopathies.

Conflicts of interest

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References

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