Letter to the Editor

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Acute Diffusion MRI Findings in Metabolic Encephalopathies are Diverse

Josef Finsterer[1, 2], Fulvio A Scorza[2]

[1]Department of Neurology, Neurology & Neurophysiology Center, Vienna, Austria; [2]Disciplina de Neurociência, Universidade Federal de São Paulo/Escola Paulista de Medicina (UNIFESP/EPM), São Paulo, Brasil

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We read with interest the excellent review article by Jeon et al. [1] about the multimodal diffusion-weighted brain magnetic resonance imaging (MRI) findings of acute cerebral lesions in metabolic encephalopathies. Metabolic encephalopathies included hyper- and hypoglycemia, hyper- and hyponatremia, uremia, hyperammonemia, and vitamin B_1 deficiency [1]. Acute metabolic encephalopathies were usually found to share bilaterally symmetrical imaging abnormalities and manifest as vasogenic or cytotoxic edema, both of which can be reversible [1]. The study is appealing but raises the following questions and concerns.

Diffusion-weighted imaging (DWI) abnormalities of disturbances in the oxidative metabolism are missing in the review. Impaired mitochondrial energy production frequently manifests in the brain, resulting in either clinical or imaging abnormalities. Although most mitochondrial disorders (MIDs) have a genetic background, some of them are acquired. Hence, they fall within the scope of the index review. Due to the genotypic and phenotypic heterogeneity of MIDs [2], clinical and cerebral imaging abnormalities are highly heterogeneous. However, various patterns of acute imaging abnormalities can be extracted from these variable presentations.

The most specific pattern on imaging is that of a stroke-like lesion (SLL), which partially resembles ischemic stroke with respect to the clinical presentation but is completely different with respect to the imaging findings [3]. SLLs are dynamic lesions that frequently originate from the cortex and expand to the subcortical white matter in a non-vascular distribution. DWI of SLLs, which frequently occur in a temporo-occipital distribution, shows a homogeneous hyperintensity, which can be hyper-, iso-, or hypointense in a homogeneous or inhomogeneous distribution on apparent diffusion coefficient (ADC) maps [3]. On perfusion weighted imaging, these lesions present as hyperintensity [4].

Another acute acquired encephalopathy not considered in the review is Hashimoto's encephalopathy, also known as steroid-responsive encephalopathy, which most frequently occurs in association with autoimmune thyroiditis [5]. Cerebral MRI may show hyperintense signal along the gyri and sulci with diffuse leptomeningeal enhancement bilaterally [6]. Since hypothyroidism can accompany small vessel disease, DWI may show acute lacunar ischemic lesions [7].

As hyperthyreose encephalopathy can be associated with venous sinus thrombosis (VST) [8], it is crucial to consider ischemic stroke with its typical DWI/ADC characteristics in the acute stage.

Acute metabolic conditions may not only be acquired but may also be hereditary. Except for thiamine deficiency, all other conditions included in the review have been reported in association with hereditary conditions. Therefore, we should know if there is a difference in the DWI pattern between hereditary and acquired encephalopathy. We should also be informed on how hereditary causes of the presented metabolic disorders were excluded. Was the family history truly negative for all cases whose images were presented and were genetic causes truly excluded upon thorough genetic investigations?

Overall, the interesting review has limitations that challenge the results and their interpretation. A discussion about the imaging findings of SLLs in acquired MIDs, small vessel disease in hypothyroidism, and VST in

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Corresponding author: Josef Finsterer, MD, PhD, Department of Neurology, Neurology & Neurophysiology Center, Postfach 20, 1180 Vienna, Austria.
• E-mail: fifigs1@yahoo.de

hyperthyroidism is missing.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

ORCID iDs

Josef Finsterer
https://orcid.org/0000-0003-2839-7305
Fulvio A Scorza
https://orcid.org/0000-0002-0694-8674

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