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Letter to the Editor

Stroke Mimics in Glut-1 Deficiency Syndrome should not be Confused with Stroke-Like Episodes in Mitochondrial Disorders

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We were interested to read the article by Olivotto *et al*. on a retrospective study of eight patients aged 6-38 years with glucose transporter 1 deficiency (Glut1-DS) who developed acute neurological deficits [1]. Six of the eight patients had a stroke or "stroke-like episode" (SLE), of which cerebral imaging demonstrated a structural lesion in only three patients (patient-1, patient-2, patient-3) [1]. Acute ischemic stroke was diagnosed on imaging in only one patient (patient-3) [1]. The clinical symptoms of the eight patients included dysarthria/aplasia, oral dyskinesia, dysphagia, paresthesias, facial paralysis, hemi-/monoplegia, vomiting, headache and behavioural disturbances [1]. It was concluded that SLEs occur in 19% of Glut-1-DS patients and that stroke mimics should be considered the main feature of Glut1-DS [1]. The study is impressive, but several points need to be discussed.

The first point is that the term SLE has been used misleadingly [1]. SLEs are a hallmark of mitochondrial disorders (MIDs) and are pathognomonic for mitochondrial encephalopathy, lactic acidosis and stroke-like episodes (MELAS) syndrome [2]. SLEs in MIDs present with seizures, headaches, visual disturbances, hemiparesis, aphasia, vomiting and psychiatric abnormalities [2]. The morphologic correlate of SLE in cerebral imaging is the stroke-like lesion (SLL), which shows typical features in cerebral MRI, magnetic resonance spectroscopy and fluorodeoxy-glucose positron emission tomography (FDG)-PET [3]. In MRI, SLLs show up as hyperintensity on T2/FLAIR, DWI and PWI and as hypointensity on OEF [3]. On MRS, SLLs show a lactate peak and a reduced N-acetyl-aspartate (NAA) peak [3]. In FDG-PET, SLLs show hypometabolism [3].

The second issue is the discrepancy between the statement in the abstract that two patients had ischemic lesions and Table 1, which describes an ischemic lesion on MRI in only one patient (Patiemt-3) [1]. We should know which other patient showed cytotoxic oedema on MRI. Venous congestion was described in patient-1 and gliosis and atrophy of the right cerebellum in patient-2 [1]. Magnetic resonance angiography (MRA) showed left hemispheric hypoperfusion in patient-2 and normal MRA in patient-3 [1]. Who is the second patient with an ischemic lesion?

The third point is that the stroke-like event in patient-4 should be reclassified as a transient ischemic attack (TIA). According to Table 1, the aphasia and right hemiplegia lasted no longer than 24 hours and the cerebral CT was normal [1]. This constellation fulfils the criteria for a TIA.

The fourth point is that the acute stroke-like events apparently occurred during the pandemic. Since SARS-CoV-2 infections can be complicated by stroke, haemorrhage and venous sinus thrombosis [4], it would be imperative to report whether any of the six patients with stroke-like events had COVID-19 during the onset of the stroke-like event.

In conclusion, it can be said that this interesting study has limitations that relativize the results and their interpretation. Addressing these limitations could strengthen the conclusions and reinforce the message of the study. The term SLE should preferably be used for MID patients with a corresponding clinical picture who show a typical SLL on cerebral imaging. Stroke-like manifestations in other patients should be referred to as transient ischemic attack (TIA) or stroke mimic.

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