Consider Stroke-Like Episodes as a Differential of Migrainous Stroke

Josef Finsterer¹

¹Messerli Institute, Krankenanstalt Rudolfstiftung, Vienna, Austria

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With interest, we read the article by Sharma et al about a patient with a cortical stroke manifesting as dysarthria, hemihypesthesia, and hemiparesis, shortly after onset of a migraine attack with visual aura.¹ The patient also experienced homolateral facial cloni for <1 minute once.¹ The ischemic lesion ended up as laminar cortical necrosis (LCN).¹ The study has several shortcomings.

The lesion shown in **\sim Fig. 1** in the article by Sharma et al¹ does not comply with the clinical presentation. The patient presented with dysarthria, hemihypesthesia, and hemiparesis. A lesion in the superior parietal lobe does not necessarily explain the clinical presentation. The discrepancy between clinical presentation and imaging should be explained.

Missing are the serum lactate values and the results of the magnetic resonance (MR) spectroscopy (MRS). Since migraine can be a feature of a mitochondrial disorder (MID),² we should know if serum lactate or cerebrospinal fluid (CSF) lactate were elevated. CSF lactate can be measured either directly by determination in the CSF or by MRS.

The list of causes of LCN is incomplete. LCN may not only occur in association with cortical hypoxia, metabolic disturbances, or in association with drugs or infections but also due to seizures, venous thrombosis, or due to a stroke-like episode (SLE). SLEs are the hallmark of MELAS (mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes) syndrome and their correlate on imaging is the stroke-like lesion (SLL). SLLs are at variance from ischemic stroke with regard to clinical manifestations, presentation on imaging, dynamics, treatment, and outcome. SLLs may evolve into LCN, a white matter lesion, atrophy, a cyst, or the toenail sign as an end stage of the condition. SLLs need to be identified not to miss the point at which genetic work-up for MID should be initiated or at which appropriate treatment should be started.

Missing in the evaluation are long-term electrocardiogram (ECG) recordings, such as telemetry, Holter, or reveal implantation to confirm or exclude atrial fibrillation. Atrial fibrillation is associated with an increased risk of cardioembolic events. Documentation of atrial fibrillation is crucial as it may necessitate oral anticoagulation.

Missing are EEG recordings since the patient had cloni of the right facial muscles. No explanation for the cloni is provided, but a focal seizure needs to be excluded as seizures are a typical manifestation of a SLL.

A further shortcoming is that the MR imaging (MRI) was performed not earlier than 15 days after onset of the migraine attack. It is conceivable that the attack was a migraine accompanied (dysarthria, hemihypesthesia) and that hemiparesis which developed 14 days after onset of migraine was a completely different event. We should know after how many days the initial migraine attack resolved and if there was a further attack during the interval between initial attack and MRI. We also should know at which day the cloni of the left face occurred.

Missing is a description of the acute treatment for the stroke and the secondary prophylaxis. We should know if the patient received antithrombotic medication and a prophylaxis for migraine.

Missing is a detailed description of the cortical T2hyperintnsity on apparent diffusion coefficient (ADC). Knowing the ADC maps is crucial to differentiate if the lesion was truly ischemic in nature or if it presented as vasogenic edema.³

Missing is also the exclusion of a cerebral venous thrombosis. Nothing is reported about the D-dimer in the serum and about the MR venography. Since the patient complained of headache and a stroke, venous thrombosis needs to be excluded.

Overall, the report has several shortcomings which need to be addressed before final conclusions can be drawn. Due to the peculiarities and inconsistencies of the case, it is crucial to exclude SLLs as a differential of the cortical lesions on MRI.

Address for correspondence Josef Finsterer, MD, PhD, MesserliInstitute, Krankenanstalt Rudolfstiftung, Postfach 20, 1180 Vienna, Austria (e-mail: fifigs1@yahoo.de). DOI https://doi.org/ 10.1055/s-0040-1710136 ISSN 0976-3147. ©2020 Association for Helping Neurosurgical Sick People License terms



Author's Contribution

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None declared.

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