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Letter to the Editor

Before treating a stroke it must also be proven

¹Josef Finsterer, ²Sounira Mehri

¹Neurology & Neurophysiology Center, Vienna, Austria

²Biochemistry Laboratory, LR12ES05 "Nutrition-Functional Foods and Vascular Health", Faculty of Medicine, Monastir, Tunisia

Corresponding Author: **Josef Finsterer**

We read with interest the article by Kong *et al.* on a 75-years-old male with a syncope followed by dizziness and dysarthria since three days ^[1]. His history was positive for arterial hypertension, diabetes, renal insufficiency, and a pontine stroke 16 years earlier with complete recovery ^[1]. On examination there were square-wave jerks (SWJs), right-beating nystagmus on head shaking; exophoria, hypometric saccades; impaired convergence, truncal and limb ataxia, and inability to stand or walk ^[1]. MRI showed bilateral lesions in the superior cerebellar peduncle being interpreted as ischemic stroke ^[1]. The study is excellent but has limitations that raise concerns and should be discussed.

The main limitation of the study is that the cause and nature of the bilateral superior cerebellar peduncular lesions were not clarified. Although hyperintensity of the lesions on fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI) is compatible with a subacute cytotoxic lesion due to ischemia, this suspicion needs to be confirmed. Missing in this respect are the apparent diffusion coefficient (ADC) maps, magnetic resonance angiography (MRA) or computed tomography angiography (CTA), and susceptibility-weighted imaging (SWI) images. In case the lesions truly correspond to a cytotoxic edema, they should be hypointense on ADC. If the lesion resulted from bleeding, the SWI images should be hypointense. Angiography is particularly important as the patient had a history of previous pontine stroke 16 years before. To rule out an inflammatory process, it is mandatory to apply contrast medium.

Because the patient experienced a syncope three days prior to admission, it is also mandatory to record an electroencephalogram (EEG) and to perform a carotid ultrasound. Symptomatic seizures due to the old pontine stroke are conceivable.

Assuming that the peduncular lesions are ischemic in nature, it is mandatory to rule out cardioembolism and endocarditis not only by transthoracic but also by transesophageal echocardiography. In particular, we should know if the history was positive for paroxysmal atrial fibrillation or heart failure. To assess if there was subclinical atrial fibrillation long-time ECG recordings by means of reveal recorder over months are warranted. The available transthoracic echocardiography should be revised for systolic dysfunction, Takotsubo syndrome, cardiomyopathy, and non-compaction. All these conditions can potentially go along with intra-cardiac thrombus formation and are thus critical for assessing the thromboembolic risk.

Another limitation of the study is that the long-term outcome of the index patient was not reported in detail. We should know what symptoms and signs resolved after three and six months, and whether the peduncular hyperintensities completely resolved on cerebral imaging.

Overall, the interesting study has limitations that call the results and their interpretation into question. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Before treating a stroke it should also be proven.

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