## Letters to the Editor

# **Consider Stroke-Like Episodes as a Differential in Children with Acute Hemiparesis**

### Sir,

We read with interest the article by Chinnabhandar *et al.* about a study of the causes and imaging findings in 55 children aged 3–14 years admitted for acute hemiparesis.<sup>[1]</sup> Interestingly, the most common cause of hemiparesis was central nervous system infection.<sup>[1]</sup> We have the following comments and concerns.

A shortcoming of the study is that the cause of hemiparesis could not be detected in 10 of the 55 patients.<sup>[1]</sup> Thus, we should be informed about blood, cerebrospinal fluid (CSF), and imaging findings in these ten patients. How many had a normal cerebral magnetic resonance imaging (MRI) and in how many was the cerebral MRI abnormal? Which differential diagnoses were considered in these ten patients? Differential diagnoses that were not considered are postictal hemiparesis and stroke-like episodes (SLE). Thus, it should be reported how many of the ten patients with hemiparesis of unknown cause had epilepsy or clinical or laboratory indications for seizures? We would like to know the results of electroencephalogram recordings and whether creatine kinase was elevated on admission in these ten patients. SLEs most commonly occur in patients with Mitochondrial encephalomyopathy, lactic acidosis and stroke-like episodes syndrome, but other specific mitochondrial disorders, such as Leigh syndrome, Kearns-Sayre syndrome, myoclonic epilepsy with ragged red fibers, chronic progressive external ophthalmoplegia, Leber's hereditary optic neuropathy, mitochondrial recessive ataxia syndrome, SLSJCOD, POLG1-related mitochondrial disorder, or mitochondrial multiorgan disorder syndrome, may also manifest with SLEs. Thus, we should know if cerebral MRIs in the ten patients with unknown cause were indicative of a stroke-like lesion (SLL) the morphological equivalent of a SLE. SLLs show up on cerebral MRI as hyperintensity on diffusion-weighted imaging and as hyperintensity on apparent diffusion coefficient maps (vasogenic edema) in the acute stage of a SLE and are not confined to a particular vascular territory.<sup>[2]</sup> SLLs may undergo dynamic changes over days or weeks, ending up as completely normal MRI, white-matter lesions, recurrence of the SLL, toe-nail sign, laminar cortical necrosis, or as cysts.<sup>[3]</sup> It could be helpful to apply MR spectroscopy or fludeoxyglucose-positron emission tomography to the ten patients with hemiparesis of unknown cause, to find if there was lactate elevation in the CSF or hyper- or hypo-metabolism or not.

To identify the cause of hemiparesis in the ten patients with hemiparesis of unknown cause, it could be helpful to follow-up these patients for a longer period of time, to find if they develop migraine, carcinosis, heart failure, or arrhythmias.<sup>[4]</sup> The frequency of patients developing complicated migraine or systolic dysfunction or heart failure during follow-up should be reported. Supraventricular or ventricular arrhythmias were detected on long-term electrocardiogram recordings. Transitory ischemic attack due to cardio-embolism as well as arterio-cerebral embolism with a carotid or vertebral artery stenosis should be considered as a differential in these patients. Thus, we should be informed about the imaging of the extra- and intra-cranial arteries and if there were any indications for stenosis or occlusion.

The statement in the results that neuroimaging revealed cerebral infarction in 54.5% of the cases is discrepant to the findings reported in Table 3, in which hemiparesis was attributed to a vascular cause in only 22%.<sup>[1]</sup> This discrepancy requires an explanation. Was stroke in 54.5% of the cases attributable to a SLE in some of these cases?

Overall, this interesting study has several shortcomings, such as short follow-up period and no extensive workup particularly among those with hemiparesis of unknown cause. Furthermore, the differential diagnoses excluded and the criteria for exclusion should be mentioned. Acute hemiparesis in children should not remain unexplained.

## Financial support and sponsorship

Nil.

### **Conflicts of interest**

There are no conflicts of interest.

Josef Finsterer

KAR, Messerli Institute, Veterinary University of Vienna, Austria

Address for correspondence: Dr. Josef Finsterer, Finsterer J, Postfach 20, 1180 Vienna, Austria. E-mail: fifigs1@yahoo.de

## References

- Chinnabhandar V, Singh A, Mandal A, Parmar BJ. Acute hemiplegia in children: A Prospective study of etiology, clinical presentation, and outcome from Western India. J Neurosci Rural Pract 2018;9:504-9.
- Kim JH, Lim MK, Jeon TY, Rha JH, Eo H, Yoo SY, et al. Diffusion and perfusion characteristics of MELAS (mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episode) in thirteen patients. Korean J Radiol 2011;12:15-24.
- 3. Finsterer J. Stroke and stroke-like episodes in muscle disease.

Open Neurol J 2012;6:26-36.

4. Eom S, Lee HN, Lee S, Kang HC, Lee JS, Kim HD, *et al.* Cause of death in children with mitochondrial diseases. Pediatr Neurol 2017;66:82-8.



This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

© 2019 Journal of Neurosciences in Rural Practice | Published by Wolters Kluwer - Medknow