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【 LETTERS TO THE EDITOR 】

Cerebellar Stroke-like Lesions?

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To the Editor We read with interest the article by Muramatsu et al. about a 24-year-old female with mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes (MELAS) due to the mtDNA variant m.3243A>G, who developed stroke-like lesions (SLLs) in the cerebellum, which spontaneously disappeared at 6 months after onset (1). We have the following comments and concerns.

MELAS is usually a multisystem disease affecting not only the brain but also the muscles, ears, eyes, endocrine organs, heart, kidneys, and bone marrow (2). Thus, we should determine which other organs were affected and what manifestations developed within an affected organ.

MELAS is frequently associated with lactic acidosis in the serum/cerebrospinal fluid (CSF). Thus, we should know if the serum lactate level was elevated and if such an elevated lactate level was also detected upon direct investigation of the CSF or by MR-spectroscopy.

SLLs usually show up on multimodal MRI as vasogenic edema. Thus, we should be informed as to whether the cerebellar lesions were hyper- or hypointense on ADC maps. To confirm the nature of the lesions it would be helpful to apply oxygen extraction fraction MRI (3). This technique may show that oxygen extraction is significantly reduced within the SLL area.

Since the three main hypotheses to explain SLLs are the epileptogenic, metabolic, and vasogenic hypotheses, we should be informed if the development of the SLL was associated with seizure-activity on electroencephalogram (EEG) or if the seizures were reported prior to the onset of the SLL or during the acute stage of the SLL.

Since heteroplasmy rates may determine the phenotype of MELAS patients, we should know the heteroplasmy rates in hair follicles, buccal mucosa cells, skin fibroblasts, muscle, lymphocytes, and urinary epithelial cells.

Looking at the cerebellar lesions in the figure presented by Muramatsu, one would expect not only nausea, but also vertigo, nystagmus, dysarthria, and ataxia. Did the patient present with any other typical manifestations of a cerebellar lesion?

Since mtDNA variants are inherited via a maternal trait in 75% of the cases (4), we should be informed about the family history, particularly if the mother was genotypically or phenotypically affected.

Since SLLs are frequently treated with NO-precursors, antiepileptic drugs, steroids, or antioxidants (CoQ, edaravone, idebenone) (5), we should know if the described patient received any treatment for the SLL, for how long, and at what dosages.

Overall, this interesting article could be more meaningful, if more basic information concerning the individual and family history, multisystem involvement, and treatment had been provided. There is also a need to discuss in more detail the cause and pathophysiology of the described cerebellar lesions.

The author states that he has no Conflict of Interest (COI).

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