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Letter to the Editor

Cerebellar stroke-like lesions in Leigh syndrome may mimic cerebellar cortical bleeding

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Letter to the Editor

With interest we read the article by da Vega et al. about an 8yo male with Leigh syndrome due to the variant m.8993T > C in *MT-ATP6* [1]. The patient manifested phenotypically initially with poor language skills, incoordination, frequent falls, next with developmental regression, orofacial dyskinesia, muscle wasting, dysarthria, positive Babinski sign, and later with chorea, ataxia, and kinetic tremor and progressive disability [1]. There was lactic acidosis and cerebral MRI revealed T2-hyperintensities in the lenticular nucleus and the tectum, cerebellar swelling, and cerebellar cortical bleeding. The study has a number of shortcomings.

How can the authors be sure from the MRI that the cerebellar lesion represents blood? We should know if the cerebellar lesion was also hyperdens on cerebral CT. Furthermore, we should know if cerebrospinal fluid (CSF) investigations were indicative of acute or chronic bleeding (e.g. erythrocytosis, elevated bilirubin, xanthochromatous CSF). CSF investigations at age 8y only revealed lactic acidosis but were otherwise normal.

Since stroke-like lesions have been recently reported to occur also in the cerebellum [2,3], we should know if the symmetric cerebellar lesions represent symmetric stroke-like lesions. Bilaterally symmetric stroke-like lesions are increasingly recognised [4] and it is increasingly acknowledged that stroke-like lesions start in the cortex to spread spontaneously to more subcortical regions [4].

A further argument against cortical cerebellar bleeding is that the cerebellar lesion on cerebral MRI-2 remained unchanged 6 months later on cerebral MRI. This is an unusual behaviour of cerebral bleeding.

The pattern on multimodal MRI in the cerebellum may also represent fat-laden macrophages, which started to phagocyte debris from the nearby sroke-like lesion.

The authors hypothesise that cerebellar bleeding resulted from microangiopathy [1]. Though involvement of arteries is a commonly encountered feature in an increasing number of mitochondrial disorders (MIDs), particularly MELAS [5], there is no convincing feature on MRI that confirms the suspicion of microangiopathy. In this regards we should be informed if a conventional cerebral angiography or a CT-angiography was carried out to assess if cerebral arteries were impaired in Leigh syndrome as well.

There is a discrepancy between the description of the MRI at age 8y described with T2-hyperintens lenticular lesion and the statement “a prominent basal ganglia T2 hyperintensity, paradigmatic for Leigh syndrome, was not seen in this patient” [1]. The readership should be informed if typical symmetric T2-hyperines basal ganglia could have been found in the index patients or not.

Overall, this interesting case report has a number of shortcomings, which need to be addressed before final conclusions can be drawn. It should be stressed that stroke-like lesions may also occur in the cerebellum and may mimic ischemic stroke or cortical bleeding.

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Declaration of Competing Interest

There are no conflicts of interest.

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