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Letter to the Editor

Do Not Diagnose Thiamine Deficiency Solely on the Basis of Clinical Findings and Brain Imaging; Also Demonstrate Low Serum Thiamine Levels

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Letter to the Editor

We read with interest the article by Agaoglu *et al.* regarding two cancer patients who developed Wernicke encephalopathy due to malnutrition and recovered almost completely following thiamine supplementation ^[1]. The study is noteworthy but warrants further discussion.

A primary point is that serum vitamin B1 levels were not measured to confirm an actual serum thiamine deficiency ^[1]. A diagnosis of a thiamine-deficiency-related disorder should not be made without evidence of low serum thiamine levels. Without knowledge of the actual serum thiamine level, all differential diagnoses for bilateral thalamic hyperintensities on diffusion-weighted imaging (DWI) must be considered and carefully ruled out. Differential diagnoses for bilateral thalamic DWI hyperintensities include artery of Percheron infarction, cerebral venous sinus thrombosis, cerebral hypoxia, Leigh syndrome and other mitochondrial disorders involving the CNS, extrapontine myelinolysis, infectious encephalitis, autoimmune encephalitis, acute disseminated encephalomyelitis, Creutzfeldt-Jakob disease, Wilson's disease, vasculitis, and bilateral gliomas ^[2]. Furthermore, the possibility that bilateral thalamic DWI hyperintensities could be attributed to chemotherapy-induced leukoencephalopathy, for instance, following the administration of 5-fluorouracil, capecitabine, or high-dose methotrexate, should be considered ^[3]. Were all these differential diagnoses thoroughly ruled out?

A second point is that the cerebral imaging findings are inadequately described ^[1]. In particular, it needs to be clarified whether the DWI hyperintensities were attributable to cytotoxic or vasogenic edema. Did the corresponding ADC sequences show hypo-, iso-, or hyperintensity? Were the DWI hyperintensities in the thalamus, mammillary bodies, and tectal plate also visible on T2/FLAIR sequences, and did they show contrast enhancement on T1-weighted images? In addition to the described MRI changes, thiamine deficiency can also manifest in the periaqueductal gray matter and on the floor of the fourth ventricle ^[4]. In this regard, it should be noted that the hyperintensities do not occur within the ventricles (third and fourth ventricles) themselves, as mentioned in the discussion ^[1], but rather in the periventricular tissue ^[4]. Furthermore, information regarding the results of magnetic resonance angiography and magnetic resonance spectroscopy is missing.

A third point concerns the clinical presentation of thiamine deficiency: it manifests not only through ocular motility disorders, signs of malnutrition, cerebellar dysfunction, and altered consciousness, but can also be accompanied by fatigue, anorexia, weight loss, irritability, depression, abdominal discomfort, nausea, neuropathy, muscle weakness, muscle cramps, diminished tendon reflexes, heart failure, tachycardia, increased pulse amplitude, dyspnea, leg edema, and psychosis ^[5]. Were any of these additional symptoms present in either of the two patients?

In summary, the diagnosis of thiamine deficiency should not be based solely on the clinical picture and cerebral imaging but must be confirmed by demonstrating reduced serum thiamine levels.

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