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Letter to the Editor

Alcohol abuse is more likely than vitamin-B6 deficiency as a cause of new-onset seizures

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Of interest is the article by Nathan et al. on a 76-year-old man with new-onset symptomatic seizures attributed to vitamin-B6 deficiency presumably caused by long-term treatment with levodopa/carbidopa (1250mg/250mg) for Parkinson's disease (PD) [1]. The seizures were classified as refractory as levetiracetam, lacosamide, clobazam and clonazepam were only partially effective [1]. The seizures did not stop earlier than after vitamin B6 supplementation [1]. Some ambiguities should be clarified.

Firstly, chronic alcohol abuse was not sufficiently ruled out as a cause of the seizures. Since the patient regularly consumed 2-3 drinks per day and alcohol is one of the most common triggers of seizures, it is likely that the alcohol abuse and not the vitamin B6-deficiency triggered the seizures. Was serum alcohol measured on admission? Were there stigmata of chronic alcoholism such as elevated MCV, elevated GGT or vitamin B12 deficiency? Is it conceivable that the seizures stopped during hospitalization because the patient was no longer allowed to drink? Is it conceivable that the PD worsened 6 months prior to admission due to alcohol abuse? Alcohol is known to aggravate PD [2]. It should have been mentioned in the case description that the patient drank alcohol regularly.

The second point is that no explanation was given for the leukocytosis [1]. Was the leukocytosis due to infection, nicotine abuse or a residual sign of chronic lymphocytic leukemia (CLL)? Were the cells examined for malignancy, particularly CLL? Was there any other evidence of CLL relapse? What was the reason for the patient's loss of appetite for four months prior to the onset of seizures? Has the leukocytosis regressed during the follow-up?

The third point is that serum vitamin-B6 was measured only once [1]. There is no serum vitamin-B6 level before the onset of seizures or during follow-up [1]. Since vitamin-B6 deficiency was blamed for the seizures, it would have been crucial to measure vitamin-B6 repeatedly, also to assess whether the supplementation was sufficient or not.

The fourth issue is that it was not reported whether levodopa/carbidopa therapy was continued, reduced or discontinued [1]. Since it has been suggested that carbidopa/levodopa is responsible for the vitamin-B6 deficiency, it is important to know how the PD was treated after the attack, in particular whether levodopa/carbidopa was reduced, discontinued or replaced.

The fifth point is that sildenafil was not considered as a trigger of seizures. For what indication and in what dosage did the patient take sildenafil and how often? Sildenafil is known to trigger seizures in rare cases [3]. It was also not stated why the patient was taking

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calcium [1]. Was the calcium level normal on admission. Since hypocalcemia can trigger seizures [4], it is important to clarify whether this could be the cause of the seizures. It should also be clarified why the patient was taking thiamine. Has he been diagnosed with Wernicke's encephalopathy or Korsakov's syndrome? Was the thiamine deficiency due to chronic alcoholism?

Finally, we should know whether the cerebrospinal fluid (CSF) was examined for malignant cells, especially lymphoblasts, and for CSF lactate. Since the serum lactate was elevated on admission, it is conceivable that it was also elevated in the CSF. Has the serum lactate decreased during follow-up? It should also be stated whether the tests for HIV and syphilis were negative.

Overall, before attributing new onset seizures to vitamin-B6 deficiency, alternative and more common triggers of seizures must be thoroughly ruled out.

Declarations

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