Seizures Related to Vitamin B6 Deficiency in Adults

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Vitamin B6 is closely associated with functions of the nervous, immune, and endocrine systems. Its deficiency may result in neurological disorders including convulsions and epileptic encephalopathy. Until today, this has only been reported in infants, children, and critically ill adult patients. We report a case of a 36-year-old man with chronic alcoholism who presented with seizures after gastrointestinal disturbance. His seizures persisted even after treatment with antiepileptic drugs, but eventually disappeared after administration of pyridoxine. Hence, vitamin B6 deficiency may cause seizures in adult patients with chronic alcoholism. (2015;5:23-24)

Key words: Vitamin B6 deficiency, Seizure

Introduction

Vitamin B6 is part of the vitamin B group complex and can serve as a cofactor in the metabolism of glucose, amino acids, and lipids. Since it is essential for neurotransmitter synthesis, its deficiency causes various neurological disorders.1 Seizures associated with vitamin B6 deficiency are usually observed in pediatric patients and these are characterized by refractoriness to antiepileptic drugs and a positive response to pyridoxine administration.2 Here, we report a case of adult-onset seizures associated with vitamin B6 deficiency and discuss the pathophysiological mechanism.

Case

A previously healthy 36-year-old man visited our hospital because of recurrent loss of consciousness and convulsions. He had had poor nutritional intake, nausea, and vomiting for about 6 weeks. He was a heavy drinker with a reported alcohol intake of more than two bottles per day of Soju (Korean whiskey) for 10 years. Recently, he had reported drinking more than three bottles per day without meals. He continued to drink without abstinence.

Upon physical and neurological examination, no specific abnormalities were observed. Complete blood count revealed that his hemoglobin level was as low as 9.4 g/dL. Mean cell volume was slightly elevated (104.7 fl, normal value 80-100 fl), but the mean corpuscular hemoglobin concentration was normal (33.6 g/dL, normal value 32-36 g/dL). White blood cell and platelet counts were normal. Liver function tests revealed aspartate aminotransferase, alanine aminotransferase, and alkaline phosphatase levels of 59 IU/L (normal value 0-40 IU/L), 8 IU/L (normal value 0-40 IU/L), and 234 IU/L (normal value 42-128 IU/L), respectively. Chest X-ray radiographs and an electrocardiogram revealed no abnormalities. Brain magnetic resonance imaging and an electroencephalogram also showed no abnormalities.

The patient had a generalized tonic-clonic seizure in the emergency room prior to admission. The seizure ceased after administration of 4 mg of lorazepam and 1 g of valproate. After one day, another seizure developed and 800 mg of controlled-release carbamazepine was administered. Vitamin B6 levels were measured to be 4.4 nM/L (normal value: 20-202 nM/L), serum homocysteine levels

Table 1. Serum Laboratory test at admission

| Measure          | Normal range |
|------------------|--------------|
| Complete blood count |             |
| WBC count        | 4.91 x 10^3/μL | 4.0-10.0 |
| RBC count        | 2.68 x 10^12/μL | 4.0-6.0  |
| Hemoglobin       | 9.4 g/dL     | 13.0-18.0 |
| MCV              | 104.7 fL     | 80.0-100.0 |
| MCH              | 35.2 Pg      | 20.0-35.0 |
| MCHC             | 33.6 g/dL    | 32.0-36.0 |
| PLT              | 193 x 10^3/μL | 130.0-450.0 |
| Liver function test |           |
| AST              | 59 IU/L     | 0-40      |
| ALT              | 8 IU/L      | 0-40      |
| ALP              | 234 IU/L    | 42-128    |
| T-bilirubin      | 1.5 mg/dL   | 0.2-1.2   |

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were 65 μM/L (normal value 5.0 ~ 13.9 μM/L) and vitamin B1 level was normal (79.8 ng/mL).

The patient abstained from alcohol and had regular meals after admission. Pyridoxine (12 mg/day) and carbamazepine were administered for 2 months. Since discharge, the patient has been seizure-free for over 6 months. Follow-up vitamin B6 levels were also normalized (59.3 nM/L).

Discussion

Vitamin B6 is a water-soluble vitamin with the active form being pyridoxal 5′-phosphate (PLP).³ PLP is essential for the metabolism and synthesis of amino acids, gluconeogenesis, hematopoiesis, hormone regulation, immunologic functions, and synthesis of neurotransmitters.⁴ Importantly, PLP is a crucial cofactor in the synthesis of γ-aminobutyric acid (GABA) from glutamate because of its involvement in the mechanism of decarboxylation. Therefore, PLP deficiency results in the lowering of seizure thresholds by impairing the synthesis of GABA.⁵

In this case, Wernicke’s encephalopathy and hepatic encephalopathy must be differentiated. Our patient showed no physical or laboratory findings compatible with those disorder, i.e. profound alteration of mentality before and after seizures, no stigmata of chronic alcoholism and nutritional deficiencies who show refractoriness to antiepileptic drugs.

Our patient, who previously had recurrent seizures despite treatment with antiepileptic drugs, became seizure-free after normalization of vitamin B6 levels. We propose that a differential diagnosis for vitamin B6 deficiency needs to be considered in patients with chronic alcoholism and nutritional deficiencies who show refractoriness to antiepileptic drugs.

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