LUPINE PUBLISHERS

Online Journal of Neurology and Brain Disorders

DOI: 10.32474/OJNBD.2023.06.000244

ISSN: 2637-6628

Case Report

Vitamin B6 Induced Sensory Polyneuropathy: About Two Clinical Observations

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Received: November 16, 2022 **Published:** February 17, 2023

Summary

Polyneuropathy related hypovitaminosis B6 (pyridoxine) are well recognised. In contrast, the association between elevated serum levels of pyridoxine and neuropathy is less well described. We report a peripheral neuropathy with sensory disorders subsequent to a hypervitaminosis B6 (pyridoxine) in two female patients were seen the neurology consultation at the neuropsychiatric centre Dr Joseph Guislain in Lubumbashi (DRC). Also, in low- and middle-income countries hypervitaminosis B6 should be considered in the differential diagnosis of any sensory or sensorimotor polyneuropathy.

Introduction

Hypervitaminosis B6 is neurotoxic and can cause progressive sensory neuropathy [1].Vitamin B6 is widely used in nutritional supplements [2]. Vitamin B6, which is contained in many medications and nutritional supplements, can be responsible for severe neurological disorders in case of overdose [3,4]. We report the observation of two patients who, after taking vitamin B6 over a protracted period, presented with sensory polyneuropathy.

Case Report 1

A 17-year-old female patient consulted for paraesthesia of the whole body, since five months. At first, these symptoms were subacute and bearable. For these symptoms, she consulted a traditional healer and was recommended several 'medicines'. In the patient's history loss of appetite for several months was reported. She consumed a daily dietary supplement to stimulate her appetite. This product, which contained vitamin B6, had sometimes been prescribed by her physician; nonetheless she often also adopted it as self-medication. During the clinical examination, the patient presented with subjective generalized sensory disorder, with tingling paraesthesia in fingers and lower limbs accompanied by a feeling of cold feet. An electro-neuromyogram (ENMG) showed diffuse decrease in sensory responses (amplitudes and speeds) to

electrical stimulation in all four limbs. A vitamin assay in search of a vitamin deficiency revealed instead a hypervitaminosis B6 at 150 nmol/L for a normal value between 15 and 75 nmol/L . Haematological, renal, thyroid and blood glucose levels were within the norms. Discontinuation of pyridoxine (dietary supplement) was recommended. Within six months het clinical picture improved significantly to normalize ten months later. The results of the pyridoxine assay and the ENMG performed ten months after removing the dietary supplement became normal (vitamin B6 at 60 nmol/L).

Case Report 2

A 38-year-old female patient complained of sensations of heat and tingling in her hands and feet since. The intensity of the paraesthesia gradually worsened. She had no medical or surgical history. The medical history revealed regular use of a product containing pyridoxine as a dietary supplement. The ENMG was normal. On the other hand, serum vitamin B6 dosage demonstrated a hypervitaminosis B6 at 200 nmol/L. Haematological, renal, thyroid and blood glucose levels showed no abnormalities. The discontinuation of the pyridoxine-containing products consumed by the patient allowed the gradual decrease in the intensity of these paraesthesia. Vitamin B6 dosage at four months was normal (55 nmol/L). Thus, vitamin B6 has been implicated as the probable cause of this neuropathy.

Discussion

The first cases of pyridoxine induced neuropathy were reported in the 1980 [5]. The authors had reported seven patients who had been taking high doses of pyridoxine for several months, and who were developing severe sensory ataxia. Motor impairments were also reported. Other studies have demonstrated the toxicity of pyridoxine and particularly peripheral neuropathies [6]. Toxicity is dose-dependent, and the onset of symptoms usually appears after several months or even years of treatment. Discontinuation of pyridoxine usually improves symptoms, but residual abnormalities may persist. The mechanism of pyridoxine-induced toxicity remains unknown [7-9]. In our observation, the anamnesis, clinical and electrophysiological examinations confirmed the diagnosis of neuropathy, and the various complementary examinations made it possible to rule out another aetiology (diabetes, alcohol, hypothyroidism, renal failure, infections, etc.). In addition, the regression of neuropathy after discontinuation of vitamin B6 with normalization of blood pyridoxine levels and ENMG supports the hypothesis of drug-induced neuropathy related to pyridoxine. The daily dose of vitamin B6 taken by the patients and the exact duration of exposure could not be provided.

Conclusion

Excess pyridoxine intake is a possible aetiology in front of a picture of peripheral neuropathy. Clinicians should keep this potential toxicity in mind, especially considering vitamin supplements during the medical history taking. Clinicians should be encouraged to re-evaluate the indication for any vitamin supplementation. Because high-dosage vitamin B6 supplements are readily available for consumers, it is important consumers are made aware, by health authorities, pharmacists and physicians of the possible risks associated with the use of these products.

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