

Painful Leg Spasms in Patient with Hemifacial Spasms and Vitamin D and Vitamin B12 Deficiencies - A Case Report

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Abstract

Lower limb cramps and spasms underly a multitude of etiologies and are common complaints in the neurological outpatient clinic. Cramps including nocturnal cramp syndromes can often be refractory to therapy. Hence there is a need to appropriately investigate and identify potentially treatable causes of cramps and spasms such as vitamins B12 and D deficiencies. In this case report, we describe the resolution of lower limb spasms/cramping as well as improvement of hemifacial spasms following treatment for vitamins B12 and D deficiencies.

Keywords: *Vitamin B12 Deficiency; Vitamin D Deficiency; Cramps; Hemi-Facial Spasms*

Introduction

Spasms and cramps are common complaints and may underly multiple etiologies. Given that these can be difficult to manage or treat, it is important to identify treatable causes. Vitamin B12 deficiency can cause myelopathy, neuropathy and dementia, but it can cause more discrete neuromuscular manifestations including cramps [1].

Although primary hemifacial spasms are typically caused by vascular compression at the root entry zone of the facial nerve by a blood vessel in the posterior circulation, demyelination resulting from either vitamins B12 or D or combined deficiencies represents a secondary etiology. Ulusoy (2018) found lower serum vitamin D levels in HFS (hemifacial spasmic) patients than controls and hypothesized that vitamin D deficiency may trigger mechanism causing demyelination resulting in spasms [2]. Vitamin deficiencies such as vitamin D are common and can mimic musculoskeletal disorders including cramps [3-5].

Case Report

A 65-year-old female with a history of hemifacial spasms, type 2 diabetes mellitus, and lumbar stenosis (prior L4 to S1 decompression surgery) presented to the emergency department with an 8-months history of painful bilateral calf and thigh muscles spasms with a nocturnal preponderance. She was treated with diazepam and referred to the outpatient neurology clinic. Upon evaluation in the clinic, her examination was remarkable for frequent mild to moderate right upper and lower facial spasms. Gait was antalgic with palpable tenderness along the left lateral thigh. Her MRI brain and MR angiogram of the Circle of Willis and carotids showed a branch of the right MCA (middle cerebral artery) near the cisternal right facial nerve without definite contact. Her facial spasms had responded well to botulinum toxin injections. Laboratory investigations revealed vitamin B12 (212 pmol/L) and D (19 nmol/L) deficiencies as well as increased serum parathormone (89 ng/L) but normal serum, phosphate, and ionized calcium levels.

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Full resolution of the lower limb spasms occurred within 4 weeks of initiation of vitamin B12 1000 mcg daily with normalization of serum levels to 625 pmol/L at 6 weeks. Vitamin D3 supplementation was subsequently initiated with 50,000 units once weekly for 8 weeks and thereafter switched to 2000 units daily. Serum PTH (parathyroid hormone) and vitamin D levels normalized to 44 ng/L and 106 nmol/L, respectively. Although repeat reassessments at 6 and 8 weeks did not reveal any noticeable change in severity of hemifacial spasms, a significant improvement in severity from previously moderate to mild both clinically and with the patient's subjective report at 6 months following the start of vitamin D therapy/supplementation. This resulted in a reduction in her botulinum toxin dose at the 6-months post initiation of B12 supplementation.

Discussion

This case report illustrates specific clinical features and neurological manifestations associated with vitamin B12 and vitamin D deficiencies. It is known that vitamin B12 deficiency can manifest with a wide range of symptoms including paresthesia, cognitive impairment, sensory ataxia, cramps, and neuropsychiatric symptoms such as depression¹. This case report illustrates the role of vitamin B12 deficiency as a treatable cause of cramps. In addition, our patient had a concomitant vitamin D deficiency which on its own is another cause of muscle cramping, chronic pain, and other neurological complications [4]. The noted interval improvement in hemifacial spasms is further supportive of the potential role of vitamin D deficiency in hemifacial spasms². A malabsorptive etiology for the dual vitamin deficiency was presumed but a more definitive etiology is yet to be established in this patient [5].

Conclusion

It is important to consider vitamin deficiencies especially vitamins B12 and D in patients presenting with limb spasms and cramps. Low-normal serum vitamin B12 levels should also be considered for supplementation in this setting. These are two treatable and reversible etiologies compared with other cramping syndromes which are often refractory to therapy. Further studies looking at severity of hemifacial spasms and serum vitamin D levels are needed to establish any causal relationship that might be of therapeutic relevance.

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