Vitamin B12 Deficiency as a First Sign of Acquired Horizontal Pendular Nystagmus

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Rec date: Jan 21, 2015; Acc date: Feb 26, 2015; Pub date: Feb 28, 2015

Abstract
Eye movement disorders are rarely reported in vitamin B12 deficiency. Reported eye movement disorders in vitamin B12 deficiency were downbeat nystagmus and INO in 3 cases each, upward gaze palsy in 2 cases, bilateral abducens palsy, total ophthalmoplegia, bilateral horizontal gaze evoked nystagmus in 1 case each. We describe a case with recently developed horizontal pendular nystagmus due to vitamin B12 deficiency. He received replacement therapy but his eye movement disorder did not respond to treatment. The early diagnosis of vitamin B12 deficiency is vital because when axonal damage is started recovery might not occur. To the best of our knowledge, this is the first reported case with acquired horizontal pendular nystagmus caused by vitamin B12 deficiency.

Introduction
Vitamin B12 deficiency (B12D) causes numerous hematological, psychiatric, ophthalmic and neurological abnormalities [1]. In contrast to frequent optic nerve involvement, eye movement disorders in B12D, which is extremely rare, has only been reported 11 times in the literature [2–10]. We aim to present a case with conjugate horizontal pendular nystagmus due to vitamin B12 deficiency.

Case Report
A 17 year-old male patient presented with complaints of bilateral horizontal constant pendular nystagmus appearing 14 months ago (Video). He did not have any other ocular or systemic complaints. None of the other neurologic signs and symptoms was found in the patient. There was no history of drug use. Nystagmus in both eyes is symmetrical and has the same amplitudes in different directions in each eye. Eye movements to the right increased the frequency of the nystagmus in each eye. No instability occurred in any positions of the body and head. His best-corrected visual acuity (BCVA) was 20/20 in both eyes. Cranial, orbital MRIs and MRI of the temporal bone were normal. Videonystagmography was also normal. There was not any history of inadequate diet or alcohol use. Serum vitamin B12 level (VB12) was 111 pg/ml (214–864). Other blood tests were normal. He received VB12 1 mg i.m. daily for 10 days and the monthly, but the nystagmus did not improve over 6 months follow up.

Discussion
Reported eye movement disorders in vitamin B12 deficiency can be seen in Table 1. To the best of our knowledge, our case is the first reported case with acquired horizontal pendular nystagmus caused by vitamin B12 deficiency.

<table>
<thead>
<tr>
<th>Year</th>
<th>Age</th>
<th>Eye movement disorders</th>
<th>Response to therapy</th>
<th>MRI findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1986[5]</td>
<td>69</td>
<td>Downbeat nystagmus</td>
<td>Mild improvement</td>
<td>Mild cerebellar atrophy</td>
</tr>
</tbody>
</table>
In the 11 previously reported patients with eye movement disorder and vitamin B12 deficiency, the response to therapy was variable. Five cases responded completely (n=2) or partially (n=3) to VB12 therapy (46%), 3 cases did not (27%). In the other 3 cases there was no mention of response to treatment. In our case, conjugate horizontal pendulernystagmus did not improve over 6 months of follow-up. One might say that considering the absence of the response, vitamin B12 deficiency might not be the cause of or related to the eye movement disorder. However, absence of the response can be associated with long-term vitamin B12 deficiency, which could have caused permanent damage to nervous tissue.

In conclusion, early diagnosis of vitamin B12 deficiency is vital; since, when damage to nervous tissue starts, its therapy might become unresponsive [11]. Whereas eye movement disorders are hardly caused by vitamin B12 deficiency, we believe that serum vitamin B12 should be measured in any patient with unexplained eye movement disorder, as the response to delayed therapy in vitamin B12 deficiency can be poor.

References