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

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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 constant 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.

Year  Age  Eye movement disorders  Response to therapy  MRI findings
1986[5]  69  Downbeat nystagmus  Mild improvement  Mild cerebellar atrophy

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 1: The reported patients with eye movement disorders caused by vitamin B12 deficiency.

<table>
<thead>
<tr>
<th>Year</th>
<th>Age</th>
<th>Eye Movement Disorder</th>
<th>Response to Therapy</th>
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<tbody>
<tr>
<td>1984</td>
<td>47</td>
<td>Upward gaze palsy</td>
<td>Mild improvement</td>
</tr>
<tr>
<td>1938</td>
<td>64</td>
<td>Total ophthalmoplegia</td>
<td>Very mild improvement</td>
</tr>
<tr>
<td>1998</td>
<td>60</td>
<td>Bilateral horizontal gaze</td>
<td>Not mentioned</td>
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</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