Risk Factors in Spasmus Nutans

Czynniki ryzyka spasmus nutans

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Abstract

Background. Nystagmus, head nodding, and anomalous head position are symptoms of spasmus nutans. The etiology for spasmus nutans is unknown. Much remains to be understood about infantile-onset nystagmus, particularly in the mechanisms of spasmus nutans. Spasmus nutans was reported to be more frequent in crowded sections of large cities and in children of low socioeconomic families. Factors reflecting a lower socioeconomic status were more prevalent in patients with spasmus nutans.

Objectives. The purpose of this study is to describe these risk factors in infants, which may cause transient, idiopathic nystagmus in infants.

Material and Methods. In this study, the authors retrospectively examined 18 patients with spasmus nutans.

Results. Ten of the 18 patients with spasmus nutans were found to have iron deficiency anemia, 2 patients had rickets and 2 patients had both. Twelve of them came from lower socioeconomic families.

Conclusions. Ocular motor stability undergoes a period of postnatal maturation. Iron or D vitamin deficiency may cause transient abnormal eye and head movements. We believe that low socioeconomic status may represent a risk factor, such as iron deficiency anemia, rickets or both for the development of spasmus nutans (Adv Clin Exp Med 2011, 20, 2, 183–186).

Key words: spasmus nutans, infant, anemia, rickets.

Streszczenie


Cel pracy. Przedstawienie tych czynników ryzyka u dzieci, które mogą wywołać przejściowy samoistny oczopląs u niemowląt.

Materiał i metody. Oceniono retrospektywnie 18 pacjentów ze spasmus nutans.

Wyniki. U 10 z 18 pacjentów ze spasmus nutans stwierdzono występowanie niedokrwistości z niedoboru żelaza, u 2 osób krzywicę i 2 pacjentów miało obie choroby. Dwunastu z nich pochodziło z rodzin o niskim statusie społeczno-ekonomicznym.


Słowa kluczowe: spasmus nutans, niemowlę, anemia, krzywica.

Spasmus nutans (SN) is a self-limiting benign clinical entity. Findings include head titubation, torticollis, and rapid, asymmetric, low-amplitude nystagmus. Components of this triad may develop at various times. In many cases, symptoms usually last a few months, sometimes years. The cause of this classic type of SN, which resolves spontaneously, is unknown. SN is usually described as a result of clinical observation. Signs usually develop within the first or second years of life. The diagnosis is established by the constancy of the characteristic triad and the elimination of the other causes of nystagmus [1, 2].
SN in infants and young children can be caused by many different conditions. There have been several reports of patients presenting with signs mimicking that of SN with asymmetric nystagmus and head nodding, who were subsequently found to have intracranial pathology [3]. Some infants have no physical findings of anterior visual pathway disease and no discernible neurological abnormalities. Sometimes no explanation can be offered to a family regarding their child’s sustained nystagmus. Clinicians may then make the diagnosis of motor nystagmus, even though there is no clear evidence that unexplained, sustained nystagmus is caused by any particular defect of ocular motor control.

Factors reflecting a lower socioeconomic status were more prevalent in patients with SN [4]. SN was reported to be more frequent in crowded sections of large cities and in children of low socioeconomic families. More recent studies confirm these observations [4].

The aim of this retrospective study was to examine visual functions of former patients with SN and to analyze their laboratory findings whether iron deficiency anemia or rickets can represent a risk factor for the symptoms of SN. In this study, the authors retrospectively examined 18 patients with SN.

**Material and Methods**

Retrospectively, eighteen children (7 males and 11 females) were reviewed in a multi-center cohort study. All these patients from different centers were referred to eye centers or pediatric neurology centers for further evaluation. Infants with SN seen from 2003 to 2009 were retrospectively analyzed. The age of onset varied from 6 to 24 months (mean age = 13.7 months). The inclusion criteria were rapid, asymmetric, low-amplitude nystagmus, head titubation, and torticollis. The excluding criteria in this study were history of prematurity, developmental delay, or other systemic problems. Thirteen of the patients in this study were presented originally to an ophthalmologist. Five of the infants were primarily referred to a pediatric neurologist. Cranial imaging was performed to ensure that all patients have an accurate diagnosis of SN. Twelve patients, previously diagnosed SN infants, had MR of the brain and 6 patients had already undergone brain CT scans. Patients with known neurological defects were excluded from the study. The authors retrospectively reviewed the medical records of all patients and carefully recorded their laboratory findings. They reviewed the patients’ history, paying special attention to socioeconomic, demographic status and their laboratory findings. Researchers followed the tenets of the Declaration of Helsinki.

10 of the 18 patients with SN were found to have iron deficiency anemia, 2 patients had rickets and 2 patients had both. 12 of them came from lower socioeconomic families. Table 1 and 2 summarize the findings in patients with SN.

**Table 1. Laboratory findings of the study group with spasmus nutans and anemia**

<table>
<thead>
<tr>
<th>No of the patient (Numer pacjenta)</th>
<th>Gender (Płeć)</th>
<th>Age – month (Wiek – miesiące)</th>
<th>Annual household income (Roczniki dochód gospodarstwa domowego)</th>
<th>Hemoglobin (Hemoglobina)</th>
<th>MCV (FL)</th>
<th>Ferritin (ng/mL)</th>
<th>Serum-Fe (mcg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt-1</td>
<td>male</td>
<td>18 low</td>
<td>09.74</td>
<td>62</td>
<td>5.2</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Pt-2</td>
<td>female</td>
<td>14 low</td>
<td>11.14</td>
<td>65</td>
<td>6.0</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Pt-3</td>
<td>male</td>
<td>13 low</td>
<td>10.14</td>
<td>65</td>
<td>5.5</td>
<td>38</td>
<td></td>
</tr>
<tr>
<td>Pt-4</td>
<td>male</td>
<td>15 intermediate</td>
<td>9.12</td>
<td>62</td>
<td>4.2</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Pt-5</td>
<td>female</td>
<td>11 low</td>
<td>09.08</td>
<td>58</td>
<td>5.0</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>Pt-6</td>
<td>female</td>
<td>13 intermediate</td>
<td>10.04</td>
<td>62</td>
<td>6.5</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Pt-7</td>
<td>male</td>
<td>14 low</td>
<td>11.12</td>
<td>58</td>
<td>7.0</td>
<td>37</td>
<td></td>
</tr>
<tr>
<td>Pt-8 (Pt-a)</td>
<td>male</td>
<td>12 low</td>
<td>09.80</td>
<td>54</td>
<td>6.3</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>Pt-9 (Pt-b)</td>
<td>male</td>
<td>13 low</td>
<td>10.40</td>
<td>64</td>
<td>6.8</td>
<td>34</td>
<td></td>
</tr>
<tr>
<td>Pt-10</td>
<td>female</td>
<td>14 low</td>
<td>8.9</td>
<td>55</td>
<td>5.8</td>
<td>29</td>
<td></td>
</tr>
</tbody>
</table>
Discussion

Nystagmus, head nodding, and anomalous head position are symptoms of SN. This disorder appears in early childhood and is thought to be self-limited. The etiology for the SN seen in these children is unknown; these cases suggest that the ocular motor control mechanism is likely to be potentially unstable and flexible during a short period of infancy [5].

Much remains to be understood about infantile-onset nystagmus, particularly in mechanisms of SN. Current understanding of infantile-onset nystagmus is mostly based on clinical observations and eye movement recordings [6].

A wide range of potential etiologies for SN in infants are unknown [7]. The authors have seen children in whom they could find no explanation for the nystagmus other than iron deficiency anemia and rickets. The purpose of this study is to describe these risk factors which may cause transient, idiopathic nystagmus in infants. The authors conclude that low socioeconomic status represents risk factors such as iron deficiency anemia, rickets or both, for the development of SN.

In 1897, Raudnitz et. al. described an association of SN with inadequate light exposure and rickets [8]. Several studies confirmed a high incidence of rickets in SN. In these studies onset of nystagmus was more frequent in darker months of the year and low social and hygienic conditions in the families of patients with SN [9, 10]. Vitamin D deficiency may also occur in unsupplemented dark-skinned infants or in breast-fed infants of mothers unexposed to sunlight [11]. In this study, 12 of the 18 patients were found to come from lower socioeconomic families. Four of them had low serum calcium and phosphorus levels with high alkaline phosphates which were suggestive of active rickets.

Factors reflecting a lower socioeconomic status were more prevalent in SN patients [4, 9, 10]. It is known that low socioeconomic status represents a risk factor for the development of SN. This syndrome was reported to be more frequent in crowded sections of large cities. The incidence of SN was much higher in those of low socioeconomic status and disturbed mother-child relationships. Therefore, it seems more likely that a low socioeconomic background can be a risk factor for SN [4, 12]. These findings are also supported by current findings.

This study suggests that ocular motor stability may undergo a period of postnatal maturation. Iron or vitamin D deficiency can transient eye and head movements with delayed cortical visual maturation [11, 12]. The authors of the current study believe that these risk factors can lead to SN which may be due to low socioeconomic status. These factors could be a contributing factor for the development of SN. This is the first study in the medical literature in which the risk factor in spasmus nutans is reported. Further studies with larger patient groups are needed.

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References


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