Benign paroxysmal vertigo of childhood

Łagodne napadowe zawroty głowy typu dziecięcego

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Summary

Objective. To relate the authors' experience to the diagnosis and follow-up of patients with benign paroxysmal vertigo of childhood (BPV) who were followed-up at the Children's Hospital of Bydgoszcz between 1999 and 2004, and to review and discuss controversial issues regarding the disease. **Methods.** Among 124 children suffering from vertigo 14 were classified as having BPV. All the children were submitted to differential diagnosis protocol which consisted of meticulous history, otolaryngological, ophthalmological, psychological, neurological examination, biochemical tests and standard neurootological examination including caloric tests. The children were followed-up and the tests were repeated if no improvement was observed. **Results.** All the children suffered from episodic vertigo of variable intensity and frequency. All of them were neurologically intact. In 8 patients pathologic ENG results were found, only 1 patient with canal paresis could be considered as having peripheral lesion, 7 patients had central/mixed pathology. The follow-up was favorable in majority of patients. Six of them recovered completely, in 6 an improvement was noted and in 2 no improvement was observed. Three patients after remission of BPV attacks developed migraine. One child before development of BPV attacks suffered from paroxysmal torticollis of infancy. **Conclusions.** Childhood BPV is a disorder of vestibular system with the onset occurring mainly in preschoolers aged 1–7. Older children with the onset of BPV - like symptoms should be suspected for functional background of the disease. There are no typical ENG features for BPV. The only objective evidence of vestibular dysfunction is the presence of nystagmus during the attack. The disease is probably of vascular origin and there is strong evidence for close relationship between spasmodic torticollis, BPV and migraine.

Key words: vertigo, dizziness, migraine, spasmodic torticollis, ENG

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INTRODUCTION

Although vertigo in children is not a common disorder, the causes of the symptom may be multiple and making a differential diagnosis is not easy [2–4].

Benign paroxysmal vertigo of childhood is a relatively rare condition - which manifests itself as a paroxysmal, non-epileptic recurrent episode, characterized by subjective or objective vertigo that occurs in children. It is probably underestimated in its frequency due to the fact that the diagnosis and systematic approach to vertigo are much more difficult in children than in adults. There are several features, which are regarded to be characteristic for this entity [1, 11]:

– It occurs most often in preschoolers, but may also be found in elementary school-age children and in young teenagers

– Sudden brief attacks of dizziness and nystagmus (abrupt attacks of turning sensation lasting

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from seconds to minutes), are usually unrelated to any position

– It is frequently associated with pallor, nausea and vomiting

– Patients are neurologically intact

– It always ends in complete resolution of symptoms and return to normal activities.

– Usually has favorable prognosis

Numerous articles on the BPV can be found in the literature. However several features such as results of vestibular testing, and the age of patients are not consistent [2, 4, 7–11]. Etiology of the disease is unclear too. The objective of this paper is to relate the authors' experience to the diagnosis and follow-up of children with BPV, who were treated at the Children's Hospital of Bydgoszcz and to review and discuss controversial issues regarding the BPV.

MATERIAL AND METHODS

Among 124 children with vertigo, referred to our department in the years 1999–2004, 14 (11%) patients were considered to represent well documented cases of BPV. There was a slight female prevalence – 8 girls and 6 boys. The mean age of children with the onset of the disease was 4,4 and the range 1–7 years.

After a careful personal and family history, the children were submitted to complete examination consisting of:

– Neurological, otolaryngological, ophtalmological, psychological examination

– Standard neurootological examination (Romberg, Fukuda, Unterberger, Dix-Halpike test)

– ENG – calibration, spontaneous nystagmus, tracking, optokinetic nystagmus, positional nystagmus, caloric tests

– Hearing tests including BERA in selected cases

– MRI or CT imaging in selected cases

– Biochemical tests excluding metabolic disease

The children were systematically followed-up and selected tests were repeated if no recovery was observed.

An improvement was noted with respect to frequency and duration of vertigo episodes and the occurrence of accompanying symptoms, i.e. headaches, nausea and balance problems.

RESULTS

All the children suffered from recurrent attacks of vertigo of variable duration: 5-10 seconds to hours, and variable frequency : daily to monthly. Balance problems were observed in 6 patients, a headache accompanying vertigo attacks in 2 patients, nausea and vomiting in 4 patients, tinnitus in 1 patient, and nystagmus at the time of the attack was noted in 6 patients (Table I).

Some symptoms occurring at the attack-free intervals and being not related to the attacks were also reported (Table II).

Four children had a family history of migraine. Two patients had vision problems. Audiometric tests were normal in all patients except one, who had conductive hearing loss due to temporary episodes of otitis media with effusion (OME). We included her in the study since vertigo attacks were observed in her, both at periods with- and without middle ear effusion, which was well-documented by tympanometry. We realize that there are some patients who may have balance problems due to middle ear effusion, but apparently vertigo was not related to OME in the case described above [13].

ENG results:

Nystagmic responses were normal and symmetrical in 5 patients in caloric tests. Eight patients presented pathologic responses (Table III).

Only one patient with canal paresis could be considered as having peripheral lesion of the vestibular system, in 7 patients central/mixed pathology was found.

Follow-up of patients:

Duration of the disease was from 6 months to 9 years, with the mean period being 3.1 years. Complete remission was observed in 6 patients, an improvement after reduction in frequency, intensity and duration of attacks was observed in 6 patients. In 2 patients no improvement was noted. Three patients after remission of the BPV attacks developed classical symptoms of common migraine. One patient before development of vertigo attacks and classical symptoms of BPV suffered from periodic attacks of paroxysmal torticollis in infancy.

DISCUSSION

There is enough data to state that BPV is at present a relatively clear and defined entity. However, three important elements still require more expla-

nation – age of patients with the onset of the disease, vestibular function and etiology of the sickness.

According to early reports of Basser (1964) and Koenigsberger (1970) BPV begins within the first four years of life. More recent papers suggested the onset of attacks in older children - even in young teenagers up to twelve years old [9, 11, 14]. The age of the onset of the disease in this group varied from 1–7. At the beginning of the study we also suspected some older children even teenagers of having BPV, but they were excluded from the study since after psychological examination we diagnosed functional background of vertigo attacks in all of them. We suggest focusing a special interest on psychological problems in older children suspected of BPV because this is probably the cause of the differences between various reports (Table IV).

It was initially reported that BPV children had a hypoactive caloric test response in one ear in a form of moderate, severe, or complete canal paresis, which might represent peripheral etiology of the disease [1, 6, 10]. That finding was not confirmed by more recent data, were authors reported normal caloric responses in substantial number of patients [7, 11]. In our study only one patient had unilateral vestibular hypofunction, 5 had normal response and 7 showed a pathology of mixed type.

In our opinion neither peripheral changes nor lack of pathology in caloric tests are characteristic for BPV. Following other critical reports we think that it is hard to explain how a unilateral loss of caloric excitability could be related to a transient paroxysmal disorder such as benign paroxysmal vertigo of childhood [7, 11]. Normal vestibular response may be consistent with the transient peripheral or central dysfunction of vestibular pathways. On the other hand, transient vestibular disorder may be related to ischemic disorders of the vertebrobasiliar system which could explain central pathology found in ENG tests in some of our patients. Similar ENG findings we noted in a group of children with severe migraine [12].

The ENG results in BPV patients are not consistent and the only objective evidence of vestibular dysfunction is the presence of nystagmus during the attack. Therefore we think it is important to prepare the child's parents to look for eye movement during the attack of dizziness. After being encouraged to observe the child carefully, several parents confirmed nystagmus in their children during attacks.

Table I.

Symptoms accompanying vertigo attacks	Number of patients
Dysequilibrium	6
Headache	2
Nausea and vomiting	4
Tinnitus	1
Nystagmus (parent's observations)	6

Table II.

Symptoms not accompanying vertigo attacks	Number of patients
Headache not accompanying attacks	5
Periodic tinnitus	3
Conductive hearing loss to due episodes of ome	1
Vision problems	2
Non specyfic, insignif. abnormalities in EEG	3
Motion sickness	8

Table III.

Electronystagmography results	Number of patients
Normal	5
Pathological	8
Central/mixed pathology	7
Peripheral pathology	1
Spontaneous nystagmus	4
Irregular eye tracking test	2
Optokinetic. asymmetry	1
Gaze nystagmus	1
Positional nystagmus	6
Canal paresis	1
Directional preponderance	4

Table IV.

Authors	Age of patients with the onset of BPV
Basser 1964	1–4
Koenigsberber 1970	1–4
Watson and Steele 1974	2–10
Finkelhor 1987	4–10
Lanzi 1986	2–12
Present study 2004	1–7

Since the first diagnosis of the disease it has been suggested that there is a strong relation between BPV and migraine [2, 5, 8, 12]. Although the exact etiology is not known it has been postulated that the attacks are due to transitory vascular disturbance which produces ischaemia of the vestibular nuclei and pathways [1, 9]. In the group of our patients we have found support for this relationship. Four children had family history of migraine, 8 had motion sickness which is regarded as pathological correlate of migraine. It has also been suggested that paroxysmal torticollis of infancy, BPV, and migraine are of vascular origin and progression from one disease to another has been observed [6, 7, 9]. Three of our patients developed migraines after resolution of BPV and 1 child had previously experienced spasmodic torticollis of infancy.

CONCLUSIONS

– Childhood BPV is a disorder of vestibular system with the onset occurring mainly in preschoolers aged 1-7

– There are no typical ENG features for BPV. ENG tests results are not consistent, the only objective evidence of vestibular dysfunction is the presence of nystagmus during the attack

– The disease is probably of vascular origin and there is strong evidence for close relationship between spasmodic torticollis, BPV and migraine.

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