ORIGINAL ARTICLE

Evaluation of vestibular functions in children with vertigo attacks

A Uneri, D Turkdogan

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See end of article for authors' affiliations

Correspondence to: Dr D Turkdogan, Atakoy 9, Kisim B7, D:6 Istanbul, Turkey; dturkdogan@hotmail.com

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Aim: To examine vestibular system functions in children with episodic vertigo attacks.
Methods: Thirty four children (20 males) aged 4–18 years with paroxysmal dizziness and/or vertigo attacks were evaluated. A medical history for vestibular symptoms and migraine was taken. Vestibular and auditory functions were assessed.
Results: Chronic headache attacks consistent with migraine were reported in 12 children and motion sickness was reported in 30. Family history in first degree relatives was positive for migraine in 29 children and for episodic vertigo in 22. Electronystagmography and videonystagmography showed two types of nystagmus: spontaneous vestibular nystagmus (41%) and benign paroxysmal positional nystagmus (BPPN) (59%). The first type of nystagmus was assessed as a sign of vestibulopathy and the patients with BPPN were diagnosed as having benign paroxysmal positional vertigo (BPPV). Audiometric examination in four cases revealed bilateral sensory neural hearing loss in low frequencies. Pure tone averages in 30 cases were within normal ranges; however low frequencies in 28 of them were approximately 10 dB lower than high frequencies. Unilateral caloric responses diminished in eight children.
Conclusions: Peripheral vestibular problems in childhood present in a wide spectrum, which varies from a short episode of dizziness to a typical vestibular attack with fluctuating sensory neural hearing loss or episodes of BPPV. A considerable number of these vestibular problems might be related to the migraine syndrome.

Episodic vertigo and/or dizziness in children are not frequent symptoms. The most common cause of vertigo in children without any detectable ear disease or hearing loss has been suggested to be benign paroxysmal vertigo of childhood (BPVC).[1–3] BPVC was originally described as a typical vestibular attack including nystagmus, nausea, vomiting, and diaphoresis.[2] The age of occurrence is usually 1 to 5 years.[3] Another known feature of BPVC is its relation with migraine.[1–3] Migraine is a well known cause of episodic vertigo.[4–6] Johnson[5] suggested migraine related vertigo and vestibular or atypical Meniere's disease to be the same pathological condition presenting with a similar symptom complex. Recently, Baloh and colleagues[6] reported a relation between migraine and benign paroxysmal positional vertigo (BPPV), the most common cause of periodic vertigo syndromes of adults, although these are rarely reported in children.[4] BPPV occurs when freely floating otoconia enter one or more of the semicircular canals and move under the influence of gravity.[7–10]

We aimed to investigate characteristics of childhood peripheral vestibular disorders based on clinical and neurotological features of a series of 34 patients presenting with periodic dizziness and/or vertigo attacks.

MATERIALS AND METHODS

The study group consisted of 34 children (20 males; aged 4–18 years, mean 11.5 (SD 2.8) years) who were examined for paroxysmal attacks of dizziness and/or vertigo in outpatient clinics of the Departments of Pediatric Neurology and Neurotology between July 1999 and October 2001.

A detailed history of attacks of vertigo and other accompanying symptoms, migraine, motion sickness, and family history of migraine and episodic vertigo was obtained. Neurological and neurotologic examination, audiometric evaluation, electronystagmography (ENG), and videonystagmography (VNG) tests were performed.

Migraine was diagnosed based on criteria proposed by the International Headache Society.[11] Standard electroencephalography (EEG) was recorded in seven patients during a headache attack.

The Dix-Hallpike (D-H) test associated with VNG was used to diagnose and classify the features of nystagmus. The diagnosis of BPPV was based on the presence of positional nystagmus, which changes direction by changing the position of the head in the D-H manoeuvre.[5 6] Spontaneous vestibular nystagmus detected by VNG or ENG while the patient was sitting in a neutral position or induced by the D-H manoeuvre with no change of direction was regarded as a diagnostic finding of vestibulopathy.

RESULTS

All patients, except four with a history of a typical vestibular attack, had non-specific symptoms such as dizziness. Twelve patients also had attacks of migraine headache, which did not have a temporal relation with vertigo. Motion sickness was positive in 30 patients. Family history of migraine headache in first degree relatives was positive in 29 patients. Twenty two patients had a positive family history of episodic vertigo.

VNG analysis revealed spontaneous vestibular nystagmus in 14 patients diagnosed as vestibulopathy and positional nystagmus in 20 diagnosed as BPPV.

EEG examination in all seven patients was inconclusive.

Audiometric examination revealed normal pure tone average (PTA) in 30 patients. However, 28 of 30 patients had

Abbreviations: BPPN, benign paroxysmal positional nystagmus; BPVC, benign paroxysmal vertigo of childhood; EEG, electroencephalography; ENG, electronystagmography; PTA, pure tone average; SNHL, sensory neural hearing loss; VNG, videonystagmography

decreased thresholds at low frequencies (especially at 250 Hz) compared to high frequencies. As the low frequency was 250 Hz, PTA was in the normal ranges.

Mild sensory neural hearing loss (SNHL) in frequencies of 250, 500, and 1000 Hz was found in four patients. Two were diagnosed as BPPV and two as vestibulopathy.

ENG analysis revealed that all of the spontaneous nystagmus and positional nystagmus, which were in the horizontal plane, were recorded. Unilateral mild hypoactive caloric response ranging from 25% to 40% in eight patients (four with vestibulopathy and four with BPPV) was noted. No correlation was found between the severity of the symptoms and hypoactive caloric response in ENG testing.

Only five cases with BPPV had second vertigo attacks during the follow up period ranging from six months to four years. The only finding was a spontaneous nystagmus of vestibulopathy, but not a BPPV attack.

DISCUSSION

Vertigo attacks are less dramatic in children compared to adults. However, parents are highly anxious because of the lack of diagnosis. Determination of diagnosis and provision of information to the patient and parents would eliminate anxiety.

Most of our patients presented with mild to severe dizziness rather than a full blown typical vestibular attack.

A significant number of our patients were diagnosed as BPPV involving the posterior or horizontal channel. BPPV is rarely diagnosed in children with episodic vertigo. A series of 255 patients with BPPV did not include any case younger than 20 years of age.[10] Baloh and colleagues[4 7] reported two cases aged 11 and 12 years with BPPV and three relatives with BPPV and migraine of childhood onset. BPPV has usually been diagnosed based on the criteria of the Dix-Hallpike test since 1952.[6 7 12] VNG completely eliminates optic fixation and detects all nystagmus, even weak cases that cannot be seen by routine tests. Lack of routine use of VNG has probably led to underestimation of some weak positional or spontaneous nystagmus. ENG can be valuable in the diagnosis of horizontal planed spontaneous vestibular nystagmus if VNG is not available.

We found unilateral hypoactive caloric response in four patients with BPPV and four with vestibulopathy. Spontaneous nystagmus was the only finding in 71.5% patients with vertigo related to vestibulopathy. The presence of decreased caloric responses in 20% of patients with BPPV indicates an association of BPPV with vestibulopathy.

We believe that the presence of nystagmus rather than decreased caloric response in association with a positive history is diagnostic for peripheral vestibular disorders.

The most outstanding audiometric characteristic of our series is the lower thresholds of low frequencies (88%). There was a difference from 10 to 20 decibels between 250 Hz and 1000 or 2000 Hz regardless of normal PTA. The lowered thresholds in low frequencies may be helpful in determination of actual pathology.

The pathogenesis of vestibulopathy and its association with migraine is not known for certain. Baloh and Honrubia[4] hypothesised that vasospasm of the labyrinth arteries is responsible for the peripheral vestibular symptoms as suggested in the pathogenesis of migraine symptoms. It is also known that ischaemic damage has a role in the aetiopathogenesis of BPPV.[13]

Motion sickness, a clinical phenomenon provoked by passive locomotion or movement of the visual environment only, consists of tiredness, weakness, pallor, diaphoresis, hypersalivation, nausea, and emesis. It has been reported in 60% of patients with migraine, whereas the incidence in the normal population is only 20%.[14] The high incidence (88%) of motion sickness in our study group may implicate an association between motion sickness and migraine or vestibular symptoms.

We therefore suggest that patients who have a connection with migraine (by self or by first degree relatives), and experience periodic vestibular symptoms, possibly share a similar pathogenesis with other migraine symptoms. Periodic vestibular symptoms appear in a wide spectrum consisting of attacks of dizziness with the only finding being spontaneous vestibular nystagmus on one side and typical vestibular attack episodes with fluctuating SNHL or BPPV on the other side. We also believe that BPVC is part of the same spectrum instead of being a separate entity.

Authors' affiliations
A Uneri, D Turkdogan, Marmara University, Institute of Neurological Sciences, Istanbul, Turkey

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