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The Migraine: Benign Paroxysmal Vertigo of Childhood Complex

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Abstract: The migraine—a benign paroxysmal vertigo of childhood complex is the most frequent etiology of pediatric dizziness, with an incidence of 34.7%. We present a series of 34 children in whom this entity was diagnosed. We describe the most important characteristics and discuss the possible etiological factors. We review the theories about a common pathophysiological origin for migraine, benign paroxysmal vertigo of childhood, and paroxysmal torticollis. Evolution of these entities confirms the idea of a common origin and a different vestibular symptomatology, depending on the age of the child experiencing dizziness.

The migraine—a benign paroxysmal vertigo of childhood complex (BPVC)—is the most frequent cause of pediatric dizziness presented in the majority of patients in series reviews [1–3]. Such a high incidence has not resulted in a greater knowledge about the pathophysiology of this disorder. The first description of this entity was proposed by Basser [4] in 1964, as a vestibular neuronitis. Since then, many authors have proposed different theories to explain the common physiology of both entities. The new International Headache Society classification (IHSC) [5], published in 1988, groups BPVC (IHSC 1.5.1.), paroxysmal torticollis of childhood, periodic vomiting, and basilar migraine in the migraine family. Patients present different clinical patterns of vestibular symptoms with a common pathophysiology: vasoconstriction of vestibular nuclear vessels followed by vasodilation.

BPVC appears in children younger than age 4. It shows rotatory vertigo or disequilibrium attacks without loss of consciousness, lasting from seconds to minutes; no apparent provocation or prodromes are seen; autonomic nervous system signs are common, though nystagmus could appear; and no cochlear symptoms are evident. Spontaneous recovery is the tendency. Initially, headache is not so frequent, but some of the patients will develop migraine cephalagias in adolescence.

Vertebrobasilar migraine (IHCS 1.2.4.), described by Bickerstaff [6] in 1961, must be defined by two of the following symptoms according to IHSC criteria: vertigo, tinnitus, hearing loss, ataxia, dysarthria, visual symptoms, diplopia, paresthesia, or decreased level of consciousness. Pulsatile headache follows these clinical manifestations. It appears before age 20.

Paroxysmal torticollis of childhood, described by Snyder [7] in 1969, appears in the first year of life. It shows spells of head tilt lasting from minutes to days and, often, associated autonomic signs and ataxia. Spontaneous resolution is the rule, but it can be followed by BPVC, classic or common migraine, and Meniere's disease [7].

MATERIALS AND METHODS

We have reviewed a group of 34 individuals managed during a period of 10 years at our ear, nose, and throat clinic. Selection criteria included pediatric age (newborns to age 15) and diagnosis of migraine or BPVC. We also present one case of paroxysmal torticollis in childhood.

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General otolaryngologic and ophthalmologic assessment was performed in all individuals. An exhaustive neurootolaryngologic exploration and different audiological tests were carried out: impedance and stapedius reflex measurement (GSI 33 Gason Stadler) in all cases; audiometry (MAICO MA 41) in 26 patients; and brainstem auditory evoked responses (Pathfinder II, Nicolet Biomedical) in 13 patients.

Vestibular assessment was performed with electronystagmography studies in all cases but one, using both caloric and rotatory stimulation (CFDP 77B-V, Racia S.A.). Dinamic Posturography (Smart Balance Master, Neurocom Int.), radiological exploration (computed tomography, magnetic resonance imaging), electroencephalography, and blood tests were carried out if necessary.

RESULTS

From July 1987 to July 1997 (10 years), we have observed a total of 24,580 patients in our otolaryngology clinic. Pediatric dizziness was the diagnosis in 98 cases (0.39%). Thirty-four patients presented with the complex of migraine–BPVC–paroxysmal torticollis in childhood (incidence: 34.7% pediatric vertigo, 0.14% of all the patients seen in that period). Migraine was diagnosed in 18 cases (52.9%), BPVC in 15 cases (44.1%), and paroxysmal torticollis in 1 case (2.9%).

The average age was 7.54 years (8 months to 15 years). On average, migraine patients presented at an age older than that of BPVC patients (9.52 versus 5.55 years, respectively). A total of 62.5% patients were male and 37.5% were female. A total of 41.9% of children were referred from pediatric clinics, 35.3% were referred from otolaryngology clinics, and 23.5% were referred from neurology clinics. Family history for migraine was found in 53% of migraine patients, whereas only 14.3% of BPVC.

Otolaryngologic assessment included abnormal impedance test results in nine patients and the absence of stapedius reflex in two. Brainstem auditory evoked response exploration was performed in seven migraine patients (two with longer retrocochlear latency and one with an affected cochlea because of later parotiditis) and in five BPVC patients (one case with longer retrocochlear latency).

Audiological findings showed the presence of hearing loss in one case of parotiditis. Seven of the patients presented with mild signs of otitis media with effusion in the first exploration performed in our clinic. Four children described unilateral tinnitus and, in two cases, tinnitus was bilateral.

Vertigo was of the rotatory type in most cases (66.6%). Duration of the episodes was from seconds to minutes, with a frequency of two or more monthly. No loss of consciousness was found, and vegetative symptoms were present in 33.3% of the migraine patients and in 40% of BPVC patients.

Electronystagmography was carried out in all cases but two. Results are listed in Table 1. Dynamic posturography was performed in one migraine patient. Stability average was 60%, with a disfunctionality in condition 5 of the sensory organizing test. Radiological exploration in 23 children showed only a mild ethmoidal sinusitis in three cases. CT and magnetic resonance imaging were normal.

Medical treatment (2-pirrolidona acetamide (piracetam), diazepam, N-((etil-1 pirrolidinil-2) metil) metoxi-2 sulfanoil-5 benzamine (sulpiride)) was indicated in 66.7% of migraine patients and in 33.3% of BPVC patients. Two patients were referred to a neurology clinic.

DISCUSSION

The pathophysiology of BPVC still is unclear, but it has been related to vascular disturbances of the posterior cerebral circulation, affecting the vestibular nuclei secondarily [8]. The mechanisms altered are represented by quantitative or qualitative modifications of the neuromediators, probably acting at the central level. Possible mechanisms that can explain the aura in migraine or the paroxysmal vertigo disorder [9,10] are an ischemia by vasoconstriction due to abnormal accumulation of vasoactive substances (serotonin, tyramine, prostaglandin) or a neural dysfunction located in a specific area mediated by neurons containing serotonin. The presence of nystagmus during vertigo attacks reaf-

Table 1. Electronystagmography (Bithermal-Rotational Test) Results

Finding	Migraine (no.)	BPVC (no.)	Paroxysmal Torticollis (no.)
Hyporeflectivity	9 (unilateral in 6)	5 (unilateral in 2)	1
Normoreflectivity	5	6	—
Hypereflectivity	4 (unilateral in 1)	2 (unilateral in 1)	—

BPVC = benign paroxysmal vertigo of childhood

firms a vestibular pathway origin. A bidirectional nystagmus described in some of the cases and the lack of audiological signs (deafness and tinnitus) may rule out a peripheral origin for the alteration [6,11].

In the presence of paroxysmal ischemia of the vestibular nuclei, various clinical manifestations have been described, depending on the most affected area of the nuclei. Superior-part disturbances would more likely cause nystagmus, whereas torticollis would be caused by disturbances in the inferior part. In younger children, the inferior part of the vestibular nuclei still is involved. This theory would explain why children affected by ischemic disturbances in the inferior part manifest paroxysmal torticollis earlier in childhood. The same mechanisms would affect the superior part, exhibiting paroxysmal vertigo later in life. We have found varying age averages, depending on the entities: 18 months for paroxysmal torticollis patients, 5 years for BPVC patients, and 9.5 years for migraine patients. This finding could mean that the identical process is occurring in different age stages.

Migraine and BPVC show similar vestibular symptoms: paroxysmal attacks of dizzines accompanied by nystagmus. The presence of a headache after an aura with vestibular symptoms is typical of migraine. Vercelleto [12] has proposed some criteria to identify a migraine as the cause of vertigo spells:

Definitive: vertigo accompained by typical migraine symptoms (visual disorders, headache)
Probable: isolated vertigo spells with mild headache and some typical migraine symptoms in some spells
Possible: isolated vertigo spells, family history of migraine

Between 30% and 50% of BPVC cases can evolve to classic migraine in older children [13]. Meniere's disease has been described as an end point of BPVC evolution [14].

Although many authors refer to BPVC as a migraine equivalent, other authors are more skeptical concerning this theory. Basser [4] and Paparella and Shumrick [15] deny any migraine similarity in BPVC. Walquist et al. [13] found a migraine heredity in 63.64% of his series. Toupet [14] offered an otolithiasis explanation for BPVC, similar to positional paroxysmal vertigo. This wide field of theories will require more thorough research in the future.

Differential diagnosis of the migraine-BPVC complex is necessary to prevent or treat more severe implications: epilepsy of the temporal lobe, presenting vertigo as an aura or being the seizure itself, and posterior fossa tumors, vestibular neuronitis, Menieres disease, head trauma, and acute labyrinthitis with severe hearing impairment.

The principles for a common pathophysiology of migraine and BPVC have been presented in this review. Evolution of these entities confirms the idea of a common origin and a different vestibular symptomatology, depending on the age of children affected by dizziness. To verify these theories, follow-up studies are required to assess the possible evolution to other entities included in the same complex.

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