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Review Article

Child Migraine Spectrum

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Abstract

Migraine is a heterogeneous disorder and prevalence of migraine increasing. Migraine phenotype differs somewhat in the developing brain, and childhood episodic syndromes may arise before typical migraine headache. Some studies showed abnormalities in the maturation of brain functions in migraine children and adolescents. Migraine has two major subtypes but migraine is spectrum that involved different clinical face from headache to hemiplegia. Migraine without aura is a clinical syndrome characterized by headache with specific features and associated symptoms. Migraine with aura is primarily characterized by the transient focal neurological symptoms that usually precede or sometimes accompany the headache. Hemiplegic migraine description is migraine with aura including motor weakness. Retinal migraine is repeated attacks of monocular visual disturbance, including scintillations, scotomata or blindness, associated with migraine headache. Migraine and epilepsy are prototypical examples of paroxysmal brain disorders. Although migraine-like headaches are quite frequently seen in the epileptic postictal period, sometimes a seizure occurs during or following a migraine attack. Childhood episodic syndromes such as recurrent gastrointestinal disturbance, cyclical vomiting syndrome, abdominal migraine, benign paroxysmal vertigo, benign paroxysmal torticollis that may be associated with migraine although historically noted to occur in childhood, they may also occur in adults. Migraine preventive strategies are particularly important in children. The first essential step is to explain the condition to the child and his or her parents. Explanation and reassurance may be the only treatment needed in some cases. As with migraine, the best treatment is prevention. The aim was to establish the occurrence of migraine spectrum in child neurology practice and among migraine, and to discuss their presentation.

Keywords: Migraine; Childhood; Headache; Neurology

Introduction

Migraine is a heterogeneous disorder: attacks vary in pain intensity, duration, pattern of associated features, and frequency of occurrence. Some migraineurs have recurrent attacks without remission periods; others experience symptom-free intervals lasting several years; a third group becomes free of attacks for the rest of their life. Migraine is the second most common cause of chronic recurrent headache in school children [1,2].

Migraine without aura and migraine with typical aura are common in pediatric neurology, but migraine variants occur rarely [3]. Migraine variants represent migraine attacks, characterized by serious neurological or gastrointestinal signs but with mild or absent headache [4].

The aim of this study was to establish the occurrence of migraine variants among all patients in pediatric neurology practice, as well as among migraine, and also to discuss their presentation and their place in the ICHD-III [5].

Epidemiology

The prevalence of migraine has been studied across all ages starting in early childhood. There is a slight predominance in boys in the pre-pubertal years, and the overall occurrence increases throughout adolescence into young adulthood when there is a transition to predominance in girls [1,2,6]. Subsequent epidemiological studies have continued to show the high frequency of headaches in children and adolescents, with migraines being the most common disabling type various criteria have been used to define migraine, although most recent studies from several countries and geographical locations have used the second edition of the International Classification of Headache Disorders (ICHD-II) [7,8]. Epidemiological studies have documented its high prevalence and high socio-economic and personal impacts. Migraine is a frequent disease with a prevalence of 3-15% in children and adolescents [8-11]. Positive family history for headache is commonly reported with a frequency of 60–77.5% [8,12].

Pathophysiological Mechanisms of Migraine

Migraines are self-limiting dysfunctions of grey matter. It is primarily a neuronal sensory dysfunction which secondarily involves the vascular systems. Involvement of the sensory nerve fibers within meningeal blood vessels gives rise to head pain [13,14].

The exact mechanism of the central nervous system pathophysiologic dysfunction in migraine is unclear. It is generally accepted that spreading of neuronal depression, neurogenic inflammation, and the activation of trigeminovascular system are involved [15-19].

The neurophysiologic investigation of the pathophysiological mechanisms subtending migraine in children and adolescents could be particularly interesting, since during the developmental age the

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migrainous phenotype is scarcely influenced by many environmental factors that can typically act on adult headache patients. The neurophysiologic abnormality most frequently found in adult migraineurs, that is the reduced habituation of evoked potentials, was confirmed also in migraine children. Some studies showed abnormalities in the maturation of brain functions in migraine children and adolescents. While the visual system maturation seems slowed in young migraineurs, the psychophysiological mechanisms subtending somatosensory spatial attention in migraine children are more similar to those of healthy adults than to those of age-matched controls. There are some still unexplored fields that will have to be subjects of future studies. The nociceptive modality, which has been investigated in adult patients with primary headaches, should be studied also in pediatric migraine. Moreover, the technique of transcranial magnetic stimulation, not yet used in young migraineurs, will possibly provide further elements about brain excitability in migraine children [20].

Regional cerebral blood flow imaging shows no changes suggestive of cortical spreading depression (CSD) during attacks of migraine without aura, although blood flow changes may occur in the brainstem, as may cortical changes secondary to pain activation. This contrasts with the pathognomonic spreading oligaemia of migraine with aura. Although the bulk of the literature suggests that CSD does not occur in migraine without aura, some recent studies disagree. Furthermore, it has been suggested that glial waves or other cortical phenomena may be involved in migraine without aura. The messenger molecules nitric oxide (NO), 5-hydroxytryptamine (5-HT) and calcitonin gene-related peptide (CGRP) are involved. Although the disease was previously regarded as primarily vascular, the importance of sensitization of pain pathways, and the possibility that attacks may originate in the central nervous system, have gained increasing attention over recent decades [20].

Classification of Migraine

The International Classification of Headache Disorders, 3rd edition (ICHD-3) has been released by the 'International Headache Society' in May 2013 [5]. As this version is based on a large body of research on headache, in contrast to previous editions that were mostly based on opinion of experts, it is being considered as a major step forward in the diagnosis and management of headache [21]. Migraine has two major subtypes but migraine is spectrum that involved different clinical face from headache to hemiplegia.

Migraine without aura is a clinical syndrome characterized by headache with specific features and associated symptoms. Migraine with aura is primarily characterized by the transient focal neurological symptoms that usually precede or sometimes accompany the headache. Some patients also experience a premonitory phase, occurring hours or days before the headache, and a headache resolution phase. Premonitory and resolution symptoms include hyperactivity, hypo activity, depression, cravings for particular foods, repetitive yawning, fatigue and neck stiffness and/or pain [21] Table 1.

Table 1: Classification of migraine.

1.1 Migraine without aura
1.2 Migraine with aura
1.2.1 Migraine with typical aura
1.2.1.1 Typical aura with headache
1.2.1.2 Typical aura without headache
1.2.2 Migraine with brainstem aura
1.2.3 Hemiplegic migraine
1.2.3.1 Familial hemiplegic migraine (FHM)
1.2.3.1.1 Familial hemiplegic migraine type 1
1.2.3.1.2 Familial hemiplegic migraine type 2
1.2.3.1.3 Familial hemiplegic migraine type 3
1.2.3.1.4 Familial hemiplegic migraine, other loci
1.2.3.2 Sporadic hemiplegic migraine
1.2.4 Retinal migraine
1.3 Chronic migraine
1.4 Complications of migraine
1.4.1 Status migrainosus
1.4.2 Persistent aura without infarction
1.4.3 Migrainous infarction
1.4.4 Migraine aura-triggered seizure
1.5 Probable migraine
1.5.1 Probable migraine without aura
1.5.2 Probable migraine with aura
1.6 Episodic syndromes that may be associated with migraine
1.6.1 Recurrent gastrointestinal disturbance
1.6.1.1 Cyclical vomiting syndrome
1.6.1.2 Abdominal migraine
1.6.2 Benign paroxysmal vertigo
1.6.3 Benign paroxysmal torticollis

Migraine

Migraine without aura

Migraine is recurrent headache disorder manifesting in attacks lasting 4-72 hours. Typical characteristics of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia.

Migraine headache in children and adolescents (aged less than 18 years) is more often bilateral than is the case in adults; unilateral pain usually emerges in late adolescence or early adult life. Migraine headache is usually frontotemporal. Occipital headache in children is rare and calls for diagnostic caution. In young children, photophobia and phonophobia may be inferred from their behavior Table 2.

Table 2: Diagnostic criteria of migraine without aura.

A. At least five attacks1 fulfilling criteria B–D
B. Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated)
C. Headache has at least two of the following four characteristics:
1. unilateral location
2. pulsating quality
3. moderate or severe pain intensity
4. aggravation by or causing avoidance of routine physical activity (e.g. walking or climbing stairs)
D. During headache at least one of the following:
1. nausea and/or vomiting
2. photophobia and phonophobia
E. Not better accounted for by another ICHD-3 diagnosis.

Table 3: Diagnostic criteria of Migraine with aura.

A. At least two attacks fulfilling criteria B and C
B. One or more of the following fully reversible aura symptoms:
1. visual
2. sensory
3. speech and/or language
4. motor
5. brainstem
6. retinal
C. At least two of the following four characteristics:
1. at least one aura symptom spreads gradually over _5 minutes, and/or two or more symptoms occur in succession
2. each individual aura symptom lasts 5-60 minutes1
3. at least one aura symptom is unilateral2
4. the aura is accompanied, or followed within 60 minutes, by headache
D. Not better accounted for by another ICHD-3 diagnosis, and transient ischaemic attack has been excluded

Migraine with aura

Recurrent attacks, lasting minutes, of unilateral fully reversible visual, sensory or other central nervous system symptoms that usually develop gradually and are usually followed by headache and associated migraine symptoms.

The aura is the complex of neurological symptoms that occurs usually before the headache of Migraine with aura, but it may begin after the pain phase has commenced, or continue to the headache phase. Visual aura is the most common type of aura, occurring in over 90% of patients with Migraine with aura, at least in some attacks. It often presents as a fortification spectrum: a zigzag figure near the point of fixation that may gradually spread right or left and assume a laterally convex shape with an angulated scintillating edge, leaving absolute or variable degrees of relative scotoma in its wake. In other cases, scotoma without positive phenomena may occur; this is often perceived as being of acute onset but, on scrutiny, usually enlarges gradually. In children and adolescents, less typical bilateral visual symptoms occur that may represent an aura. A visual aura rating scale with high specificity and sensitivity has been developed and validated. Next in frequency are sensory disturbances, in the form of pins and needles moving slowly from the point of origin and affecting a greater or smaller part of one side of the body, face and/or tongue. Numbness may occur in its wake, but numbness may also be the only symptom. Less frequent are speech disturbances, usually aphasic but often hard to categorize. When the aura includes motor weakness, the disorder should be coded as Hemiplegic migraine or one of its sub forms.

Aura symptoms of these different types usually follow one another in succession, beginning with visual, then sensory, then aphasic; but the reverse and other orders have been noted. The accepted duration for most aura symptoms is 1 hour, but motor symptoms are often longer lasting. Patients often find it hard to describe their aura symptoms, in which case they should be instructed to time and record them prospectively. The clinical Picture then becomes clearer. Common mistakes are incorrect reports of lateralization, of sudden rather than gradual onset and of monocular rather than homonymous visual disturbances, as well as of duration of aura and mistaking sensory loss for weakness. After an initial consultation, use of an aura diary may clarify the diagnosis.

Many patients who have migraine attacks with aura also have attacks without aura; they should be coded as both Migraine with aura and Migraine without aura.

Premonitory symptoms may begin hours or a day or two before the other symptoms of a migraine attack (with or without aura). They include various combinations of fatigue, difficulty in concentrating, neck stiffness, sensitivity to light and/or sound, nausea, blurred vision, yawning and pallor. The terms 'prodrome' and 'warning symptoms' are best avoided, because they are often mistakenly used to include aura. Migraine aura is sometimes associated with a headache that does not fulfill criteria for Migraine without aura, but this is still regarded as a migraine headache because of its relation to the aura. In other cases, migraine aura may occur without headache. Before or simultaneously with the onset of aura symptoms, regional cerebral blood flow is decreased in the cortex corresponding to the clinically affected area and often over a wider area. Blood flow reduction usually starts posteriorly and spreads anteriorly, and is usually above the ischaemic threshold. After 1 to several hours, gradual transition into hyperaemia occurs in the same region. Cortical spreading depression of Lea˜o is the likely underlying mechanism.

Systematic studies have demonstrated that many patients with visual aura occasionally have symptoms in the extremities and/or speech symptoms. Conversely, patients with symptoms in the extremities and/or speech or language symptoms almost always also experience visual aura symptoms at least during some attacks. A distinction between migraine with visual aura, migraine with hemiparaesthetic aura and migraine with speech and/or language aura is probably artificial, and therefore is not recognized in this classification. They are all coded as Migraine with typical aura Table 3.

Table 4: Diagnostic criteria of migraine with brainstem aura.

A. At least two attacks fulfilling criteria B-D
B. Aura consisting of visual, sensory and/or speech/language symptoms, each fully reversible, but no motor1 or retinal symptoms
C. At least two of the following brainstem symptoms:
1. dysarthria
2. vertigo
3. tinnitus
4. hypacusis
5. diplopia
6. ataxia
7. decreased level of consciousness
D. At least two of the following four characteristics:
1. at least one aura symptom spreads gradually over ≥5 minutes, and/or two or more symptoms occur in succession
2. each individual aura symptom lasts 5-60 minutes
3. at least one aura symptom is unilateral
4. the aura is accompanied, or followed within 60 minutes, by headache
E. Not better accounted for by another ICHD-3 diagnosis, and transient ischemic attack has been excluded.

Migraine with brainstem aura

Patients with aura symptoms arising from the brainstem are coded as Migraine with brainstem aura, but they almost always have additional typical aura symptoms Table 4.

Hemiplegic and familial hemiplegic migraine (FHM)

Hemiplegic migraine description is migraine with aura including motor weakness. Patients with Hemiplegic migraine have motor weakness, and this is classified as a separate sub form because of genetic and pathophysiological differences from migraine with typical aura. Such patients often have brainstem symptoms in addition.

New genetic data have allowed a more precise definition of Familial hemiplegic migraine (FHM) than was possible previously. Specific genetic subtypes have been identified: in FHM1 there are mutations in the CACNA1A gene (coding for a calcium channel) on chromosome 19; in FHM2 there are mutations in the ATP1A2 gene (coding for a K/Na-ATPase) on chromosome 1; and in FHM3 there are mutations in the SCN1A gene (coding for a sodium channel) on chromosome 2. There may be other loci not yet identified.

It has been shown that Familial hemiplegic migraine (FHM) very often presents with brainstem symptoms in addition to the typical aura symptoms, and that headache almost always occurs. Rarely, during FHM attacks, disturbances of consciousness (sometimes including coma), confusion, fever and CSF pleocytosis can occur Familial hemiplegic migraine (FHM) may be mistaken for epilepsy and (unsuccessfully) treated as such. FHM attacks can be triggered by (mild) head trauma. In approximately 50% of FHM families, chronic progressive cerebellar ataxia occurs independently of the migraine attacks Table 5.

Table 5: Diagnostic criteria of hemiplegic migraine.

A. At least two attacks fulfilling criteria B and C
B. Aura consisting of both of the following:
1. fully reversible motor weakness
2. fully reversible visual, sensory and/or speech/
language symptoms
C. At least two of the following four characteristics:
1. at least one aura symptom spreads gradually over ≥ 5 minutes, and/or two or more symptoms occur in succession
2. each individual non-motor aura symptom lasts 5–60 minutes, and motor symptoms last <72 Hours
3. at least one aura symptom is unilateral,
4. the aura is accompanied, or followed within 60 minutes, by headache
D. Not better accounted for by another ICHD-3 diagnosis, and transient ischaemic attack and stroke have been excluded.

Retinal migraine

Repeated attacks of monocular visual disturbance, including scintillations, scotomata or blindness, associated with migraine headache. Some patients who complain of monocular visual disturbance in fact have hemianopia. Some cases without headache have been reported, but migraine cannot be ascertained as the underlying etiology. Retinal migraine is an extremely rare cause of transient monocular visual loss. Cases of permanent monocular visual loss associated with migraine have been described. Appropriate investigations are required to exclude other causes of transient monocular blindness Table 6.

Chronic migraine

Headache occurring on 15 or more days per month for more than 3 months, which has the features of migraine headache on at least 8 days per month.

Complications of Migraine

Complicated migraine encompasses several individual clinical syndromes of migraine. Such a syndrome in children frequently presents with various neurological symptoms in clinical departments. The diagnosis of complicated migraine is basically clinical. The common differential diagnosis includes seizure, transient ischemic attack, and migraine like syndromes. Their relationships are complex. Migraine and seizure are common conditions and they may coexist in the same patient [21,23]. Migraine variants are common during infancy and early childhood in males by Teixeira et al. [24]. On the other hand, complicated migraines other than migraine variants are common in young females.

In practice, most complicated migraine presents with the isolated sign or symptom. The isolated manifestation may occur independently, together, or in the midst of the migraine attack [25]. However, some may present with more than one neurologic or medical manifestation. Persistent aura symptoms are rare but well documented. They are often bilateral and may last for months or years [5].

Table 6: Diagnostic criteria of retinal migraine.

A. At least two attacks fulfilling criteria B and C
B. Aura consisting of fully reversible monocular positive and/or negative visual phenomena (e.g. scintillations, scotomata or blindness) confirmed during an attack by either or both of the following:
1. clinical visual field examination
2. the patient's drawing (made after clear instruction)
of a monocular field defect
C. At least two of the following three characteristics
1. the aura spreads gradually over ≥ 5 minutes
2. aura symptoms last 5-60 minutes
3. the aura is accompanied, or followed within 60
minutes, by headache
D. Not better accounted for by another ICHD-3 diagnosis, and other causes of amaurosis fugax have been excluded.

Migrainous infarction

One or more migraine aura symptoms associated with an ischaemic brain lesion in the appropriate territory demonstrated by neuro imaging. Migrainous infarction mostly occurs in the posterior circulation and in younger women. A two-fold increased risk of ischaemic stroke in patients with migraine with aura patients has been demonstrated in several population-based studies. However, it should be noted that these infarctions are not migrainous infarctions. The mechanisms of the increased risk of ischaemic stroke in migraine sufferers remain unclear; likewise, the relationship between frequency of aura and the nature of aura symptoms denoting the increase in risk is unknown. Most studies have shown a lack of association between migraine without aura and ischaemic stroke.

Migraine aura-triggered seizure

Migraine and epilepsy are prototypical examples of paroxysmal brain disorders. Although migraine-like headaches are quite frequently seen in the epileptic postictal period, sometimes a seizure occurs during or following a migraine attack. This phenomenon, sometimes referred to as migralepsy, is a rare event, originally described in patients with Migraine with aura. Evidence for association with Migraine without aura is still lacking.

Episodic syndromes that may be associated with migraine

This group of disorders occurs in patients who also have Migraine without aura or Migraine with aura, or who have an increased likelihood to develop either of these disorders. Although historically noted to occur in childhood, they may also occur in adults. Additional conditions that may also occur in these patients include episodes of motion sickness and periodic sleep disorders including sleep walking, sleep talking, night terrors and bruxism.

Recurrent gastrointestinal disturbance

Recurrent episodic attacks of abdominal pain and/or discomfort, nausea and/or vomiting, occurring infrequently, chronically or at predictable intervals, that may be associated with migraine.

Table 7: Diagnostic criteria of cyclic vomiting syndrome.

A. At least five attacks of intense nausea and vomiting, fulfilling criteria B and C
B. Stereotypical in the individual patient and recurring with predictable periodicity
C. All of the following:
1. nausea and vomiting occur at least four times per hour
2. attacks last _1 hour and up to 10 days
3. attacks occur _1 week apart
D. Complete freedom from symptoms between attacks
E. Not attributed to another disorder.

Cyclic vomiting syndrome

Cyclic vomiting syndrome is characterized by recurrent, discrete, self-limited episodes of severe nausea and vomiting, interspersed with sign-free periods [26] Table 7.

Recurrent episodic attacks of intense nausea and vomiting, usually stereotypical in the individual and with predictable timing of episodes. Attacks may be associated with pallor and lethargy. There is complete resolution of symptoms between attacks. Cyclic vomiting syndrome is typically a self-limiting episodic condition occurring in childhood, with periods of complete normality between episodes. The cyclic nature is the hallmark, and is predictable. The clinical features of this syndrome resemble those found in association with migraine headaches, and multiple threads of research over the last years have suggested that cyclic vomiting syndrome is a condition related to migraine [5].

Abdominal migraine

An idiopathic disorder seen mainly in children as recurrent attacks of moderate to severe midline abdominal pain, associated with vasomotor symptoms, nausea and vomiting, lasting 2–72 hours and with normality between episodes. Headache does not occur during these episodes. Pain of Abdominal migraine is severe enough to interfere with normal daily activities. In young children the presence of headache is often overlooked. A careful history of presence or absence of headache must be taken and, if headache or head pain during attacks is identified, a diagnosis Migraine without aura should be considered.

Children may find it difficult to distinguish anorexia from nausea. Pallor is often accompanied by dark shadows under the eyes. In a few patients, flushing is the predominant vasomotor phenomenon. Most children with abdominal migraine will develop migraine headache later in life.

Abdominal migraine in children is a condition characterized by recurrent episodes of periumbilical pain severe enough to interfere with daily activities for which no organic cause is found. It is associated with vasomotor and gastrointestinal symptoms, including anorexia, nausea, vomiting, and pallor. It is also distinguished by an absence of symptoms in the intervening periods. Attacks typically last between one and 72 hours and are recurrent within a 12-month period.1

Table 8: Diagnostic criteria of abdominal migraine.

A. At least five attacks of abdominal pain, fulfilling criteria B–D
B. Pain has at least two of the following three characteristics:
1. midline location, periumbilical or poorly localized
2. dull or 'just sore' quality
3. moderate or severe intensity
C. During attacks, at least two of the following:
1. anorexia
2. nausea
3. vomiting
4. pallor
D. Attacks last 2-72 hours when untreated or unsuccessfully
treated
E. Complete freedom from symptoms between attacks
F. Not attributed to another disorder

While the prevalence of recurrent abdominal pain in children ranges from 9% to 15%, 2 the estimated prevalence of abdominal migraine as a subset of this is within the range of 2.4% and 4.1%.1,3 The mean age of onset is 7 years, although it has been described in infants and adults, with females affected more frequently than males [26] Table 8.

Benign paroxysmal vertigo

A disorder characterized by recurrent brief attacks of vertigo, occurring without warning and resolving spontaneously, in otherwise healthy children. Young children with vertigo may not be able to describe vertiginous symptoms. Onset is sudden (95%) [27]. With an expression of anxiety and fear on the face of the child, who may grasp a person standing nearby or any other support, or else may sway or refuse to stand. Ataxia may remain unnoticed, because some children will refuse to leave their beds. Infants may cry. Verbal children may report dizziness and nausea. Attentive parents may also report nystagmus. Neurovegetative signs may also occur, e.g., pallor, nausea, perspiration, photophobia, phonophobia, and unusual head positions. Vomiting is rather frequent, and may be vigorous [5,26]. Parental observation of episodic periods of unsteadiness may be interpreted as vertigo in young children. Posterior fossa tumours, seizures and vestibular disorders must be excluded [5] Table 9.

Benign paroxysmal torticollis

Recurrent episodes of head tilt to one side, perhaps with slight rotation, which remit spontaneously. The condition occurs in infants and small children, with onset in the first year. The child's head can be returned to the neutral position during attacks: some resistance may be encountered, but can be overcome. The differential diagnosis includes gastro-oesophageal reflux, idiopathic torsional dystonia and complex partial seizure, but particular attention must be paid to the posterior fossa and craniocervical junction where congenital or acquired lesions may produce torticollis. These observations

Table 9: Diagnostic criteria of benign paroxysmal vertigo.

A. At least five attacks fulfilling criteria B and C
B. Vertigo1 occurring without warning, maximal at onset and resolving spontaneously after minutes to hours without loss of consciousness
C. At least one of the following associated symptoms or signs:
1. nystagmus
2. ataxia
3. vomiting
4. pallor
5. fearfulness
D. Normal neurological examination and audiometric and vestibular functions between attacks
E. Not attributed to another disorder.

need further validation by patient diaries, structured interviews and longitudinal data collection. Benign paroxysmal torticollis may evolve into Benign paroxysmal vertigo or Migraine with aura (particularly Migraine with brainstem aura), or cease without further symptoms [5].

Benign paroxysmal torticollis (BPT) is a self-limiting disorder characterised by recurrent stereotypic attacks of unusual, sustained posture of the head and neck, during which the head tilts to one side secondary to cervical dystonia [28]. Such recurrent episodes of paroxysmal dyskinesia result in apparent torticollis or even retrocollis [29]. They are often accompanied by vomiting, pallor, irritability, ataxia, apathy and drowsiness [28,30]. The periodic episodes of BPT typically start in infancy and resolve by 5 years of age [29-31]. Neurologic examination, electroencephalogram (EEG) and brain imaging tests are usually normal in patients with BPT [29,31,32]. The etiopathogenesis of BPT is unknown. Many different underlying disorders involving vestibular and cerebellar structures, immaturity of brain, or even a channelopathy were proposed [33]. Possible channelopathy has been linked to mutation in the CACNA1A gene which induces a loss of channel function due to impaired gating by voltage and much lower current density [34,35]. Additionally, BPT has been associated with familial PRRT2 mutations [36] Table 10.

Management of Migraine Spectrum

The first essential step is to explain the condition to the child and his or her parents. Explanation and reassurance may be the only treatment needed in some cases. As with migraine, the best treatment is prevention. The clinician should search for avoidable trigger factors, such as stress, travel, prolonged fasting, irregular sleeping habits, exposure to glaring or flickering lights, and exercise. If trigger avoidance proves unsuccessful, then a clinical psychologist can provide cognitive therapy, relaxation programs with or without biofeedback, or other behavioral approaches to management. Alternative nondrug approaches to prophylaxis include simple dietary management, usually involving an avoidance of foods rich in amines or xanthine, together with any foods that the family suspects of triggering attacks. Food and symptom diaries are sometimes useful

Table 10: Diagnostic criteria of benign paroxysmal torticollis.

A. Recurrent attacks in a young child, fulfilling criteria B and C
B. Tilt of the head to either side, with or without slight rotation, remitting spontaneously after minutes to days
C. At least one of the following associated symptoms or signs:
1. pallor
2. irritability
3. malaise
4. vomiting
5. ataxia
D. Normal neurological examination between attacks
E. Not attributed to another disorder.
Notes:
1. Attacks tend to recur monthly.
2. Ataxia is more likely in older children within the affected age group.

in identifying triggers. Reducing anxiety may substantially lower the frequency of attacks [37].

During an attack, lying down in a quiet, darkened place and being left alone may provide relief for some children. Reassurance and rest may help more than medications. Drug therapy is often precluded by anorexia or vomiting, but simple oral analgesics can be tried, with or without metoclopramide or domperidone. Analgesic or antiemetic suppositories are also useful. Some authors successfully used both injected and nasal sumatriptan, but this treatment has not been subjected to a formal trial [38].

Drug prophylaxis should be restricted to children who have not responded to nondrug measures, and whose signs adversely affect their lives. Only pizotifen has been subjected to a double-blind, placebo-controlled trial. The use of propranolol and cyproheptadine has received support [39,40]. Other authors obtained good results with clonidine and sodium valproate [38].

Conclusion

The clinical features and prevalence of the spectrum of migraine of childhood are well-documented. The diagnosis relies on a careful semiologic analysis, mainly based on a parental description of manifestations. The family should be informed of the favorable prognosis.

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