Chapter 13
Management of Migraine and Other Headaches

Jens Böhmer and Alyssa A. LeBel

Abstract Headache is a common symptom in childhood and adolescence. Primary headaches, including migraine and tension-type, are most frequent, especially in adolescence, often occur together in an individual, and are readily differentiated from secondary headaches (such as traumatic, neoplastic, or infectious headache) by history and targeted physical examination. A prompt and definitive diagnosis of a primary headache disorder facilitates effective treatment. Migraine variants and "periodic syndromes" are more likely in children, including paroxysmal torticollis and vertigo, cyclic vomiting syndrome and abdominal migraine, and confusional migraine. A rare genetic syndrome, familial hemiplegic migraine, is associated with a neuronal channel defect, suggesting a role for sodium and calcium channel dysfunction in the pathophysiology of some migraine presentations. Chronic daily headache and analgesic overuse headache are increasingly recognized in pediatrics.

Treatment of all primary headaches include both pharmacologic and nonpharmacologic modalities, including education, lifestyle modification, cognitive-biobehavioral therapy, and adjustment of unrealistic expectations. Reports of

J. Böhmer, MD (✉)
Department of Pediatrics, Queen Silvia Children's Hospital, Sahlgrenska University, Gothenburg, Sweden
e-mail: jensbohmer@vgregion.se

A.A. LeBel, MD
Pediatric Headache Program, Children's Hospital Boston, Harvard Medical School, Boston, MA, USA

Chronic Pain Clinic, Children's Hospital Boston, Harvard Medical School, Boston, MA, USA

Pain Medicine, Children's Hospital Boston, Harvard Medical School, Boston, MA, USA

Department of Neurology, Children's Hospital Boston, Harvard Medical School, Boston, MA, USA

Department of Anesthesia, Children's Hospital Boston, Harvard Medical School, Boston, MA, USA

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controlled clinical trials have increased the choice of treatments for acute and chronic headaches in pediatrics. However, drug therapy for pediatric headaches remains primarily based on adult studies.

Keywords Pediatric headache • Migraine • Tension-type headache • Complicated migraine • Prophylactic treatment of migraine • Aura • Childhood periodic syndromes • Chronic daily headache

13.1 Introduction

13.1.1 General Introduction

Headache as a common complaint in childhood is a frequent cause of visit to the emergency department [1]. There has been a great effort to develop classification systems to help diagnose childhood headache (see Chap. 3); however, clear borders between the different headache entities do not exist in reality, especially in children. In fact, many children present with mixed forms of different primary headaches (i.e., tension headache and migraine). When migraine is diagnosed, it is of great importance to point out that cure is not a realistic aim. Migraine has usually come to stay, but the severity and frequency of the attacks can be strongly influenced by lifestyle changes and abortive and preventive medication [2].

Tension-type headache does not differ significantly in its presenting symptoms in childhood from those in adulthood and comprise bilateral headache of milder severity than migraine, without associated autonomic symptoms or aura. Migraine with aura was previously called “classic migraine.” It represents 20 % of all migraine cases and consists of two distinct phases, both of which can be present to a variable degree. The first is a depression of neuronal activity, spreading from back to front over the cortex in a slow pattern, 3 mm/min, combined with decreased blood flow. This is the aura that most commonly involves visual symptoms, both “plus symptoms” (sparkles, colors, hallucinations, micropsia) and “minus symptoms” (blurred vision, hemianopsia, transient blindness, blind spots) that the child may describe. The second phase is the headache with the same features as in migraine without aura.

Migraine without aura is more common and sometimes challenging to diagnose. The severity of headache, the family history, and associated symptoms (especially nausea and vomiting that are not typical for other primary headaches) as well as the relapsing course with attacks after symptom-free intervals help secure the diagnosis. Migraine with aura is easier to diagnose as primary migraine, but more difficult to differentiate from serious secondary headaches in the first attack, since the aura represents a neurological deficit. The absence of warning signs in the neurological examination; the reversal of aura symptoms, usually within minutes; and a positive family history help avoid unnecessary investigations, such as EEG and CT.

However, there is the infrequent possibility that migraine-like headache can be due to a serious underlying disorder (see Chap. 3). The importance of an accurate physical and neurological examination cannot be overemphasized.

13.1.2 Classification

Migraine is one of the primary headaches for which the International Headache Society has developed and revised a classification system (see Chap. 3) [3]. When used in children, it proves to be somewhat rigid; both regarding the times of an attack (that can be as short as 20 min in a younger child) and the mixed forms of headache that many young patients experience.

13.1.3 Epidemiology

Migraine affects even small children <5 years with a prevalence rate of approximately 2–4 %, with a slight male preponderance. During school years, the rates rise to about 10 % without sex differences. From adolescence into adulthood, there is a clear female predominance (at least 2:1), and migraine may affect 20–30 % of the population.

Migraine often persists beyond childhood; at least 50 % of pediatric migraineurs continue with their disease into late adulthood. Boys have a much higher likelihood to become migraine-free than girls.

It has been speculated whether children and adolescents with migraine show a different psychological profile; systematic reviews do, however, not report more psychological dysfunctioning or more psychiatric comorbidities in this group [4]. The most common finding in children and adolescents with migraine is that they tend to have little or nearly no leisure time that is not already scheduled.

13.1.4 Pathophysiology

13.1.4.1 Genetics

Migraine is a disease with a strong genetic background that puts the individual at risk for developing attacks. Twin studies indicate a 60–70 % genetic influence. Migraineurs with aura seem to have a higher genetic burden than those without aura. Usually one of the parents has or has had migraine (90 % of the cases); a negative family history should raise suspicion whether the diagnosis is correct. Mutations in several ion channels and ion pumps have been linked to certain migraine variants, such as familial hemiplegic migraine. Several of those channels are also found to be

mutated in epilepsy syndromes such as the Dravet syndrome, providing evidence for commonalities of migraine and epilepsy. The involvement of channels and pumps shows the importance of balanced neurotransmission. Polymorphisms play a substantial role, however, complicating the risk prediction for the individual. Environmental factors contribute significantly, necessitating that patients identify and avoid their personal risk factors. It is speculated that early-onset disease reflects increased genetic susceptibility to environmental triggers [5, 6].

13.1.4.2 The Attack

Migraine was classically believed to be primarily a vascular disease, but other theories have developed, with the theory of a cortical spreading neuronal depression explaining the aura phenomenon as the scientifically most recognized. Notably, the stereotyped, transient disturbance of brain function that an aura represents cannot be sufficiently explained by vascular theories alone. The aura resembles epileptic seizures, with the main difference being that the brain disturbance in a seizure spreads faster and lasts shorter. The theory of cortical spreading depression describes waves of depolarization that spread on the cerebral cortex in a creeping pattern, with a comparably low speed of about 3 mm/min. This wave, believed to be triggered by brainstem-derived circuits, usually starts in the occipital cortex and then spreads frontally, thus explaining the most common aura symptoms of the visual system. The neuronal and glial cells initially depolarize for seconds, as well, followed by neuronal silence that lasts several minutes. These neurophysiological changes may reflect the sensory symptoms of the aura. At the same time and probably initiated by those changes, there is vasoconstriction in the areas of aura, followed by vasodilatation as the aura subsides. This stimulates pain-sensitive structures within the vessels and initiates headache [7].

13.1.5 Triggering Factors

The factors that commonly can trigger a migraine attack are very diverse and may not always be completely identified during an initial consultation: it is more important to put the patient in charge of finding out patterns that worsen or improve their headache. Headache diaries are of great importance. Nevertheless, the most common trigger factors should be discussed and include external or internal stress (family, peer group, school), especially the time after a stressful period, e.g., after a school week, and physical exercise. Dietary factors include certain cheeses, chocolate, red wine and caffeine, and many more, including skipped meals. Changes in sleep pattern as well as too much or too little sleep can provoke attacks. Hormonal changes, most commonly menstruation, as well as minor head trauma are other common trigger factors. Dehydration as a trigger of migraine is common especially during summer. Lack of exercise or overtraining can worsen migraine.

13.2 Migraine

13.2.1 Presenting Symptoms of Migraine

13.2.1.1 Migraine with Aura

Factors that can help in distinguishing migraine with aura from stroke are the presence of "plus symptoms" (stroke is more commonly associated with deficits) as well as the slowly evolving course of aura. The aural symptoms usually repeat in a stereotyped fashion in the individual. Most commonly, aura resolves within minutes to a few hours, but symptoms of 24–72 h duration may occur. Aural symptoms can furthermore affect the sensory and/or motor system (hemiplegic/hemisensory migraine), the brainstem and cerebellum (basilar migraine), or the oculomotor nerves (ophthalmoplegic migraine). The aura can even disturb body image and time sense and lead to confusion and amnesia (Alice-in-wonderland syndrome/acute confusional migraine).

It may be preceded by a prodromal phase that can last for up to 3 days, during which the individual can sense an impending attack (behavioral changes, fatigue, and adephagia). Brain function during this period is reduced as measured by neuropsychological testing and functional imaging. Commonly, the aura subsides within 60 min, and headache develops as cerebral vasoconstriction is followed by dilatation. The headache is never maximal at onset but evolves in both quality (dull in the beginning, then pulsating) and severity. It is located around and behind the eyes, the forehead, and the temples. The younger the child, the more common that the headache is bilateral. Older children describe typical unilateral pain that can change side between attacks. A child experiencing a migraine attack has moderate to severe pain and associated symptoms (nausea, vomiting, photo/phonophobia), looks ill, and wants to lie down and sleep (which terminates the attack). Headache lasts between minutes in the young child, some hours in most patients, and up to 2 days in extreme cases. Migraine is not a disease of constant headache but of attacks that repeat and that can occur in clusters (e.g., during times of increased demands in school).

13.2.1.2 Migraine Without Aura

Initially called "common migraine," migraine without aura accounts for approximately 80 % of migraine cases. One individual may have episodes of both migraine with and without aura. It has to be emphasized that aura symptoms have to be specifically asked for, especially in the young child. In a typical attack of migraine without aura, a prodromal phase up to several days (the individual senses the forthcoming attack) is followed by headache that evolves slowly and then becomes more intense and pulsating. At the same time, the child looks sick and has associated symptoms such as nausea, vomiting, photophobia, and pallor. Exercise worsens the headache, and sleep terminates it. The headache can improve and then aggravate again during a longer attack.

13.2.1.3 Complicated Migraine

Hemiplegic/Hemisensory Migraine

Symptoms that antedate or accompany the onset of migraine symptoms (headache and/or vegetative symptoms such as pallor, nausea, vomiting, photophobia) include unilateral motor weakness (hemiplegia/hemiparesis), which may affect speech, and/or sensory symptoms (paresthesia, hyperesthesia, allodynia), which may persist hours after that the headache has disappeared. This headache variant has a strong heritance (autosomal dominant) and increases the risk for stroke. The treatment consists of NSAID/acetaminophen and antiemetics, as triptans (and ergotamine) are contraindicated due to their vasoconstrictive effects.

Basilar Migraine

Believed to derive from vertebrobasilar vasoconstriction, basilar migraine has concerning symptoms, such as (occipital) headache, diplopia, vertigo, tinnitus, or ataxia, dysarthria, altered consciousness, or syncope. Pathologies of the posterior fossa and the inner ear, as well as cardiac anomalies and intoxications, need to be ruled out.

Ophthalmoplegic Migraine

Ophthalmoplegic migraine is a very rare disease consisting of unilateral headache, paresis of the ipsilateral ophthalmic nerve (pupillary defects and ptosis), and possibly even monocular blindness. Sometimes the abducens or trochlear nerves are involved.

Acute Confusional Migraine

Acute confusion migraine is most common in very young children and presenting with acute episodes of confusion, unresponsiveness, dysarthria, and disorientation. Headache and/or nausea and vomiting can be present or absent.

Status Migrainosus

A migraine attack that last longer than 72 h is called status migrainosus in adults; according to the adapted criteria for children, this would imply duration of more than 48 h. However, some authors recommend referring to pediatric status migrainosus in an attack lasting more than 24 h. Intermittent disruption (sleep, medication)

is possible. Status migrainosus is commonly provoked by extensive use of over-the-counter analgesics, a fact that has to be taken into consideration when considering abortive treatment. When the aura lasts longer than expected, migraine infarction (stroke) needs to be excluded (see Chap. 15). If the headache of a migraine attack does not improve on simple analgesics and triptans, inpatient treatment with intravenous agents should be considered. There is no evidence that one agent would be superior to any other, treatment is dependent on local preferences. Ketorolac intravenously is used successfully in children as well as dihydroergotamine (DHE) (nasal, oral, and intravenously), as single drugs or in combination with oral antiemetics such as metoclopramide, even though newer studies favor the use of ondansetron, given the higher likeliness of extrapyramidal-motoric side effects (even life-threatening dystonic reactions are seen) of metoclopramide in children compared to adults. Ondansetron has neither any known interactions with other drugs (it is metabolized through several cytochrome P450 enzymes of the liver) and is not sedating. Another dopamine-receptor antagonist, prochlorperazine, has shown to be successful when used intravenously, with limitations of recurrence of headache and the risk for dyskinesias. Even intravenous dexamethasone has shown to be effective as an add-on treatment in adults with refractory migraine and could be taken into consideration. Opiates show limited effect in adults and are not recommended for use in children. Intravenous fluids are commonly given and are certainly indicated in cases of heavy vomiting, but there are no studies supporting the effect.

13.2.1.4 Childhood Periodic Syndromes

Formerly known as migraine equivalents, the childhood periodic syndromes are believed to be precursors to migraine, as such they appear in the International Classification of Headache Disorders. Headache is usually not present. Features they share with migraine are heredity, episodic presentation with acute illness between symptom-free intervals, as well as the therapeutic regime that stresses reassurance and avoidance of trigger factors. Acute and preventive medication used for more common migraines is also prescribed, yet on a strictly empiric level. Since the presenting age is younger, a high suspicion for secondary causes is needed and the diagnosis—as opposed to that of migraine—is one of exclusion, often requiring neuroradiology and EEG [4].

13.2.1.5 Benign Paroxysmal Torticollis of Infancy

Manifesting between 2 and 8 months of age with a female predominance, the child presents with episodic torticollis, i.e., tilting of the head to either side, sometimes accompanied by ataxia and vomiting as well as other dyskinetic symptoms as dystonic posturing of the trunk. Attacks can last hours to days and resolve usually before school age (Table 13.1).

Table 13.1 ICHD-II diagnostic criteria for benign paroxysmal torticollis of childhood

A. Episodic attacks, in a young child, with all of the following characteristics and fulfilling criterion B:
(a) Tilt of the head to one side (not always the same), with or without slight rotation
(b) Lasting minutes to days
(c) Remitting spontaneously and tending to recur monthly
B. During attacks, signs of one or more of the following:
(a) Pallor
(b) Irritability
(c) Malaise
(d) Vomiting
(e) Ataxia
C. Normal neurologic examination between attacks
D. Not attributed to another disorder

Table 13.2 ICHD-II diagnostic criteria for benign paroxysmal vertigo of childhood

A. At least five attacks fulfilling criterion B
B. Multiple episodes of severe vertigo, occurring without warning and resolving spontaneously after minutes to hours
C. Normal neurologic examination and audiometric and vestibular functions between attacks
D. Normal EEG

13.2.1.6 Benign Paroxysmal Vertigo of Childhood

This disorder usually presents at preschool age, equally in girls and boys, with sudden onset of ataxia, mostly manifesting with the child being scared or refusing to walk. Nystagmus and vegetative symptoms are common, especially vomiting. Benign paroxysmal positional vertigo needs to be ruled out (see Chap. 6). Episodes usually last seconds to minutes, rarely hours, and tend to occur in clusters. They may decrease in frequency and intensity and will eventually resolve within months to years (Table 13.2).

13.2.1.7 Abdominal Migraine

This entity presents with sudden-onset abdominal pain of non-colicky quality that is usually located around the umbilicus and that is accompanied by migraine-like vegetative symptoms such as pallor. Prodromal and/or aura-like phenomena occur. Headache is not present or at least not dominant. Anorexia is common; nausea and vomiting happen but to a lesser extent. The age of onset is school age with a female predominance. It is very uncommon to debut in puberty. Attacks occur with weeks to months in between. It is a diagnosis of exclusion. An important difference from inflammatory bowel diseases or recurrent abdominal pain syndrome is the absence of diarrhea and constipation. The child is otherwise and between the attacks healthy but may suffer from migraine headache as other family members typically do.

Table 13.3 ICHD-II diagnostic criteria for abdominal migraine

A. At least five attacks fulfilling criterion B–D
B. Attacks of abdominal pain lasting 1–72 h (untreated or unsuccessfully treated)
C. Abdominal pain has all of the following characteristics:
1. Midline location, periumbilical or poorly localized
2. Dull or "just sore" quality
3. Moderate or severe intensity
D. During abdominal pain, at least two of the following:
4. Anorexia
5. Nausea
6. Vomiting
7. Pallor
E. Not attributed to another disorder

Attacks tend to be provoked by trigger factors such as stress or lack of sleep. The attacks cease in most cases but can continue into adulthood. Most children with abdominal migraine later develop common migraine headache. Therapeutic strategies do not differ from those of migraine headache, with emphasis on avoidance of triggering factors, rest and/or oral analgesics during attacks, and careful identification of those in need of prophylactic treatment (Table 13.3).

13.2.1.8 Cyclic Vomiting Syndrome

This is an entity that can manifest in all ages but that is most often diagnosed at the age of 5, including a delay of 2–3 years due to diagnostic uncertainties. It consists of a repetitive and stereotyped (but with great individual differences) pattern of nausea and vomiting that is typically preceded by a prodromal phase of several hours, followed by severe vomiting (6 to >10/h) and signs of extreme discomfort (nausea, abdominal pain, pallor, photophobia, apathy). This phase lasts most commonly 24 h and is followed by a recovery phase of usually about 6 h until the patient is restored to full health. Important differences as compared with migraine headache attacks are the signs of a stress reaction during the vomiting phase (e.g., tachycardia, high blood pressure, low-grade fever, and neutrophilia). Also, the patient might not only be apathetic and sick but also oppositional, demanding, and irritated. Triggering events should be found, and the family history is usually consistent with migraine. Most children have no further episodes after 10 years of age, yet most ultimately develop migraine headache. The syndrome is very difficult to diagnose. There may be absence of findings on the general and neurological examination, a nonprogressive course, as well as negative results of laboratory studies and gastroenterologic procedures. A trial of prophylactic migraine medication is indicated. Of great importance is the presence of the prodromal phase that allows abortive medication with oral analgesics, a triptan, and/or antiemetics. During the attack, fluid loss due to excessive vomiting commonly requires intravenous replacement and control of serum electrolytes (Table 13.4).

Table 13.4 ICHD-II diagnostic criteria for cyclical vomiting

A. At least five attacks fulfilling criteria B and C
B. Episodic attacks, stereotypical in the individual patient, of intense nausea and vomiting lasting from 1 h to 5 days
C. Vomiting during attacks occurs at least 4 times/h for at least 1 h
D. Sign-free between attacks
E. Not attributed to another disorder

13.2.2 Diagnosis of Migraine

There are two main goals in diagnosing pediatric migraine: first, to give a diagnosis to patient and parents. This alone will guarantee a higher likeliness of appropriate treatment. Caregivers are commonly so worried about malignancies or stroke as the reason of their child's headache that they will not listen until they are reassured (which, unfortunately, sometimes even requires neuroimaging in absence of warning signs). Teenagers will not adhere to the designated treatment if they are not convinced by what their doctor explains to them.

Second, the patients requiring prophylactic treatment, including those in need of detoxification due to excessive use of over-the-counter analgesics, need to be identified and referred to appropriate centers. There is clear evidence showing that the earlier the diagnosis, the earlier the treatment, the better the outcome. See Chap. 3 for diagnostic criteria and differential diagnosis of headaches.

13.2.3 Recommended Treatment of Migraine

13.2.3.1 Abortive Treatment

Abortive treatment of migraine primarily consists of classical over-the-counter analgesics, mainly ibuprofen and acetaminophen, both of which have shown to be superior to placebo. Furthermore, there are migraine-specific drugs, namely, the triptans and ergotamine, as well as drugs that address migraine-specific comorbidities, i.e., nausea and vomiting. Only nasal sumatriptan has enough evidence to be recommended to the pediatric population in Europe (over 12 years). It is very important to keep in mind that migraine attacks tend to be shorter the younger the patient is—it is not uncommon that attacks in schoolchildren last for only 30–60 min, making medication difficult or unnecessary. At the same time, children in a migraine attack have an imperative need to sleep—putting them to bed terminates the attack (see Table 13.5) [9–13].

13.2.3.2 Prophylactic Treatment

The preventative treatment of migraine should be discussed early whenever there are signs and symptoms such as chronic or frequent and severe episodes of pain,

Table 13.5 Abortive treatment

Simple analgesics	
Acetaminophen	15 mg/kg po/pr/iv, max 60 mg/kg/day
Ibuprofen	10 (–15) mg/kg po/pr, max 40 mg/kg/day
Acetylsalicylic acid	>12 years: 500 (–1,000) mg po, max 3 times/day
Antiemetics	
Metoclopramide	<6 years: max 0.5 mg/kg/day
	6–14 years: 5–10 mg, max 3 times/day
	>14 years: 10–20 mg, max 3 times/day
Ondansetron	0.1 mg/kg iv (max 4 mg), max 8 mg/day
Migraine-specific analgesics	
Sumatriptan nasal spray	>12 years: 10 mg, max 20 mg/day

disruptive to the child's normal routine. As a general rule, the number of schooldays the child misses should not exceed one per month. Other reasons to consider prophylaxis are severe attacks (e.g., lasting >1 day), inefficiency or contraindications for abortive treatment (e.g., dyspepsia or hemiplegic migraine), and, most of all, analgesic overuse. The number of days when the individual medicates against migraine should not be more than 9 days/month for simple analgesics and 6 days/month for triptans. If those numbers are exceeded, withdrawal is warranted: this can include preventive medication as well as medical certificates for both child and caregiver to stay at home during the expected 2–3 weeks' time of initial worsening.

Once the headache and/or aura symptoms are considered to have a severe impact on the child's life and are not controlled by simple lifestyle changes (i.e., identification of trigger factors) and abortive medication, referral to a specialist in a tertiary center for childhood headache is warranted.

There are a number of medications that are commonly and successfully used for the prophylaxis of migraine, though none of them was initially developed for this purpose. Most belong to the groups of antihypertensive agents, antiepileptic drugs, and antidepressants. No agent is superior to another; it is more the individual that leads to the drug of choice: beta-blocking agents such as propranolol should be considered if no history of asthma is present and if the child does not tend to have hypotension (that relative contraindication is doubted by several authors, though). Antidepressant agents (e.g., amitriptyline) can help if migraine has negative effects on the mood and sleep. Valproate and topiramate, of the group of antiepileptic drugs, show good effects; the former is better if no problems with obesity exist, the latter tends to work better with pronounced aura and may be associated with weight loss.

The treatment has to be reevaluated regularly: efficacy cannot be expected within the first weeks, and a goal of complete resolution of migraine is unrealistic. Treatment is usually sustained over a period of 6–12 months but might have to be continued even longer.

However, nonpharmacologic treatments remain the mainstay of treatment for recurrent and chronic headaches, including regular sleep, a well-balanced diet, excellent hydration, and regular exercise. Attention must also be given to the patient's possible school and family stress and relationships with peers. Adjuvant

biofeedback, cognitive-biobehavioral therapy (CBT), physical therapy for neck and shoulder muscle spasm and general reconditioning, and massage therapy may be considered.

13.3 Other Types of Headaches

13.3.1 Tension-Type Headache

Tension-type headache is one of the primary headaches that presents with similar features in children as in adults, that is, bilateral headache of mild to moderate severity, of pressing, non-pulsating character that lasts between hours to days and that is not accompanied by classical migraine features such as nausea, vomiting, pallor, or aura phenomena. Stress is a common trigger and patients often have an increased tenderness on palpation of the neck and scalp muscles. See Chap. 3 for the diagnostic criteria. There is no good evidence for treatment options in children and adolescents, but simple over-the-counter analgesics are commonly used with good effect. Importance has to be given to the comorbidity with migraine and to the risk of abusive drug use.

13.3.2 Secondary Headaches

Secondary headache is defined as headache in the presence of another disorder known to be able to cause headache. There should be evidence of a close temporal relationship or another causal relationship to the headache; headache should be resolved or greatly reduced within 3 months by treating the underlying disorder.

Secondary headaches need to be recognized and often, reassurance is curative to the worried patients and their parents. Treatable underlying disorders need to be dealt with (see Chap. 3). Nevertheless, the headache itself needs to be taken care of with simple analgesics in adequate dosages, as long as no complicated headache like chronic daily headache is suspected.

13.3.2.1 Chronic Daily Headache Syndrome

There are different definitions of the chronic daily headache (CDH) syndrome in the pediatric population. A simple and feasible approach is to define every headache that occurs for more than 4 h a day, more than 15 days per month in at least a month as CDH. The International Classification of Headache provides criteria for four types of CDH: transformed migraine, chronic TTH, new daily persistent headache, and hemicrania continua. CDH comprises about one third of all headache patients referred to a pediatric neurologist (prevalence of 1 % in the general population). Most commonly, it occurs in teenage girls. The headache can be primary or

secondary (for the differential diagnosis, see Chap. 3), this chapter dealing with the primary type. It usually has the characteristics of a primary headache such as migraine or TTH, very often of both, and may interfere strongly with the patient's life (preventing school attendance). Most patients have a known recurrent headache disorder (migraine or TTH); an external factor such as analgesic overuse or stress (e.g., in the beginning of the new school year) or a (often rather mild) head trauma transforms it into CDH. Genetic factors may contribute to chronification and there is strong correlation with psychiatric diseases (depression and anxiety disorders), necessitating a multidisciplinary approach with drugs being only part of the treatment. Referral to a headache expert is always warranted. Management can require prophylactic drugs as well as an inpatient course (see status migrainosus). Girls, those presenting in a young age (<13 years) and those with a psychiatric disorder are at high risk for persistence and need to be followed throughout adolescence and later referred to an adult neurologist [14, 15].

13.4 Conclusion

Headache is very common in childhood and adolescence, even though visits for the primary reason of headache represent not more than 1–2 % of the visits to the ED. Most commonly, those patients suffer from a benign secondary headache. The absence of red flags in history and the physical examination supports refraining from unnecessary investigations. Of great importance is the recognition of those that really need to be taken care of: migraineurs and especially all those that already have developed a chronic daily headache [16]. Most commonly, the referral to a pediatric neurologist (or a pediatrician interested in headache) is the right choice, but in acute exacerbations of all different types of headache, an inpatient course might be necessary to step out of the vicious circle. The minute a primary headache disorder is suspected, responsibility of identifying triggering factors should be shared between doctor and patient, who also has to be advised to keep a diary of their headache including the intake of analgesics. It needs to be mentioned that many patients that seek to the ED for a primary headache have already had it for some days—including trials of different analgesics—representing a difficult-to-treat group per se.

Furthermore, the doctor has to have knowledge about the periodic syndromes of childhood (see above), all of which most probably represent migraine equivalents to prevent patients from years of investigations without proper treatment.

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