Chapter 46

Young Boy with Visual and Headache Symptoms After Soccer

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Case History

A 15-year-old boy came to our observation as an outpatient for three headache episodes of severe intensity, accompanied by photophobia, phonophobia, nausea, and vomiting, preceded by visual field deficits. The first visit was carried out after the third episode with characteristics similar to two previous events in the last week.

From the history, it emerged that the boy was an amateur soccer player and practiced twice a week. For three times in the last 4 weeks, immediately after a workout, the boy developed a visual disturbance described as a difficulty in clearly seeing images in the right hemifield of the right eye, with superimposed scintillating figures in the area of blurred vision. The flickering figures were described as little stars that leave a trail, moving from the center of the right hemifield of the right eye toward the periphery.

The visual disturbance gradually developed within 5 minutes and bothered the boy, who continued to perceive them while returning home by motorcycle. It took him 10 minutes to travel the few kilometers between the soccer field and home.

Once home, he began to complain, as in the previous two times, of a pulsating headache of severe intensity, bilaterally located in the frontoparietotemporal regions, associated with photophobia and phonophobia, nausea, and repeated episodes of vomiting, weakness, irritability, and dizziness. The visual disturbance disappeared 30 minutes after the start of head pain. The headache attack lasted 3 hours.

In the last attack, the headache did not respond to over-the-counter analgesics, as in the previous ones. In particular, the boy unsuccessfully took acetaminophen 500 mg for the first attack, nimesulide 100 mg for the second, and acetaminophen again for the third attack. Due to the headache, the boy laid down in bed and tried to rest, with a pillow pressed over the temples, in the dark and in silence. As in the previous attacks, the pain lasted 3 hours and was relieved after several vomiting episodes.

The boy recalled that about 1 year ago, he had lost consciousness for a few seconds, for which he underwent an electrocardiogram and echocardiogram, both normal. Moreover, 3 years ago, the parents remembered that the boy, while watching television, complained of blurred vision in both eyes for a few seconds, and for this reason underwent an eye examination, also normal.

His past medical history revealed no other relevant diseases. He received all required vaccinations; among the

childhood exanthems, sixth disease at age 3 years, measles at age 6 years, and mumps at age 7 years were reported. He did not suffer from allergies. At 8 years of age, he underwent surgery for correction of cryptorchidism. The only family history worthy of note was a maternal uncle with a childhood brain tumor and convulsive crises. No family history of headache was reported.

Questions on the Case

Please read the questions, try to answer them, and reflect on your answers before reading the authors' discussion.

- Do the visual disturbance, the headache, and the dizziness associated with repeated vomiting episodes in the young boy after the workout was done suggest a secondary headache diagnosis?
- If a secondary headache is excluded, are all the mandatory criteria satisfied for making a diagnosis of migraine with aura?
- Because the symptoms appeared only after soccer practice, could it be considered a potential trigger factor for migraine with aura episodes?
- Should a differential diagnosis with primary exertional headache be considered?
- Which kind of investigations are the most appropriate?
- Which kind of therapeutic approach is the most appropriate?

Case Discussion

Diagnosis

The general and neurologic examinations were normal. An eye examination did not reveal any abnormalities, which was the same as in previous examinations. Routine hematologic and biochemical tests, thyroid hormone, folate, and vitamin B_{12} levels, and an electrocardiographic examination were normal.

A headache diary was given to the boy to record the aura symptoms and their duration, the characteristics of headache attacks (location, intensity, quality of pain), and the preceding and accompanying symptoms.

During an observation period of 6 months, two other episodes were experienced by the boy after soccer practice and were recorded in the headache diary.

The attacks described by the patient at the time of the first visit have the characteristics of typical migraine with aura headache (1.2.1) according to the revised International Headache Society (IHS) criteria, and because at least two attacks occurred, a diagnosis of migraine with aura could be made at the time of the first visit, after the exclusion of an underlying disease mimicking a migraine disorder (Table 46-1).

The symptoms suggesting migraine as described by the patient were suggestive of this primary headache disorder, and the neurologic examination was normal. Nevertheless, in the past, the boy had presented with an episode of amaurosis and an additional episode of loss of consciousness, which were investigated with ophthalmologic and cardiologic examinations, respectively, but in neither case by a neurologic examination. Such past symptoms could suggest a secondary headache with migraine with aura-like characteristics, such as a transient ischemic event involving the posterior brain circulation, an arteriovenous malformation, a sentinel hemorrhage preceding a subarachnoid hemorrhage (SAH) due to a bleeding aneurysm, as well as partial idiopathic epilepsy with occipital waves.

A transient ischemic attack involving the posterior brain circulation, due to a vertebral artery dissection, could manifest itself after a physical effort, and in at least a quarter of cases, headache can occur. In these cases, the presence of headache associated with focal neurologic symptoms can mimic a migraine with aura attack infrequently. In this case, in fact, the onset of hemianopic disturbance and scintillating scotomatas followed by a severe intensity headache, accompanied three times by vomiting and vertigo, exclusively after physical activity lasting 2 hours in an adolescent, could also suggest a vertebral (or carotid) dissection.

An arteriovenous malformation should also be considered in the case of migraine attacks precipitated by exercise. Little evidence is available for a migraine with aura-like attack due to the presence of an arteriovenous malformation, whereas in the literature, there are reports of secondary cluster headache, short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT), and also chronic migraine precipitated by physical effort.

Sentinel hemorrhage preceding the occurrence of an SAH due to a bleeding aneurysm may sometimes manifest itself with migraine-like headache, often after physical exercise, without other relevant neurologic signs. It is therefore important to exclude this condition through appropriate examinations, in particular computed tomography scans and lumbar puncture, if necessary.

Although the visual deficits described by the patient are highly suggestive of a typical migraine aura, a partial benign occipital epilepsy should in any case be excluded. In this disorder affecting young children and adolescents, seizures begin with elementary visual symptoms (lamps, spots, lines or flashes, generally colored, in a visual hemifield), indicative of the contralateral primary visual cortical area involvement. Seizure is often followed by a

Table 46-1 Diagnostic Criteria of the International Headache Society for Selected Migraine Syndromes with Aura

1.2 Migraine with Aura

Previously used terms:
Classic or classical migraine, ophthalmic, hemiparesthetic, hemiplegic or aphasic migraine, migraine accompagnée, complicated migraine

Coded elsewhere:
13.17 Ophthalmoplegic "migraine"

Description:
Recurrent disorder manifesting in attacks of reversible focal neurologic symptoms that usually develop gradually over 5 to 20 minutes and last for less than 60 minutes. Headache with the features of migraine without aura usually follows the aura symptoms. Less commonly, headache lacks migrainous features or is completely absent.

Diagnostic criteria:
A. At least two attacks fulfilling criterion B
B. Migraine aura fulfilling criteria B and C for one of the subforms 1.2.1 to 1.2.6
C. Not attributed to another disorder

Note:
History and physical and neurologic examinations do not suggest any of the disorders listed in groups 5 to 12, or history and/or physical and/or neurologic examinations do suggest such disorder but is ruled out by appropriate investigations, or such disorder is present but attacks do not occur for the first time in close temporal relation to the disorder.

Comments:
The aura is the complex of neurologic symptoms that occurs just before or at the onset of migraine headache. Most patients with migraine exclusively have attacks without aura. Many patients who have frequent attacks with aura also have attacks without aura (code as 1.2 migraine with aura and 1.1 migraine without aura).

Premonitory symptoms occur hours to a day or two before a migraine attack (with or without aura). They include various combinations of fatigue, difficulty in concentrating, neck stiffness, sensitivity to light 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.

The majority of migraine auras are associated with headache fulfilling criteria for 1.1 migraine without aura. For this reason, the entity 1.2.1 typical aura with migraine headache has been singled out below. Migraine aura is sometimes associated with a headache that does not fulfil criteria for 1.1 migraine without aura, and in other cases, migraine aura may occur without headache. These two subforms are also now distinguished.

Aura with similar features has also been described in association with other well-defined headache types, including cluster headache; the relationships between aura and headache are not fully understood.

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 including an even wider area. Blood flow reduction usually starts posteriorly and spreads anteriorly and is usually above the ischemic threshold. After one to several hours, gradual transition into hyperemia occurs in the same region. Cortical spreading depression of Leão has been implicated.

Systematic studies have demonstrated that many patients with visual auras occasionally have symptoms in the extremities. Conversely, patients with symptoms in the extremities virtually always also suffer visual aura symptoms. A distinction between migraine with visual aura and hemiparesthetic migraine is probably artificial, and therefore, is not recognized in this classification. Patients with motor weakness are classified separately because of the dominantly inherited form, 1.2.4 familial hemiplegic migraine, and because of clinical differences. The genetic relationship between migraine with aura and familial hemiplegic migraine has not been established.

The previously defined syndromes, migraine with prolonged aura and migraine with acute-onset aura, have been abandoned. The great majority of patients with such attacks have other attacks that fulfil criteria for one of the subforms of 1.2 migraine with aura, and should be coded to 1.6.2 iprobable migraine with aura, specifying the atypical feature (prolonged aura or acute-onset aura) in parenthesis.

1.2.1 Typical Aura with Migraine Headache

Description:
Typical aura consisting of visual and/or sensory and/or speech symptoms. Gradual development, duration no longer than 1 hour, a mix of positive and negative features, and complete reversibility characterize the aura that is associated with a headache fulfilling criteria for 1.1 migraine without aura.

Diagnostic criteria:
A. At least two attacks fulfilling criteria B to D
B. Aura consisting of at least one of the following, but no motor weakness:
   1. Fully reversible visual symptoms including positive features (eg, flickering lights, spots, or lines) and/or negative features (ie, loss of vision)
   2. Fully reversible sensory symptoms including positive features (ie, pins and needles) and/or negative features (ie, numbness)
   3. Fully reversible dysphasic speech disturbance
C. At least two of the following:
   1. Homonymous visual symptoms and/or unilateral sensory symptoms
   2. At least one aura symptom develops gradually over ≥ 5 minutes and/or different aura symptoms occur in succession over ≥ 5 minutes
   3. Each symptom lasts ≥ 5 and ≤ 60 minutes
D. Headache fulfilling criteria B to D for 1.1 migraine without aura begins during the aura or follows aura within 60 minutes
E. Not attributed to another disorder

Notes:
1. Additional loss or blurring of central vision may occur.
2. History and physical and neurologic examinations do not suggest any of the disorders listed in groups 5 to 12, or history and/or physical and/or neurologic examinations do suggest such disorder but is ruled out by appropriate investigations, or such disorder is present but attacks do not occur for the first time in close temporal relation to the disorder.

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Table 46-1 Diagnostic Criteria of the International Headache Society for Selected Migraine Syndromes with Aura

Comments:
This is the most common migraine syndrome associated with aura. The diagnosis is usually evident after a careful history alone, although there are rare secondary mimics including carotid dissection, arteriovenous malformation, and seizure.

Visual aura is the most common type of aura, often presenting as a fortification spectrum; that is, 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 variable degrees of absolute or 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. 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 and face. Numbness may occur in its wake, but numbness may also be the only symptom. Less frequent are speech disturbances, usually dysphasic, but often hard to categorize. If the aura includes motor weakness, code as 1.2.4 familial hemiplegic migraine or 1.2.5 sporadic hemiplegic migraine.

Symptoms usually follow one another in succession beginning with visual and then sensory symptoms and dysphasia, but the reverse and other orders have been noted. Patients often find it hard to describe their symptoms, in which case they should be instructed in how to time and record them. After such prospective observation, the clinical picture often becomes clearer. Common mistakes are incorrect reports of lateralization of headache, of sudden onset when it is gradual, and of monocular visual disturbances when they are homonymous, as well as incorrect duration of aura and mistaking sensory loss for weakness. After an initial consultation, use of an aura diary may clarify the diagnosis.

1.2.6 Basilar-type Migraine

Previously used terms:
Basilar artery migraine, basilar migraine

Description:
Migraine with aura symptoms clearly originating from the brainstem and/or from both hemispheres simultaneously affected, but no motor weakness

Diagnostic criteria:
A. At least two attacks fulfilling criteria B to D
B. Aura consisting of at least two of the following fully reversible symptoms, but no motor weakness:
   1. Dysarthria
   2. Vertigo
   3. Tinnitus
   4. Hypacusia
   5. Diplopia
   6. Visual symptoms simultaneously in both temporal and nasal fields of both eyes
   7. Ataxia
   8. Decreased level of consciousness
   9. Simultaneously bilateral paresthesias
C. At least one of the following:
   1. At least one aura symptom develops gradually over ≥ 5 minutes and/or different aura symptoms occur in succession over ≥ 5 minutes
   2. Each aura symptom lasts ≥ 5 and ≤ 60 minutes
D. Headache fulfilling criteria B to D for 1.1 migraine without aura begins during the aura or follows aura within 60 minutes
E. Not attributed to another disorder

Note:
History and physical and neurologic examinations do not suggest any of the disorders listed in groups 5 to 12, or history and/or physical and/or neurologic examinations do suggest such disorder but is ruled out by appropriate investigations, or such disorder is present but attacks do not occur for the first time in close temporal relation to the disorder.

Comments:
Basilar-type attacks are mostly seen in young adults. Many patients who have basilar-type attacks also report attacks with typical aura (code for both disorders).

If motor weakness is present, code as 1.2.4 familial hemiplegic migraine or 1.2.5 sporadic hemiplegic migraine. Patients with 1.2.4 familial hemiplegic migraine have basilar-type symptoms in 60% of cases. Therefore, 1.2.6 basilar-type migraine should be diagnosed only when no motor weakness occurs.

Many of the symptoms listed under criterion B are subject to misinterpretation as they may occur with anxiety and hyperventilation.
Originally, the terms basilar artery migraine or basilar migraine were used, but since involvement of the basilar artery territory is uncertain (ie, the disturbance may be bihemispheric), the term basilar-type migraine is preferred.

Adapted from Headache Subcommittee of the International Headache Society, 2004.

postcritical headache, gastric discomfort, and vomiting, with either normal or only mildly impaired responsiveness. Electroencephalogram (EEG) features include normal background activity, but typical EEG findings are occipital spikes and waves.

Additional instrumental examinations were therefore carried out to exclude secondary headache disorders. Considering the possible disorders for a differential diagnosis, brain magnetic resonance imaging (MRI) and magnetic resonance angiogram (MRA) scans were also normal. Other investigations such as a transcranial Doppler, a Doppler of the cervical vessels, and an EEG were also normal. The negative findings of these examinations confirmed the clinical diagnosis of migraine with aura.

Other than the diagnosis of migraine with aura (code 1.2), a diagnosis of primary exertional headache (code 4.3) was considered, since all three migraine attacks occurred exclusively after the soccer practice. According to the revised IHS classification (Table 46-2), for the diagnosis of primary exertional headache, it is mandatory that headache occur only during or after physical exertion, but in the comments, it is clearly specified that exercise-induced migraine is coded in Chapter 1 (migraine).

Nevertheless, the relationship between the soccer practice and the migraine attacks experienced by the boy supports physical activity as a precipitating factor for his migraine with aura attacks. Although physical activity is frequently described as a trigger for migraine attacks, reports of this trigger exclusively for migraine with aura are lacking in the literature. It could be hypothesized that in a predisposed subject, sports could trigger the cortical hyperexcitability underlying migraine aura, and subsequently the neuronal and vascular mechanisms involved in head pain.

Another diagnostic consideration should be given to basilar-type migraine, coded as 1.2.6 in the revised IHS classification, within the subtypes of migraine with aura. In basilar-type migraine, aura should include at least two symptoms originating from the brainstem or from the simultaneous involvement of both hemispheres. The aura symptoms in the three attacks presented by our patient were, however, unilateral, and vertigo occurred during headache, and was not related to the visual aura symptoms. We therefore excluded this migraine with aura subtype.

Investigation

In the context of examinations that are routinely performed in our medical center to complete the diagnostic work-up of patients, further biochemical and neurotransmitter determinations included intracellular monocyte and erythrocyte magnesium, plasma and lympho/monocyte beta-endorphins, and plasma met-enkephalins. The patient underwent blood drawing at the second visit, after 2 months, and intracellular levels of magnesium and beta-endorphins were lower than the normal cutoff point obtained from a group of age- and sex-matched controls.

Although such biochemical information is not needed for the clinical diagnosis, the data obtained further support the diagnosis of migraine, considering the findings of reduced intracellular magnesium levels obtained in young and adult migraineurs, and reduced intracellular beta-endorphins, which have been found both in adults and young people affected by migraine.

If reduced beta-endorphin levels seem to be indicative of a dysfunction of the endogenous opioid systems, then reduced magnesium levels have been considered as a biochemical marker of a latent neuromuscular excitability in migraine. This was confirmed by the positive finding of multiplets on electromyogram (EMG) ischemic testing, supporting previous results obtained both in adult and young migraine patients with and without aura. When questioned further about the symptoms related to this condition, the boy reported that after soccer practice, he often easily experienced abnormal fatigue and blepharospasm. A battery of neuropsychiatric tests (Minnesota Multiphasic Personality Inventory-2, Blacky Pictures test, Children's Depression Inventory, anxiety scale) was also administered to document any comorbid psychiatric disorders, which were all excluded.

Management Strategies

The main management question to be considered in the case of our patient is whether the attacks can be completely abolished by avoiding the lone recognized trigger factor, the soccer practice. This is a pivotal question that is very important for the approach to the case and for the pathophysiologic implications. Unfortunately, we could not verify if avoiding prolonged exercise could induce the complete disappearance of migraine with aura attacks experienced by the patient. The patient refused, in fact, to

Table 46-2 Diagnostic Criteria of the International Headache Society for Benign Exertional Headache

4.3 Primary Exertional Headache
Previously used terms: Benign exertional headache
Coded elsewhere: Exercise-induced migraine is coded under 1.0 migraine according to its subtype.
Description: Headache precipitated by any form of exercise. Subforms such as "weightlifters' headache" are recognized.
Diagnostic criteria: A. Pulsating headache fulfilling criteria B and C B. Lasting from 5 minutes to 48 hours C. Brought on by and occurring only during or after physical exertion D. Not attributed to another disorder
Note: On first occurrence of this headache type, it is mandatory to exclude subarachnoid hemorrhage and arterial dissection.
Comments: Primary exertional headache occurs particularly in hot weather or at high altitude. There are reports of prevention in some patients by the ingestion of ergotamine tartrate. Indomethacin has been found to be effective in the majority of the cases. Headache described in weightlifters has been considered a subform of 4.3 primary exertional headache; because of its sudden onset and presumed mechanism, it may have more similarities to 4.2 primary cough headache.

Adapted from Headache Subcommittee of the International Headache Society, 2004.

suspend soccer practice to avoid the attacks. Had he agreed, and had his attacks stopped, then we would have been dealing with migraine attacks exclusively triggered by exertion.

None of the symptomatic drugs for migraine attacks within the family of analgesics and nonsteroidal anti-inflammatory drugs (NSAIDs) has been demonstrated to directly relieve aura symptoms. These drugs are sometimes not required for head pain following migraine aura, when attacks are infrequent, and pain is of short duration and mild. But in the case of our patient, severe headache and accompanying symptoms were so important as to justify symptomatic treatment. Considering that the over-the-counter drug acetaminophen and nimesulide, an NSAID, were ineffective for treating the attacks of our patient, we recommended oral acetylsalicylic acid 900 mg and oral metoclopramide 10 mg. This was partially effective, reducing the intensity of pain from severe to mild within 2 hours, and inducing a partial relief of nausea and vomiting in the same time period. After 2 months, we therefore decided to begin treating the attacks of migraine with aura experienced by our patient with sumatriptan nasal spray 20 mg, taking into account the efficacy and tolerability data obtained on migraine in adolescents. At the time that we saw this patient, the use of all triptans in Europe had not been approved for those under 18 years of age; therefore, to overcome this difficulty, we proposed a protocol to our Ethics committee on the use of triptans (both oral and nasal spray) in adolescents, which was accepted. Based on this protocol, we obtained a signed consent from both parents and the boy before beginning administration of the triptan.

Sumatriptan nasal spray administered immediately at the start of the headache pain, after the disappearance of the aura symptoms, demonstrated to be effective and reduced the intensity and the associated symptoms. It also reduced the duration of the headache to less than 30 minutes.

In the majority of cases, migraine with aura has a low frequency, which does not justify undertaking prophylactic treatment. However, based on the presentations of five attacks within a period of 1 month and the lack of efficacy of self-administered treatment, we began, in association with the combination symptomatic treatment of oral acetylsalicylic acid 900 mg and oral metoclopramide 10 mg, a daily prophylactic treatment with magnesium pidolate (1.5 g per day) for 2 months. The choice of this approach was supported by EMG evidence of a latent neuromuscular hyperexcitability and the demonstration of low intracellular levels of magnesium.

At the follow-up visits, an initial improvement of migraine was recorded, with a reduction of the attack frequency to less than one per month. Thereafter, the headache frequency increased upon suspension of the prophylactic treatment, while the symptomatic treatment became ineffective. A new prophylactic cycle was begun with calcium and magnesium supplementation, but after 15 days, the boy informed us that he experienced the attacks after each soccer practice, twice a week. With the consent of the parents and the boy, attack therapy with sumatriptan nasal spray 20 mg and sodium valproate 800 mg per day as prophylactic treatment was begun. The dosage of the sodium valproate was increased very gradually from 200 mg per day up to 800 mg tid (200 mg at breakfast, 200 mg at lunch, and 400 mg at dinner time) over 15 days. Currently, the boy experiences less than one attack per month with a successful response to the triptan.

Case Summary

- The patient was a 15-year-old boy who complained of attacks of migraine with typical aura exclusively after soccer practice.
- Investigations excluded a secondary headache.
- He was given acetylsalicylic acid, which was partially effective, followed by sumatriptan nasal spray, which effectively reduced head pain and accompanying symptoms. Given the proven effectiveness of triptans in juvenile headache, their use should be considered with caution, and informed consent is needed.
- Because of the frequency of migraine attacks, a prophylactic treatment with magnesium was no longer effective. A second preventive treatment was therefore started with sodium valproate, which significantly reduced migraine frequency and severity.

Differential Diagnosis: Secondary Disorders

Although the characteristics of attacks experienced by the boy fulfil the criteria of migraine with aura, the past history and the occurrence of migraine with aura exclusively after prolonged exercise suggested cerebrovascular disorders; in particular, internal carotid artery (ICA) disease and subarachnoid bleeding from an arteriovenous malformation or aneurysm.

Some aspects of this case should be considered and are examined in the following sections.

Isolated Neuro-ophthalmologic Symptoms as Sole Manifestations of Internal Carotid Artery Disease

According to recent data, exclusively neuro-ophthalmologic manifestations consisting of visual deficits and homonymous hemianopsia, which can be reversible, and a transient monocular blindness, were the sole presenting signs in 16% of ICA dissection and 9% of ICA thromboembolic

occlusion. The time lag between neuro-ophthalmologic disturbances and the onset of stroke can be variable, ranging from a few hours to several days.

In a young boy playing soccer, a minor unrecognized trauma could be considered as the putative cause of carotid or cervical dissection, but a spontaneous dissection, without evidence of a minor trauma, cannot be excluded. A thromboembolic event may rarely occur in a young boy, whereas it is more often responsible for isolated neuro-ophthalmologic symptoms as the initial clinical picture of carotid disease in older people.

Headache and neck pain are often present in ICA dissection as the only accompanying symptoms of neuro-ophthalmologic symptoms. The pain sometimes has migrainous features, but few reports are available concerning migraine with aura-like attacks.

The time interval between neuro-ophthalmologic symptoms and stroke onset may indicate that the process is still reversible, and the time interval could be sufficient for intervention. A prompt and early diagnosis is therefore crucial, and all appropriate investigations should be conducted quickly (transcranial Doppler and Doppler of the cervical vessels, MRI and MRA, and angiography).

Subarachnoid Hemorrhage

The classical presentation of an aneurysmal SAH is an acute-onset, severe headache associated with a stiff neck, photophobia, nausea, vomiting, and reduced level of consciousness to coma, and is easily differentiated from migraine. This catastrophic presentation is often preceded by a more minor hemorrhage that can signal the likelihood of a major rupture within hours, days, or weeks, and may be more difficult to diagnose.

In most series of patients with minor leaks related to SAH, headache, nausea, and vomiting are common, loss of consciousness is less common, and seizures or cranial nerve findings are rare. Less severe headache can be mistaken for a migraine or tension-type headache. The SAH headache may be in any location, may be localized or generalized, may resolve spontaneously, and may be relieved by analgesics and triptans. Often, exercise may precipitate headache (symptomatic exertional headache). In the case of migraine attacks exclusively precipitated by exertion, therefore, the patient should be evaluated for SAH, since early recognition and surgery might lead to improved outcome.

Diagnosis

After the exclusion of secondary disorders underlying migraine with aura attacks, a differential diagnosis should be made between migraine with aura and primary exertional headache.

Migraine with Aura

The characteristics of migraine aura and the number of attacks permit the diagnosis of typical aura with migraine headache (code 1.2.1) according to the revised IHS criteria.

Gradual development of visual aura, duration not longer than 1 hour, and complete reversibility of visual symptoms are in fact associated with headache fulfilling criteria B to D for migraine without aura (code 1.1).

Exercise as a Precipitating Factor for Attacks of Migraine with Aura

Physical activity has been recognized as a trigger factor for migraine attacks. No specific reports are available for attacks of migraine with aura exclusively precipitated by physical efforts. The fact that, in the boy, only prolonged exercise was responsible for the onset of attacks of migraine with aura requires the exclusion of primary exertional headache.

The frequency of this form of headache varies widely in population studies from 0.2 to 12%, depending on the study design and criteria, including both short-lasting and long-lasting attacks, sometimes with migraine features precipitated by exertion. This form should be distinguished from symptomatic exertional headache attacks secondary to intracranial disorders. Onset of the primary forms is more frequent under 30 years of age and is often present in childhood and adolescence. From the principal epidemiologic studies, the pain begins during exertion and is never considered as explosive. Headache is described as throbbing, of moderate to severe intensity, and lasting between several minutes and 2 days.

According to the revised IHS classification, primary exertional headache is a headache precipitated by any form of physical exertion; that is, specifically brought on during and immediately after physical exertion. The pain is bilateral, throbbing in quality at onset, and lasts from 5 minutes to 48 hours. A close temporal relationship between pain and physical exercise is present. There are no accompanying symptoms, and the disorder is not attributed to another underlying disease (in particular an SAH for the acute-onset form).

In the literature, there are several reports of exertion as a trigger for typical migraine. The headache does not resolve when the activity stops, and may occur minutes or hours into the activity or after cessation of the activity. In a study by Williams and Nukada, effort-triggered migraine was experienced by 9% of 128 subjects. Such headaches began in childhood and adolescence, with 15 years as the average age of onset. Aura was noticed by all, nausea by the majority, and vomiting and neck stiffness were frequent. Spontaneous migraine, which was not related to sport or exercise, was experienced by 55%, with a positive migraine history in 64%. The authors suggest-

ed that low oxygen tension may trigger effort headache by an as yet unknown mechanism.

Additional triggers for migraine could be heat, altitude, bright light, dehydration, low blood sugar, stress, and the use of certain athletic equipment, such as goggles for swimming, mouth guards, and tight helmets.

From a pathophysiologic point of view, it can be hypothesized that all these factors in predisposed individuals can precipitate neuronal instability and hemodynamic changes, as well as the trigeminal reflex underlying migraine attacks. In the case of migraine with aura, physical exertion can be the precipitating factor. Accentuated cortical hyperexcitability is believed to underlie aura symptom deficits, with the subsequent activation of the trigeminovascular system related to head pain occurring after the aura.

Based on the above considerations, exclusion of a secondary headache, and clinical history, the headache experienced by our patient can therefore be classified as an exercise-induced migraine with aura and adds further evidence to previous reports of so-called "footballer's migraine."

This patient is a candidate for triptan therapy for the acute attacks, although the optimal time to dose in migraine with aura remains controversial. Preventive antimigraine drugs such as magnesium and sodium valproate could be helpful to reduce the cortical hyperexcitability, which renders the subject more susceptible to exertion and additional triggers.

Recommendations

- In the case of a headache with migraine with aura characteristics exclusively precipitated by exercise, it is pivotal to exclude an underlying disorder responsible for the attacks.
- In a young boy, the differential diagnosis includes, in particular, a vertebral or carotid dissection, even in cases in which a minor trauma is not recognized, and an aneurysm or arteriovenous malformation.
- The visual aura symptoms should be differentiated from visual symptoms due to a benign occipital epilepsy.
- Removing trigger factors could be enough to reduce or eliminate migraine attacks.
- Treatment options include drugs effective on migraine headache such as NSAIDs; if no symptomatic drugs for migraine attacks have been able to relieve pain, triptans may be used with caution in young people under 18 years.
- The approval of the local Ethics Committees and a written informed consent from both parents and young patients are mandatory in Italy.
- When the trigger cannot be avoided and the attacks of migraine with aura are frequent, a prophylactic treatment can be prescribed. This includes magnesium supplementation to counteract neuronal hyperexcitability or an antiepileptic drug.
- Sodium valproate has been demonstrated to be effective in migraine in young patients.

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Editorial Comments

These authors delineate the key difference between an exercise-induced IHS migraine with typical aura and primary exertional headache: in the latter, headache occurs only during or after physical exertion, while in the former, exercise can be one of many triggers. They also point out the importance of doing a careful work-up in patients with exercise triggers, as there lurk many secondary headaches that can masquerade as exercise-induced migraine with aura-like symptoms, specifically vascular anomalies such as arteriovenous malformation or aneurysm. Combining the suspicions with an appropriate evaluation and using the new IHS criteria will allow a clinician to arrive at the correct diagnosis. Furthermore, this case in particular addresses the management of migraine in the adolescent and the problems that arise in trying to use new agents as well as limiting activities that are triggers. Finally, some young men develop blurred vision within a few minutes of being hit on the head by a soccer ball; the visual disturbance can be followed by a headache and pressure on the head, the classic "footballers' migraine."

Final diagnosis:

Adolescent typical aura with migraine headache, with exercise trigger