Advances in Pediatrics 62 (2015) 283–293
ADVANCES IN PEDIATRICS

Pediatric Headaches

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Keywords

• Migraine • Headache • Aura

Key points

- Pediatric headaches are common, and many may never require intervention by a health care provider.
- However, migraines can become more difficult to treat, especially if they become chronic daily headaches.
- Pediatric headache is a subjective and unique experience that requires attention to both psychological and physiologic components in diagnosis and treatment.

INTRODUCTION

Headaches are one of the most common types of pediatric pain. The frequency of headaches increases with age, ranging from 20% [1] in children younger than 5 years to 75% in children aged 15 years [2]. The frequency of migraines is less, averaging from 3.9% to 7%. Three other reports suggest that 4% to 20% of adolescent boys and 10% to 27% of adolescent girls will develop migraines [3]. Although the International Headache Society has adjusted its definition of migraine headaches over the year, the basic distinction between migraine headaches and tension headaches persists.

MIGRAINE DEFINITION

A migraine headache without aura is defined as multiple attacks (at least 4 or 5) of headaches with a temporal throbbing sensation, usually unilateral but sometimes bilateral. These headaches (especially in younger patients) are accompanied by nausea, vomiting, photophobia, and phonophobia and are made worse by movements. These attacks can occur at anytime (even waking the patient) and last from 1 hour (in younger patients) to 48 to 72 hours. Sleep can help

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0065-3101/15/$ – see front matter
http://dx.doi.org/10.1016/j.yapd.2015.04.006

ameliorate symptoms. Younger patients may have motion sickness, vomiting, and abdominal pain before developing head pain. Only about 15% of children have auras with their migraines. Auras are defined as unilateral reversible symptoms in visual, sensory, speech or language, motor, brainstem, or retinal areas that spread gradually over 5 minutes and last 5 to 60 minutes followed by a migraine headache. Patients can have 2 or more symptoms occurring in succession [4].

PATHOPHYSIOLOGY

Migraines seem to be due to a combination of inherited genetic susceptibility (60%–70%) and environmental factors. Insights into the responsible mechanisms and the interrelationship with neuronal components such as astrocytes have come from studies of familiar hemiplegic migraine, in which several responsible genes have been identified: CACNA1A, ATP1A2, and SCN1A [5–7]. Abnormalities in calcium metabolism are thought to be involved in some migraines.

There are 2 current explanations for migraine pain: the vasogenic theory and the neurovascular theory. The vasogenic theory is based on the observation that migraines have painful, distended, pulsing extracranial vessels during attacks. Stimulation of intracranial vessels has been shown to cause an ipsilateral migraine [8]. In addition, vasodilatory substances such as nitrates can cause headaches, whereas vasoconstrictive compounds such as ergotamines and caffeine can abort migraines. It is important that migraineurs have uniform exposure to caffeine (preferably none). Even though caffeine is vasoconstrictive, frequent exposure can trigger migraines because of rebound vasodilation.

In the neurovascular theory, the aura (if present) is due to an altered cerebral susceptibility to migraine attacks. Vascular changes observed are the result rather than the cause of the attack. Aura changes are seen as focal neurologic effects. The expanding neurologic effects covering many different areas of the brain are attributed to a spreading cortical depression [9]. Spreading cortical depression results from hyperpolarization followed by suppression and blood flow decreases [10]. Fluid changes in the cells also occur in the wake of the spreading cortical depression, increasing the pain.

Recently, PET imaging performed at the start of several migraines (without aura) has shown increased blood flow in an area in the ipsilateral brainstem, leading to speculation that this could be a migraine generator [11]. In these patients, areas usually associated with aura have shown decreased blood flow (visual cortex and auditory regions). Neocortex activation leads to release of nociceptive substances into the interstitial space. Released substances activate pain nerves and trigeminovascular fibers that surround pial vessels. As more nociceptive compounds accumulate, the trigeminal nerves are activated, along with C fibers. Fibers travel from the activated trigeminal ganglion to other areas of the brain involved in the headache responsible for the patients' perception of pain.