Expert Opinion

Triggering Migraine in a 20-Year-Old Athletic Male

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(CLINICAL HISTORY

A 20-year-old male presented with a 3-year history of paroxysmal headaches. All of the headaches except for the first one possessed a trigger, and these triggers have included playing tennis on a hot day, running inside, swimming for 2 hours, weight lifting, playing golf in Vail (after being there for 3 days), and sudden immersion of the head in cold water while kayaking. About 20 minutes after the triggering activity is completed, the patient has a visual aura, wherein he perceives a black spot in the central and sometimes peripheral visual field of both eyes that persists for 10 to 20 minutes. Twenty minutes later, he develops a severe, bitemporal, throbbing headache with nausea, light and noise sensitivity, and, about one-third of the time, vomiting. The headaches last 6 to 8 hours. The headaches occur every 2 to 3 months, and he generally can perform similar or identical physical activities without triggering a migraine.

Questions.—What is the difference between an exertional headache and a migraine triggered by physical activity? How often are migraines triggered by physical activity? Is sudden immersion of the head in cold water while kayaking a common precipitant? Can you distinguish between a primary migraine attack and acute mountain sickness? Is a 20-minute delay between the end of aura and the onset of headache common?

EXPERT COMMENTARY

This is a case involving phenomenology and susceptibility. The incidence of migraine at age 17 is approximately 8%, and at this age an annual frequency of four to six headaches associated with aura is not unusual or worrisome. The latency between the “trigger” and aura onset may reflect the time required for brain structures to activate biologically, with the most commonly cited example of this being the latency between nitroglycerin exposure and induced migrainous headache in research subjects. The latency reported here between the end of aura and the onset of headache is consistent with the International Headache Society (IHS) classification system, which requires that “Headache follows aura with a free interval of less than 60 minutes.” As is typical of such patients, this young man notes that a migraine may not result each time a given activity is undertaken (thus the continued presence of benign coital headache in the gene pool).

Is exertional headache different from migraine triggered by physical activity? On the surface, our patient’s activities appear similar: all involve a sudden or repetitive movement of the head and/or neck; some involve extensive overheating or continuous light exposure; and some occur in environments associated with increased headache risk, such as high altitude and low temperature. Controlled studies have demonstrated that during an attack, physical activity worsens migraine but not tension-type headache, but exercise is not a particularly common or consistent trigger for migraine.† More common triggers are stress, weather changes, missed meals, certain dietary components, and bright sunlight.‡,§ Although certain athletes

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may have a higher prevalence of migraine, the extracranial vasculature reacts equally to exercise and mental arithmetic, perhaps reflecting changes in central processing. Several studies have shown regular exercise to be preventive of migraine frequency and intensity.

Exertional headache, per se, is clearly defined: a) specifically brought on by physical exercise; b) bilateral and throbbing and may develop migrainous features in those susceptible to migraine (italics mine); c) lasts 5 minutes to 24 hours; d) is prevented by avoiding excessive exertion; and e) is not associated with any systemic or intracranial disorder. In one investigation of individuals suffering from “sports headache,” only 15% of the headaches reported were migraine. Thus, it appears possible to differentiate exertional headache from triggered migraine in susceptible individuals; clearly, the latter will be easier to establish by eliciting a history of migrainous headaches occurring at other times.

The presentation of acute mountain sickness and migraine can be very similar. Acute mountain sickness is characterized by headache (often pulsatile, usually bilateral, but may be unilateral) associated with at least one of the following: anorexia, nausea, or vomiting; fatigue or weakness; dizziness or lightheadedness; and difficulty sleeping. The onset is usually within 12 hours of arrival at altitude (usually above 8,200 feet), but may be delayed by several days. Given the apparent absence of these associated features, this patient’s history is more consistent with a diagnosis of migraine with aura triggered by altitude.

Headache triggered by sudden immersion is a fascinating phenomenon, and the provocation by weight lifting is of some concern. Immersion in water, cold or otherwise, invokes two reflexes in the central nervous system. The diving, or bradycardic, reflex involves autonomically mediated shunting of blood away from nonvital structures, thus increasing blood flow to the brain. It is unlikely that the brief period of immersion described in this individual induced this reflex. The trigeminal reflex, which can be provoked by even brief exposure of the face to water, involves excitation of the facial, vagal, sympathetic/parasympathetic, and trigeminal nerves and their respective nuclei. This cold water stimulus, like so many provocateurs that influence mucosal structures (eg, seasonal rhinitis), may be sufficient in the susceptible individual to trigger the upstream (no pun intended) cortical structures that generate migraine attacks.

Is exercise induction a marker for malignant causes of headache? Exertional headache, especially when associated with valsalva, has been associated with abnormalities in the posterior fossae or more generalized increases in intracranial pressure, and differentiating exertional migraine from episodic headache associated with increased intracranial pressure can be difficult.

My diagnosis is migraine with aura. Brain magnetic resonance imaging will exclude structural anomalies such as Chiari 1 or, less likely, a developmental tumor of the nervous system; the likelihood of finding an abnormality is low, but the utility of the test in ruling out a treatable cause of headache may justify the study. Indomethacin, isometheptine, ergots, and even triptans can be considered as a “stat” preventive, but attacks often occur unpredictably. This patient is a candidate for triptan therapy for the acute attacks, although the optimal time to dose in migraine with aura remains controversial.

Ultimately, the most interesting question in such cases is: why then? Clinically or subclinically, something must occur in the 24 to 48 hours prior to a triggered attack that “primes the pump,” preparing the brain for the acute migraine. Regardless, I think this patient has a benign primary headache disorder, and he should be able to use typical antimigraine drugs. I think he is safe, and can use the drugs effectively. Everything else is phenomenology and susceptibility.

REFERENCES