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When Exercise is a Pain in the Head

Rebecca D. Granger
Otterbein University, granger.rebecca@otterbein.edu

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Rebecca D. Granger, RN, LMT
Otterbein University, Westerville, Ohio

Introduction

Headache is one of the five most common chief complaints in the US, resulting in nearly 5 million visits to the ED (Lange, 2011). With great variance in quality, etiology, pathophysiology, and as a potential indicator of a serious underlying problem, it is critical that these headaches be accurately diagnosed, primarily for rapid identification of life threatening factors, but also to offer treatment and education specific to the particular type of headache the patient is experiencing. This will result in better outcomes for the patient through better control and will subsequently save health care dollars by reducing ED visits and unnecessary imaging.

Case Study

A 35 year old Caucasian woman presents to her primary care provider complaining of severe headache precipitated by strenuous exercise. Her exercise regime includes weight lifting and cardiovascular activities such as running, biking, and rowing. During the course of her work out she suddenly experiences a severe throbbing headache, along with nausea and dizziness. While the nausea and dizziness subside within an hour of the work out, the headache lasts for several hours. Denies aura or visual disturbances. States that headache is, "Just terrible pain-on both sides. I get it every time I push myself so hard." Vitals are: temp-98.4 degrees F, pulse-76, BP- 118/80, RR- 14. She has a BMI of 32.6, which puts her in the obese category. Otherwise she is in reasonably good health and takes no medications.

What is a possible diagnosis?

Primary exertional headache (PEH.)

What should be considered in the differential diagnosis? What needs to be ruled out immediately?

Subarachnoid hemorrhage and arterial dissection need to be ruled out immediately. Other differentials include: AV malformation, spontaneous intracranial hypotension, cervical disc disease, cerebral venous thrombosis, pheochromocytoma, cardiac cephalalgia, intracranial hypertension Type I Chiari malformation, tumor, and sinus infection. (Halker & Vargus, 2013),(Pascual, Gonzalez-Mandly, Martin, & Oterino, 2008), (Mayo Clinic Staff, 2012)

What tests will the provider likely order?

After taking a thorough history, if the provider is unable to make a diagnosis of another, more common type of benign headache, a CT without contrast, possibly followed by angio MRI and/or lumbar puncture, to differentiate between primary and secondary headache, may be ordered.. (Lange, 2011),(Pascual et al., 2008)

What is the appropriate treatment?

When possible, avoid the trigger by eliminating the provoking activities, or by reducing the intensity of the activity. If patient is unable or unwilling to modify activity, prophylactic drug therapy is indicated. Indomethacin is the drug of choice. Beta blockers such as propranolol and nadolol, other NSAIDS, ergotamine, and flunarizine, may also be used with good results. (Halker & Vargus, 2013), (Cutrer & Boes, 2004), (Pascual et al., 2008), (Robbins, 1998)

What information should be included in the patient education?

Discussion can include exercise modification and/or alternative activities, adequate hydration, avoidance of strenuous activity in hot weather or at high altitudes, and supportive therapies for existing PEH such as application of cold to the head and lying in a dark room. (Allena et al., 2010), (Robbins, 1998), (McCrory, 2000)

Underlying Pathophysiology and Significance

PEH falls under the heading of "Other Primary Headaches" (Group 4) in the International Classification of Headache Disorders, 2nd edition (ICHD-II,) along with primary cough headache, primary sexual headache, and idiopathic stabbing headache (Wang & Fuh, 2010). It is "defined by ICHD-II as a pulsating headache, typically triggered in hot weather or at high altitudes, lasting 5 min to 48 h, brought on exclusively during or after physical exertion, and not attributed to any other disorder (Halker & Vargus, 2013, p. 337). PEH has been recognized by the International Headache Society as a primary headache diagnosis since 1994. It is an uncommon, self-limited, and short lasting disorder that is precipitated by exertion and is frequently comorbid with migraine" (Halker & Vargus, 2013, p. 337) In fact, 46% of patients diagnosed with PEH also suffer from migraines (Allena et al., 2010). Not surprisingly, it occurs more frequently within the athlete population. "The only published study on sports related headaches found that headaches were reported by 35% of all respondents…effort exertion headaches were more common in women than in men. The sports noted to cause the problem included running/jogging, weights/gym, aerobics, and rugby football" (McCrory, 2000).

Whereas migraines have a prevalence of nearly 20% in the general population, primary exertional headache only has a prevalence of 0-2% (Pascual, Gonzalez-Mandly, Martin, & Oterino, 2008). As a result, this thunderclap look alike, has not been researched nearly to the extent that other headaches have been studied. As a result, no one conclusive explanation has been identified.

While no conclusive underlying pathophysiology of PEH has been determined, a number of viable theories have been postulated. PEH has been attributed to: "cervical muscle and ligament stretching and venous distention and arterial dilation associated with skeletal muscle activity" (Elkind, 2007, p. 76), "rapid rises in intra-abdominal pressure" (Cutrer & Boes, 2004, p. 142), magnesium deficiency, (though this is more specific to migraine) (Lange, 2011), "sudden hemodynamic changes" (Pascual et al., 2008, p. 261), metabolic and myogenic causes of impaired cerebrovascular autoregulation (Halker & Vargus, 2013), and "increased intracranial pressure" (Robbins, 1998, para. 3).

Another theory is that "patients with baroreflex impairment may have hypersensitive central trigeminal neurons (headache-prone), since baroreceptor activation has an antinociceptive effect" (Khurana, 2006, p. 1208).

Several studies have achieved more detailed, measurable, repeatable results, leading to very plausible hypotheses. Angiographic studies of exertional and sexual headaches have shown the presence of atrial spasm (McCrory, 2000). Another theory is that Valsava maneuver may be responsible. During resistance exercise, the Valsalva maneuver causes increased intrathoracic pressure which leads to an increase in systolic pressure which stresses cerebral arteries. This increased arterial pressure may dilate pain sensitive venous sinuses at the base of the brain, producing the exterional headache (Haykowsky, Eves, Warburton, & Findlay, 2003). Perhaps the most compelling theory is one that builds upon this and continues by suggesting a compounding factor, poor drainage of blood from the head. Valsalva maneuvers (VM) and the subsequent increased intrathoracic pressure reduce cerebral venous drainage. Exacerbating the problem in some individuals is the presence of internal jugular vein valve incompetence (IJVVI.) This leads to retrograde venous blood flow during VM and an increase in intracranial pressure in caused (Doepp et al., 2008). A study of individuals with PEH revealed a "significantly higher prevalence of IJVVI (70% of patients vs. 20% of controls)" (Allena et al., 2010, p. 528), yet this is not an all-encompassing explanation as not all patients with IJVVI get exertional headaches and PEH is considered self-limiting whereas IJVVI does not resolve spontaneously (Halker & Vargus, 2013).

Exertional Headaches, the Thunderclap Look-a-Like.

Implications For Nursing Care

Since PEH is a benign, self- limiting disorder, the paramount action of the provider must be to confirm that the headache is indeed primary and not secondary. In addition, appropriate treatment and patient education are goals of care.

Approximately one in five exertional headaches is secondary. (Pascual et al., 2008) (Halker & Vargus, 2013). The provider must first rule out these potentially lethal secondary causes before fully exploring other benign primary causes. Obtaining a thorough history will be a valuable tool in correctly diagnosing the headache; onset, location, duration, aggravating and alleviating factors, timing, severity, and additional symptoms should be discussed. CT without contrast is the preferred test to rule out secondary causes such as subarachnoid hemorrhage. If CT is negative, but a high suspicion of bleed exists, MRI and lumbar puncture may be performed (Lange, 2011). The provider must consider how to clinically differentiate primary and secondary headaches from one another. For instance, primary exertional headaches are usually described as throbbing, occurring during or after strenuous exercise, and usually affecting both sides of the head. Secondary exertional headaches may also have these characteristics, but in addition, may include vomiting, loss of consciousness, double vision, and neck rigidity (Mayo Clinic Staff, 2012). Treatment includes avoiding the trigger, or if patient is unwilling, by giving prophylactic indomethacin. It may be given on a scheduled basis, such as 25mg 3 times a day, or 50-75 mg given 1-2 hours before the triggering activity. Propranolol 1-2 mg/kg/day is another treatment option. (Halker & Vargus, 2013), (Pascual et al., 2008), (Robbins, 1998).

	Primary (Benign)	Secondary (Symptomatic)
Duration	Minutes to 2 days	1 - 30 days
Quality of Onset	Non-explosive throbbing	Sudden/Explosive
Nausea	Yes	Yes
Vomiting	No	Yes
Intensity	Moderate to Severe	Severe
Location	Unitlateral or Bilateral	Bilateral
Diplopia	No neurologic	Present in 1/3

Conclusion

Headaches are a common complaint, yet they vary greatly in severity, manifestation, and pathophysiology. The competent provider recognizes that headache is an incredibly common complaint, yet remains vigilant in ruling out potentially lethal secondary causes of headaches, especially those headaches which present in a similarly to a thunderclap headache. Additional objectives include seeking to improve patient outcomes and remaining fiscally responsible by correctly identifying and treating specific headache disorders.

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