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Pathophysiology of Migraine

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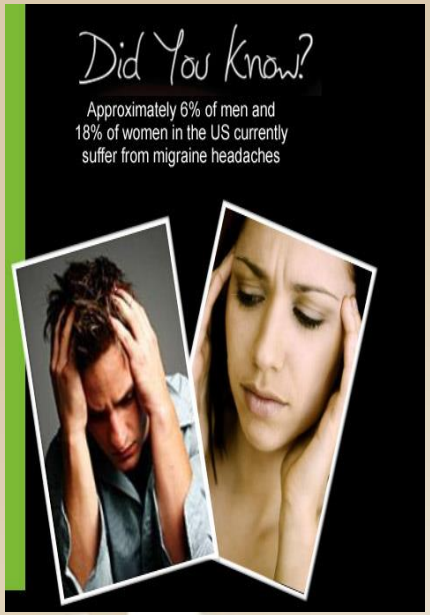


Migraine Elimination Centers (2009). *Did you Know?* [Photograph]. Retrieved from http://www.migraineeliminationcenters.com/images/side.jpg

Introduction

Migraine is a primary headache disorder and is ranked 19th among all diseases world-wide that cause disability (International Headache Society, 2013). There are more than 37 million Americans who experience migraine. Migraine is most commonly experienced by individuals between the ages of 15 and 55 and 70% to 80% of migraineurs have a family history of migraine (National Headache Foundation, 2014). Migraine is the most common headache disorder for which patients obtain medical care (Minen, Tanev, & Friedman, 2014, p. 1131). Estimated migraine healthcare costs in the United States are $19.6 billion annually (Da Silva & Tepper, 2012, p. 824). Migraine is often misdiagnosed and undertreated by healthcare providers. The pathophysiology of migraine is complex and not fully defined; however, progress in understanding migraine pathophysiology has advanced in the last 20 years. An understanding of migraine pathophysiology is necessary to provide acute and preventative treatment to individuals suffering from migraine.

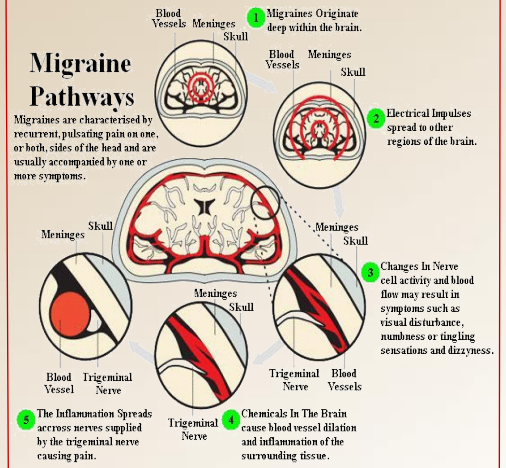
Signs and Symptoms

According to the International Classification of Headache Disorders (ICHD), a headache is diagnosed as a migraine when at least five headache attacks of the following criteria have occurred. The headache attacks last 4 to 72 hours. The headache includes two of the four following characteristics, unilateral location, pulsating quality, moderate or severe pain intensity and is aggravated by or causes avoidance of routine physical activity. Lastly, the headache is accompanied by at least one of the following, nausea or vomiting, photophobia or phonophobia (International Headache Society, 2013, p. 645). A migraine has two main subtypes, migraine without aura and migraine with aura. Migraine without aura is a clinical syndrome distinguished by a headache with precise features and associated symptoms. Migraine with aura is mainly characterized by the temporary focal neurological symptoms that precede or accompany the headache and it affects approximately one-fifth of migraine sufferers (International Headache Society, 2013).

A migraine attack consists of four phases that include premonitory (prodrome), aura, headache and resolution (postdrome); however, not every migraine sufferer experiences all four phases. The premonitory phase can occur hours or days before the headache and may be a warning that a migraine attack is forthcoming. The aura phase is the neurological symptoms that usually occur prior to the onset of the migraine attack. The headache phase is often the most debilitating part of the migraine attack and the headache pain can range from mild to severe. The resolution phase is the recovery period immediately following the headache which may last hours or days.

Common symptoms of the premonitory and resolution phase may include hypoactivity, hyperactivity, feelings of well being or euphoria, depression, sleep issues, fatigue, nausea, diarrhea, food cravings, increased thirst, increased urination, phonophobia, photophobia, concentration problems, aphasia, repetitive yawning, pallor or neck stiffness and pain (American Headache Society Committee for Headache Education website, 2014). The aura phase can consist of visual, sensory, speech/language, motor, brainstem and retinal disturbances . The symptoms may include flashing lights, wavy lines or spots, partial loss of sight, blurry vision, monocular blindness, allodynia, aphasia, auditory or olfactory hallucinations, decrease/loss of hearing, confusion, dizziness, hemiplegia, paresthesia, reduced sensation, hiccups, and neck pain (American Headache Society Committee for Headache Education website, 2014). The symptoms of the headache phase include unilateral headache pain, increased pain with physical activity, nausea and vomiting, diarrhea or constipation, dizziness, vertigo, confusion, hot flashes and chills, nasal congestion and/or runny nose, phonophobia, photophobia, osmophobia, depression, severe anxiety, and pain in the neck, eye, sinus and jaw areas (American Headache Society Committee for Headache Education website, 2014).

The National Headache foundation reports that less than half of all migraine sufferers have been diagnosed with migraine by their healthcare provider. Currently, there is no test to diagnosis migraine; therefore, establishing the history of the migraine-related symptoms and other headache characteristics in addition to a family history of similar headaches is used to diagnose a migraine headache (National Headache Foundation, 2014).



National Headache Foundation (2014). *The Pathways of Migraine* [Photograph]. Retrieved from http://www.headaches.org/education/Headache_Topic_Sheets/Migraine

Underlying Pathophysiology

Over the last several decades the understanding of migraine pathophysiology has advanced significantly; however, the exact migraine mechanisms are still unknown. Migraine is a complex brain disorder that is generally hereditary. The predominant neurovascular theory implies that the migraine sufferer's brain stays in a continual state of neuronal hyperexcitability. A condition that may be partly due to decreased intracellular magnesium and increased levels of lactic acid (Kabat & Sowka, 2011, p. 102). When certain trigger factors associated with a migraine such as bright lights, loud noise, certain odors or foods, missing or delaying a meal, hormonal changes, stress and depression are encountered a neurochemical imbalance develops. This imbalance may be perceived by patients as the premonitory phase of a migraine attack (Kabat & Sowka, 2011, p. 102). A depolarizing wave of neuron depression is eventually produced from ongoing conditions within the cortex and moves slowly across the cerebral cortex activating sensory nerves within the face and head (Kabat & Sowka, 2011, p. 102). Neuropeptides are released from the trigeminal nerve, initiating platelet aggregation and release of serotonin. Serotonin causes vasoconstriction and may be a contributing factor to the migraine aura (Kabat & Sowka, 2011, p. 102). Neuropetides including substance P and calcitonin gene-related peptide (CGRP) incite inflammation of the meningeal arteries which is thought to activate the headache and other symptoms associated with a migraine attack (Kabat & Sowka, 2011, p. 102-103).

Significance of Pathophysiology

An understanding of migraine pathophysiology provides clarification in the approach to treatment. Activation of the trigeminovascular system causes peripheral sensitization, the early stage of a migraine attack. Treatment during this phase of the migraine may terminate the attack fully (Ward, 2012, p. 758). If the attack progresses further to central sensitization the attack becomes much harder to treat (Ward, 2012, p. 758). Acute treatment of migraine with specific medication may not always resolve a migraine attack. For those individuals who experience frequent attacks or the attacks do not consistently respond to specific migraine treatment or medications, then preventative medications should be utilized to reduce migraine frequency and improve the response to the acute migraine medication.

Implications for Nursing Care

It is vital for healthcare professionals to have a good understanding of the pathophysiology of migraine, in order to accurately diagnose this neurological disorder and provide effective treatment. Healthcare providers will need to perform a general and neurological exam of the patient and obtain a detailed, dedicated headache history from the patient. Clinicians need to provide patient education regarding the disease process including the four phases of migraine, the triggering factors of migraine and the importance of identifying and avoiding these triggers as well as the appropriate use of medications and the significance of the timing of their use. A useful headache diagnosis tool for healthcare providers to utilize is the International Classification of Headaches guide.

Conclusion

Migraine pathophysiology is complex and not fully defined; however, scientific advances have allowed healthcare providers to find correlations between the clinical features of migraine and changes in the brain that have enhanced the diagnostic accuracy for migraine (Ward, 2012, p. 753). The question of whether migraine is primarily a vascular or neural abnormality has been essentially decided. Vascular changes represent an epiphenomenon and migraine is a disorder of brain excitability and sensory dysmodulation that causes head pain along with associated features (Ward, 2012, p. 753). Migraine is usually a hereditary brain abnormality. Understanding hereditary brain alterations allows for optimal migraine treatment and for progression of knowledge concerning the underlying central nervous system abnormalities that make up migraine (Ward, 2012, p. 753).

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