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Unconventional Therapy on Headache from Anatomy and Physiology Standpoint

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Abstract

This article discusses applied pharmacotherapy related to headaches and Gout. Headache is one of the frequently reported subjective complaints. Based on the causes, they are classified as primary headaches and secondary headaches. The aim of this paper is that students can know the definition of headache, know the classification of panic pain, know the anatomy and physiology of headache, know the prevention of headaches and adjunctive therapy and know the management for headaches.

Keywords: Pain, Headache, Physiology

Introduction

Pain is a common complaint in the clinic, although the term "pain" seems difficult to define. Each person's perception will be different, because this complaint comes from a person's subjective experience that is difficult to measure. Individual reactions and attitudes to identical stimuli that cause pain will be different. Therefore, the examining doctor is expected to be on the task of obtaining as complete information as possible from the patient and must also be able to imagine how the patient reacts to his pain.

Headache or cephalalgia is a discomfort in the entire head area. Headache is one of the frequently reported subjective complaints (Fendrich et al., 2007; Anttila et al., 2002; Zanchin et al., 2007; Kelman & Rains, 2005). Based on the causes, they are classified as primary headaches and secondary headaches. Primary headaches are headaches with no clear anatomic or structural abnormalities, namely migraines, tension type headaches, cluster headaches and other primary headaches (Brennan & Charles, 2009) Secondary headaches are headaches that clearly have anatomical and structural abnormalities and are chronic progressive in nature, including non-vascular disorders (Chaudhry & Friedman, 2015; Diener et al., 2010). This headache is caused by vasoconstriction of blood vessels leading to the brain caused by an increase in serotonin levels and then vasodilation According to the World Health Organization (WHO) headache is usually felt repeatedly by sufferers throughout their life. Approximately in one year 90% of the world's population experience at least one headache. The prevalence of headache in children and adolescents is increasing over time. It is estimated that the headache experienced will persist in adulthood with a relatively high percentage (about 50%) of cases.

Headache, which in common people is often referred to as a headache, dizziness and others, is pain or discomfort in the entire area of the head with the lower border from the chin to the back of the head (occipital area and parts of the head). nape). Headaches are classified into primary headaches and secondary headaches. Primary headaches are headaches where there is no clear anatomical or structural abnormality or the like. Meanwhile, secondary headaches are headaches where there are obvious anatomical or structural abnormalities or the like. Chronic progressive indicates the possibility of secondary headaches, namely headache more than three months, which increases in severity, frequency and duration and may be accompanied by the

appearance of neurological deficits other than headache (Jan, 2007; Sahai-Srivastava et al., 2012; Sharav & Benoliel, 2008).

Based on The International Classification of Headache Disorders, issue 2 of 2004 (ICHD-2), the classification of headaches is divided into; (1) Primary headaches, including migraine, tension type headaches, cluster headaches and other trigeminal-autonomic cephalgia, other primary headaches; (2) Secondary headaches, including headaches related to head and / or neck trauma, headaches related to cranial or cervical vascular disorders, headaches related to intracranial non-vascular disorders, headaches related to substance or withdrawal, headaches related to infection, headaches related to homeostatic disorders, headaches or vascular pain associated with cranium, neck, eye, ear, nose, sinuses, teeth, mouth, or other facial or cranial structures, headaches related to psychiatric disorders, cranial and central neuralgia causing facial and other headaches, cranial neuralgia , primary or central facial pain.

Headache Anatomy and Physiology

Anatomy

Headache is affected by the trigeminocervical nucleus which is an important nociceptive for the head, throat and upper neck. All nociceptive afferents of the trigeminal, facial, glossopharyngeal, vagus, and C1 - 3 nerves amify in the gray matter of this area. The trigeminocervical nucleus consists of three parts, namely the pars oralis which is associated with the transmission of discriminatory tactile sensations from the orofacial region, the interpolaris pars which is associated with the transmission of discriminatory tactile sensations such as toothache, the caudal pars associated with nociceptive transmission and temperature. There is an overlapping of the ramification process in this nucleus such as afferents from C2, apart from amifying to C2, also amifying to C1 and C3. In addition, C3 afferents will also amify to C1 and C2. This is what causes referred pain from the head and upper neck.

Referred pain is usually present in the occipital and fronto orbital regions of the head and rarely is the area innervated by the maxillary and mandibular nerves. This is caused by the afferents of these nerves that are not or only slightly extending caudally. Another case with the ophthalmic nerve of the trigeminus. This nerve afferents extend to the caudal pars. The trigeminal nerve consists of 3, namely V1, V2, and V3. V1, ophthalmicus, innervates the orbital and eye areas, frontal sinus, dura mater of the cranial fossa and falx cerebri and blood vessels associated with this dura mater. V2, maxillary, innervates the nasal area, paranasal sinuses, upper teeth, and dura mater of the medial cranial fossa. V3, mandibular, innervates the dura mater part of the medial cranial fossa, mandible and teeth, ears, temporomandibular joints and chewing muscles (see figure 3). In addition to the trigeminal nerve, there are cranial nerves VII, IX, X which innervate the external auditory meatus and thymfani membrane. The IX cranial nerves invade the middle ear cavity, besides the IX and X cranial nerves innervate the pharynx and larynx. The cervixes involved in headache are C1, C2, and C3. The dorsal ramus of C1 invades the suboccipital triangle muscles - superior obliquus, inferior obliquus and major and minor posterior rectus capitis. The dorsal ramus of C2 has lateral branches that enter the posterior superficial neck muscles, longissimus capitis and splenius while its large medial branches become the greater occipital nerve. This nerve surrounds the lower periphery of the inferior obliquus, and returns to the top and back via the semispinal capitis, where it supplies and enters the scalp through an arch surrounded by the superior nuchal line and the aponeurosis of trapezius. Through the occiput, this nerve will join the lesser occipital nerve which is a branch of the cervical plexus and reaches the scalp through the posterior periphery of the sternocleidomastoid. The dorsalis ramus of C3 provides lateral branches to the longissimus capitis and splenius. This ramus forms 2 medial branches. The medial superficial branch is the

third occipital nerve that surrounds the lateral and posterior zygapophysial C2-3 joints. The area sensitive to headache can be divided into 2 parts, namely intracranial and extracranial. Intracranial namely venous sinuses, cerebral cortex veins, basal arteries, dura mater anterior, and middle fossa and posterior fossa. Extracranial namely the blood vessels and muscles of the scalp, parts of the orbit, mucous membranes from the nasal and paranasal cavities, middle and outer ear, teeth, and gums. Whereas the areas that are not sensitive to pain are the parenchyma of the brain, the ventricular ependima, and the choroid plexus.

Physiology of Headaches

Pain is a protective mechanism that can occur at any time if any tissue is damaged, and through this pain, an individual will react by moving away from the pain stimulus. Pain begins with the stimulation of the pain receptors by a pain stimulus. Pain stimuli can be divided into three, namely mechanical, thermal, and chemical. Mechanically, muscle spasm is a common cause of pain because it can result in cessation of blood flow to tissues (tissue ischemia), increased tissue metabolism and direct stimulation to mechanically sensitive pain receptors.

Thermally, the pain caused by high temperatures does not correlate with the amount of damage that has occurred but is correlated with the speed at which tissue damage occurs. This also applies to other non-thermal causes of pain such as infection, tissue ischemia, tissue bruising, etc. At a temperature of 45 C, the tissues in the body will experience the damage found in most of the population.

Chemically, there are several chemicals that can stimulate pain such as bradykinin, serotonin, histamine, potassium ions, acids, acetylcholine, and proteolytic enzymes. Two other substances identified are prostaglandins and substance P which work by increasing the sensitivity of free nerve endings. Prostaglandins and substance P do not directly stimulate the pain. Of the various substances that have been stated, bradykinin has been known to be the main cause of causing severe pain compared to other substances. Increased potassium ion levels and increased local proteolytic enzymes are proportional to the intensity of the pain that is felt because these two substances can make the plasma membrane more permeable to ions. Tissue ischemia also includes a chemical stimulus because in the ischemia state there is a buildup of lactic acid, bradykinin, and proteolytic enzymes.

Pain can be divided into two, namely fast pain and slow pain. Fast pain, acute pain, is pain that is felt within 0.1 s after the stimulus is given. This pain is caused by the presence of mechanical and thermal stimuli. This pain signal is transmitted from the peripheral nerves to the spinal cord via the Aδ fiber with a speed of up to 6 - 30 m / s. The neurotransmitter that may be used is glutamate which is also an excitatory neurotransmitter that is widely used in the CNS. Glutamate generally only has a duration of action of a few milliseconds. Meanwhile, slow pain, chronic pain, is pain that is felt more than 1 second after the stimulus is given. This pain can be caused by the presence of mechanical, chemical and thermal stimuli but the most frequent stimulus is a chemical stimulus. This pain signal is transmitted from the peripheral nerves to the spinal cord via the C fiber with a speed of up to 0.5 - 2 m / s. A possible neurotramitter is substance P.

Migraine

According to the International Headache Society (IHS), migraine is a headache with pain attacks lasting 4 - 72 hours. Pain is usually unilateral, throbbing in nature, moderate to severe pain intensity and exacerbated by activity, and may be accompanied by nausea, vomiting, photophobia and phonophobia.

Etiology and Risk Factors for Migraine

The etiology of migraine is as follows: (1) hormonal changes (65.1%), decreased concentrations of estrogen and progesterone in the luteal phase of the menstrual cycle, (2) food (26.9%), vasodilators (histamine such as red wine, sodium nitrate)), vasoconstrictors (tyramine such as cheese, chocolate, caffeine), food additives (MSG), (3) stress (79.7%), (4) sensory stimuli such as bright light (38.1%) and a strong odor, both pleasant and unpleasant, (5) physical factors such as excessive physical activity (sexual activity) and changes in sleep patterns, (6) changes in the environment (53.2%), (7) alcohol (37.8%) , (7) smoking (35.7%).

Migraine risk factors are family history of migraine, women, and young age; (1) Epidemiology of Migraines, Migraines occur in nearly 30 million people in the United States and 75% of them are women. Migraine can occur at any age but usually appears at the age of 10-40 years and the incidence rate decreases after the age of 50 years. Migraine without aura is more often compared to migraine with aura with a percentage of 9: 1; (2) Classification Migraine, migraine can be classified into migraine with aura, without aura, and migraine chronic (transformed). Migraine with aura is migraine with one or more reversible auras indicating cerebral cortical dysfunction and / or without brainstem dysfunction, at least one aura develops over 4 minutes, the aura does not last more than 60 minutes, and the headache follows. aura in free-time intervals does not reach 60 minutes. Migraine without aura is migraine without classical aura, usually bilateral and affected periorbital. Chronic migraine is episodic migraine whose clinical appearance can change months to years and develop into chronic headache syndrome with daily pain; (3) Migraine pathophysiology, there are various theories explaining the occurrence of migraine. Vascular theory, the presence of vasospasm disorders causes the brain blood vessels to constrict resulting in brain hypoperfusion that starts in the visual cortex and spreads forward. Frontal spread continues and causes a headache phase to begin. The theory of cortical spread depression, where in migraine people the nerve threshold value decreases so that it is easy to excite neurons then occurs short-lasting wave depolarization by potassium-liberating depression (decreased potassium release) causing prolonged periods of neuronal depression. Furthermore, there will be a spread of depression which will suppress neuronal activity as it passes through the cerebral cortex.

Neovascular theory (trigeminovascular), the presence of vasodilation due to NOS activity and NO production will stimulate the trigeminal nerve endings in the blood vessels, releasing CGRP (calcitonin gene related). CGRP will bind to receptors in meningens mast cells and will stimulate the release of inflammatory mediators, causing inflammation of neurons. CGRP also acts on the cerebral arteries and smooth muscle which results in increased blood flow. In addition, CGRP will act on post junctional sites, second order neurons that act as transmission of pain impulses. The sympathetic nervous system theory, activation of this system will activate the locus sereleus resulting in an increase in epinephrine levels. In addition, this system also activates the dorsal rafe nucleus, resulting in an increase in serotonin levels. Increased levels of epinephrine and serotonin will cause constriction of blood vessels and then decrease blood flow in the brain. Decreased blood flow in the brain stimulates the trigeminovascular nerve fibers. If the blood flow is reduced then an aura can develop. If there is a decrease in serotonin levels, it will cause dilation of intracranial and extracranial blood vessels which will cause headaches in migraine headaches.

Migraine Diagnosis

History history and enforced if there are typical signs of migraine. The IHS diagnostic criteria for migraine with aura require that there must be at least three of the following four characteristics: (1) migraine with one or more reversible aura indicating cerebral cortical

dysfunction and / or without brainstem dysfunction, (2) at least one aura present. formed gradually over 4 minutes, (3) the aura does not last more than 60 minutes, (4) the headache follows the aura in free intervals not reaching 60 minutes. The IHS diagnostic criteria for migraine without aura require that there must be at least five headache attacks in a lifetime that meet the following criteria: (a) lasts 4 - 72 hours, (b) meets at least two of: (1) unilateral, (2)) a throbbing sensation, (3) moderate to severe intensity, (4) exacerbated by activity, (3) nausea, vomiting, photophobia and phonophobia may occur.

Migraine Support Examination this includes tests to rule out other diseases (if indicated) are imaging (CT scan and MRI) and lumbar puncture. Migraine diagnostic differentials; Differential diagnosis of migraine is arteriovenous malformation, cerebral aneurysm, glioblastoma, encephalitis, meningitis, meningioma, lupus erythematosus syndrome, polyarteritis nodosa, and cluster headache. Migraine Therapy; The goals of migraine therapy are to aid in psychological and physiological adjustment, prevent further extracranial dilation, inhibit the action of humoral media (eg serotonin and histamine), and prevent vasoconstriction of intracranial arteries to improve cerebral blood flow.

Acute Migraine Therapy

NSAIDs

The combination of aspirin, acetaminophen and caffeine has been approved for use by the FDA (Food Drug Administration) as the drug of first choice for the treatment of mild and moderate migraine attacks. NSAIDs prevent inflammation of the trigeminovascular system through inhibition of prostaglandin synthesis.

Ergotamine tartrate

Ergotamine tartrate and dihydroergotamine are useful in the treatment of moderate and severe migraine attacks. Ergotamine is an ergot amino acid alkaloid, whereas dihydroergotamine is an amino acid ergot alkaloid. It is a 5-HT1 receptor non-selective agonist that causes vasoconstriction of intracranial vessels and prevents neurogenic inflammation of the trigeminovascular system. The acute stage of therapy is ergotamine tatrat, subcutaneously or IM given as much as 0.25 - 0.5 mg. Dosage should not exceed 1mg / 24 hours. Orally or sublingually 2 mg can be given as soon as pain occurs. The dose should not exceed 10 mg / week. The dose for nasal administration is 0.5 mg (one spray). The dose should not exceed 2 mg (4 sprays). Contraindications are sepsis, vascular disease, thrombophebilitis, menstruating women, being pregnant or taking anti-pregnant pills. For women who are pregnant, menstruating or currently using anti-pregnant pills, give pethidine 50 mg IM. For patients with ischemic heart disease, use pizotifen 3 to 5 times 0.5 mg daily. Besides ergotamine can also be other drugs.

Antiemetic

Antiemetic therapy is useful for treating the nausea and vomiting that often accompanies migraine. A single dose of antiemetics such as metoclopramide, chlorpromazine, prochlorperazine is usually given 15-30 minutes before the administration of abortive migraine drugs. Suppository preparations can be given in case of severe nausea and vomiting.

Serotonin receptor agonists

The main class of this group is sumatripan and the second generation zolmitripan, naratripan, rizatripan, almotripan, flovatripan, and eletriptan are selective agonists of the 5-HT1B and 5-HT1D receptors.

Headache Prevention and Supporting Therapy

Prevention of headaches is by changing lifestyle patterns, namely regulating the same sleep pattern every day, exercising regularly, eating healthy and regular food, reducing stress, avoiding known headache triggers. One of the main clues to the examiner regarding the origin of the headache should come from the profile that constructs the headache symptoms, as well as the pre-history of the head injury. Just because a person has pre-injury headaches does not mean that he or she cannot develop various types of headaches or worsening pre-injury conditions after trauma. The main questions relating to the headache profile that need to be asked are expressed in "COLDER" mneumonia: Character, Onset, Location, Duration, Exacerbation, and Relief. Other descriptions include frequency, severity, associated symptoms, and presence / absence of aura, degree of functional disability associated with headache episodes, and time at which headache occurred, all important parameters for inquiring about this. Adequate physical examination is essential for proper diagnosis and should include inspection, palpation, auscultation and appropriate percussion. Neurological examination should be at the heart of this assessment, however, an adequate examination begins with examination of the skull and neck structures. Neuroimaging is generally not required for patients with primary headaches (eg migraine or chronic), but is usually indicated for secondary headaches (eg associated with an underlying pathology).

In general, MRI is considered superior to CT scan evaluating the brain parenchyma, and CT-scan is considered superior to MRI for evaluating subarachnoid hemorrhage. However, because CT-scanning is faster and more readily available, it should be performed in an emergency evaluation of patients with sudden onset, "thunderclap" headaches or the worst headache of their lives.

In patients with minor head injuries accompanied by persistent headaches, or vomiting is an indication for a head CT scan. Based on NICE, 2007, the criteria for a patient with a head injury to get an immediate head CT scan are GCS <13 at the initial examination in the ER, GCS <15 in the first 2 hours after the incident at the ER examination, suspicion of an open fracture or stress fracture. , there are signs of fracture of the base crania (haemotympanum, raccoon eyes, battle's sign, rhinorrhea or otorrhea), posttraumatic seizures, focal neurological deficits, more than 1 episode of vomiting and dementia> 30 minutes.

Conclusion

Headache, which in common people is often referred to as a headache, dizziness and others, is pain or discomfort in the entire area of the head with the lower border from the chin to the back of the head (occipital area and parts of the head). The classification of headaches is divided into primary and secondary headaches. The anatomy of headache is influenced by the trigeminocervical nucleus which is an important nociceptive for the head, throat and upper neck. Physiology of pain (pain) is a protective mechanism that can occur at any time if any tissue is damaged, and through this pain, an individual will react by moving away from the pain stimulus. Pain begins with the stimulation of the pain receptors by a pain stimulus. Prevention of headaches is by changing lifestyle patterns, namely regulating the same sleep pattern every day, exercising regularly, eating healthy and regular food, reducing stress, avoiding known headache triggers. And supporting therapy with MRI or CT-Scan. The management of headache can be treated with acetylsalicylic preparations and if the headache is very severe, ergot preparations (ergotamine or dihydroergotamine) can be given. If necessary it can be given intravenously at a dose of 1 mg dihydroergotaminmethan sulfate or ergotamine 0.5 mg. Preparations (containing 100 mg caffeine and 1 mg ergotamine) are given 2 tablets at the time of the attack and repeated in the next ½ hour.

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