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Review Article

A Review Article On Migraine And Food Supplements In The Management Of Migraine Headaches

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ABSTRACT

In two large longitudinal cohort studies, according to the Chronic Migraine Epidemiology and Outcomes Study and the American Migraine Prevalence and Prevention Study, CM patients completed both the Migraine Disability Rating Scale and the Headache Impact, suggesting that CM causes more disability than Episodic migraine. Chronic migraine is a disabling neurological disorder that affects 2% of the general population. Chronic migraine patients experience headaches at least 15 days per month, and headaches and related symptoms meet diagnostic criteria for migraine at least 8 days per month. Chronic migraine causes a great burden on patients due to frequent headaches, Hypersensitivity to visual, auditory, and olfactory stimuli, Nausea; and vomiting. It also impacts society through direct and indirect health costs. Chronic migraines usually develop after a slow increase in headache frequency over months or years. Migraine is a neurological disorder characterized by headache attacks. Hypersensitivity to visual, auditory, olfactory, and skin irritation.

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INTRODUCTION

On December 15, 2012, the special edition of The Lancet published the main findings of GBD2010. Few reports are likely to have greater significance for headache sufferers or promise a better future than these seven papers. GBD2000, conducted by the World Health Organization 12 years ago, listed migraine as the 19th leading cause of disability worldwide, accounting for 1.4 % of all years lived with disability. In GBD2000, average cases of migraines and three health states of each were agreed upon (i.e. ictal [during attacks], interictal [between attacks], and health state associated with medication-overuse headache [MOH], which was seen as a possible complication of migraine. [1] Treatment-resistant thrombocytopenia (TTH) is estimated to be 20.1% worldwide and migraine is 14.7% worldwide (second only to dental caries) among both men and women. Migraine was by far the most common cause of disability among neurological disorders (accounting for more than half of all disease-related deaths attributed to these conditions). Because migraine is so common and disabling, it is no surprise that it ranks 7th highest among specific causes of disease globally (accounting for 2.9%) and among the 10 leading causes of disability in 14 out of 21 world regions. There is little evidence of a gradient from west to east or that it is a disorder preferred by rich countries. Headache disorders are one of the commonest medical complaints: at least 10% of all adults worldwide are sometimes disabled by it, and 3% of adults live with it. [2] Male or female, any race, 18 to 65 years of age, female, child-bearing potential, negative urine pregnancy test, reliable contraception during the study period, Chronic migraine criteria as defined by Second Edition, International Classification for Headache Disorders, and subjects understood all study requirements. Exclusion Criteria: Pregnant, breast feeding, or planning to become pregnant female, headache disorder other than CM, medical

disorders that increased the risk of exposure with onabotulinumtoxin A, significant liver or kidney impairment, including kidney stones, ketogenic diet, previous use of botulinum toxin, or topiramate, regardless of indication, alcohol/drug abuse, or excessive use of acute medication. [3] Doctors consider modifying risk factors as part of migraine treatment, working to not only reduce current pain and disability but also to prevent migraine progression. Reducing attack frequency, avoiding drug overuse, using preventive medications and behavioural treatments appropriately, and promoting weight loss not only reduce current pain and disability, but also prevent chronicity. It should also be part of migraine treatment to reduce future pain and disability. Migraine is considered a potentially progressive disease. [4] Repeated episodes of pain can lead to central sensitization with free radical generation and anatomical changes in pain-controlling areas, resulting in a higher predisposition to further pain. Finally, as discussed in Section below, increased headache frequency may reflect an inherent predisposition to pain. CA refers to the perception of pain after receiving a non-painful stimulus. Trigeminal CA is a clinical marker of sensitization at the level of secondary sensory neurons, whose cell bodies are located in the caudal trigeminal nucleus. [5] Several comorbidities appear to tend to increase the risk of -CM. These include obesity, snoring, sleep apnea, psychiatric comorbidities, stressful life events, and temporomandibular disorder (TMD) AMPP Study shows that obesity is associated with headaches, It has been shown to be an aggravating factor in migraine rather than overall. Some of the inflammatory mediators that are elevated in obese individuals, such as interleukins and calcitonin gene-related peptide, are important in the pathophysiology of migraine. [6] Migraine and obesity are both prothrombotic diseases. Substances important in metabolic



regulation (such as adiponectin) exhibit nociceptive effects when levels are low. Hypothalamic dysfunction in the orexin pathway is thought to be a risk factor for both diseases. Finally, metabolic syndrome and autonomic dysfunction may also play a role in the relationship between obesity and migraine progression. It is important to emphasize that several treatable cardiovascular risk factors (such as metabolic syndrome, hyperlipidemia, and hypertension) may influence migraine progression by mediating the effects of obesity. [7] The mechanisms underlying the relationship between obstructive sleep apnea and migraine progression are not fully understood, but are likely to predispose to pain progression, hypoxia, hypercapnia, sleep fragmentation and disturbances. Fluctuations in intracranial and arterial pressure can occur during a person's snoring. [8] Anti-inflammatory drugs had a preventive effect in patients whose headaches were less than 10 days old at baseline, and triptans caused migraine progression in patients with frequent headache. The role of caffeine in the progression from episodic to chronic headaches is of great interest because dietary caffeine intake is high and prescription and non-prescription acute headache medications also contain caffeine. Shah et al, investigated the association between dietary and medical caffeine intake and frequent headaches. People with CM were more likely to consume large amounts of caffeine while experiencing 4,444 episodic headaches compared to people without CM. Abrupt withdrawal of caffeine in patients with CDH was associated with rebound headaches, further supporting the importance of this substance as a risk factor. Trauma can clearly cause recurrence of headache syndromes (post-traumatic headaches) and is considered a risk factor for migraine chronicity. Physicians should strive to prevent migraine progression in addition to reducing current pain and disability. [9 Improving the lives of today's

patients and advancing migraine prevention requires reducing attack frequency, avoiding overuse of medications, recognizing and treating temporomandibular joint disorders and comorbidities, and providing appropriate preventive medications and behavioral treatments. The use of to promote weight loss should be part of migraine treatment. [10] Migraine is a public health problem that has a major impact on both patients and society. The overall prevalence of migraine in Western countries is 6-8% in men and 15-25% in women Migraine has a strong (up to 50%) genetic component, which is more Also higher in MA, the risk may be multifactorial and polygenic. Genetic load can be considered to determine an inherent migraine threshold that is modulated by external and internal factors (migraine triggering). Multiple susceptibility loci exist on chromosomes 1q and 4q24, which are subtypes of MA, but within the skull, pain sensitivity is primarily restricted to meningeal vessels, with nociceptive seeking in the ocular part of the brain. It is densely innervated by cardiac fibers. [11]

Trigeminal nerve:

In various animal models, including non-human primates, activation of meningeal trigeminal neurovascular afferents induces activation of secondary dorsal horn neurons in the trigeminal caudal nucleus (TNC) and the two uppermost parts of the cervical cable, Brings activation. Impulses are then transmitted rostrally to brain structures involved in pain perception, such as several thalamic nuclei and the ventrolateral region of the caudal periaqueductal gray area (PAG). PAG is involved in cranial vascular pain not only through ascending projections to the thalamus but also through descending modulation (mainly inhibition) of nociceptive afferent. Activation of TGVS also causes the release of vasoactive neuropeptides contained in peripheral nerve terminals, particularly calcitonin gene-related



peptide (CGRP). Recent neurophysiological and imaging studies have shown that CM may be associated with structural and functional changes in some brain regions, particularly cortical hyperexcitability and brainstem dysmodulation. Several molecular mechanisms are involved in the pathogenesis of CM, including calcitonin gene-related peptide (CGRP), serotonin (5-HT) system, and pituitary adenylate cyclase activating polypeptide (PACAP). CGRP is secreted peripherally from trigeminal nerve afferents and mediates vasodilation and inflammatory events in the dura and trigeminal ganglia, which are important in the initiation and intensification of migraine attacks. [12]

5-HT or serotonin:

5-HT is primarily released from the brainstem and has long been thought to be involved in the pathophysiology of migraine, particularly in descending pain modulation. In addition to pain regulation, 5-HT also plays an important role in sleep pathophysiology and the development of mood disorders. Elevated levels of tumour necrosis factor alpha have been observed in the CSF of patients with CM, suggesting a role for inflammation and endothelial dysfunction in migraine progression.

Chronic migraine:

Chronic Migraine is a "fluid" condition, with many patients moving in and out of chronic migraine patterns over time. Factors that may be associated with reversal of chronic migraine include lower baseline frequency of headaches (15-19 days vs. 25-31 days/month), lack of allodynia, adherence to preventive medications, Withdrawal from used abortion pills, and gymnastics, Transition to chronic migraine, Obesity Snoring Sleep disorders, Excessive caffeine intake, Psychiatric illness, High baseline frequency of headaches, Excessive use of medications to abort migraine, Major lifestyle changes Head or neck injury, Lower socio-economic status, Paroxysmal

migraine recurrence, Adherence to migraine preventive medication Lower baseline, Absence of Pain Symptoms, Physical Activity Cessation of Migraine Abortion Pill Abuse. The depression waves were associated with dilation and narrowing of small arteries due to excessive reduction in electrical activity in cortical areas. [13]

Global Assessment of Migraine Severity (GAMS):

Migraine is a common chronic neurological disease that affects approximately 14% of adults in Europe, most commonly in women and in the 18-50-year age group. According to the World Health Organization's (WHO), Global Burden of Diseases Project, migraine ranks second among a total of 328 diseases assessed to affect people living with disabilities around the world in 2016. Global Assessment of Migraine Severity (GAMS) is a patient-reported outcome measure to document patients' perceptions of migraine severity. MIDAS is a measure of the impact of migraine on reduced productivity over the past 3 months. MIDAS captures disability information based on the number of days of absent or reduced activity due to headaches. [14]

Ophthalmology Migraine:

Ophthalmology Migraine allows you to distinguish between vision loss in the homonymous area of both eyes and vision loss in one eye. It also includes areas other than the retina, such as: Choroid or optic nerve that may be affected. Some people believe that the visual impairment associated with migraine is the result of vasospasm in the retinal or ciliary circulation. Diagnostic criteria for retinal migraine are presented in the first edition of the International Classification of Headache Disorders. This criterion required at least two attacks of fully reversible monocular visual impairment lasting less than 60 minutes and occurring before, during, or after a headache (type not specified). Nonpharmacological strategies include avoiding



potential migraine triggers. Patients should stop smoking and discontinuation of oral contraceptives may be recommended. It is obvious that maintaining healthy habits and managing stress is recommended [15].

Vestibular migraine:

VM appears to occur more frequently in migraineurs without aura than in migraineurs with aura. Benign paroxysmal vertigo in childhood is an early symptom of VM recognized by the IHS headache classification. The disease is characterized by brief bouts of dizziness, loss of balance, anxiety, and often nystagmus and vomiting that reoccur over months to years in otherwise healthy infants, 4,444 VM patients commonly report spontaneous dizziness or positional vertigo. Some experience a series of spontaneous dizziness that turns into positional vertigo after a few hours or days. Several neurotransmitters involved in the pathogenesis of migraine (such as calcitonin gene-related peptide, serotonin, norepinephrine, and dopamine) are also known to modulate the activity of central and peripheral vestibular neurons and may be implicated in the pathogenesis of migraine VM. Over the past decade, genetic defects in ion channels have been identified as the cause of various paroxysmal neurological disorders [16].

Migraine and epilepsy:

Migraine and epilepsy are common paroxysmal chronic conditions. In many ways these are clearly diseases, but there is pathophysiological overlap and overlap in clinical symptoms, particularly when it comes to visual impairments and other sensory deficits, pain, and altered consciousness, and theories about their causes have been around for centuries. Epilepsy was a brain disorder, and seizures were thought to be explosive electrical discharges in the brain that were transmitted to other parts of the nervous system. Patient with common or classic migraine has mostly normal MRI findings. Compared to controls, there is a

slight increase in small, bright lesions on T2-weighted images and fluid-attenuated inversion recovery images of, which may reflect an increased risk of stroke in migraine patients. [17]

Menstrual migraine:

The term "menstrual migraine" refers to headache disorders associated with fluctuations in estrogen levels during the menstrual cycle. Thus, attacks occur regularly in at least 2 out of 3 consecutive menstrual cycles, from 2 days before the onset of menstruation to 3 days after the onset of menstruation. There are two subtypes of menstrual migraine. In the first subtype, migraine sufferers suffer during the menstrual period, but also have attacks outside this period. [18]

Familial hemiplegic migraine :

Familial hemiplegic migraine is a rare form of migraine, but recent research has provided insight into its mechanisms, and the most common type of migraine, may lead to a better understanding of the index patient and other family members may suffer from typical migraine with or without aura. The condition is inherited in an autosomal dominant manner, with variable penetrance. Usually at least one first-degree relative of hers has an identical attack. Because of incomplete penetration, affected individuals may have no affected first-degree relatives, but asymptomatic families may have 4,444 affected children. [19]

Food and food supplements in the management:

The importance of regular meals cannot be overstated, as skipping meals can lead to headaches. Skipping meals and fasting have been reported to cause migraines in over 56% of population-based studies, 16 and between 40% and 57% in specialty clinic-based studies. Fasting and Diet The mechanism by which pulling out causes headaches may be related to changes in serotonin and noradrenaline in brainstem pathways 20 or the release of stress hormones such as cortisol. Low blood sugar can cause headaches.



In one study, three-quarters, or 21, of 4,444 participants with migraine performed a 5-hour glucose tolerance test consistent with reactive hypoglycemia. Recognizing dietary migraine triggers is important because it not only reduces migraine frequency but also gives migraine sufferers a sense of control over conditions that can leave them helpless and debilitated.

Phenylethylamine:

Phenylethylamine is a substance found in cocoa. In migraine patients, especially those with decreased monoamine oxidase B activity, phenylethylamine triggers the release of vasoactive amines such as serotonin and catecholamines. Migraine patients cite chocolate as a migraine trigger. However, the role of phenylethylamine remains unclear.

Aspartame:

Aspartame is an artificial sweetener (NutraSweet) that is 180 to 200 times sweeter than sugar. Since its introduction in 1981, many neurological or behavioral symptoms, particularly headaches, have been reported that can be attributed to aspartame use. Studies found that aspartame caused no more headaches than a placebo, but other evidence suggests that aspartame is effective at moderate to high doses (900– 3000 mg/day) over long periods of time. It has been suggested that it may cause headaches in people who consume it.

Sodium nitrite:

Sodium nitrite is a preservative used to colour foods, prevent botulism, and add salted or smoked flavour. Some people develop a headache within minutes to hours after consuming foods containing nitrites, such as sausages or other deli meats or fish. This effect is likely due to the release of nitric oxide and subsequent vasodilation. The interaction between nitrite and blood pigment may also be involved in the development of methemoglobinemia.

Alcohol:

Alcohol, especially red wine, is a common migraine trigger. It has an immediate (within 3 hours) or delayed (hangover) effect. Some patients even report that alcohol can cause a headache within minutes.

Caffeine:

Caffeine is a common dietary substance found in coffee, tea, soda, and chocolate. It is also found in 4,444 different prescription (Fioricet, Fiorinal, Esgic) and over-the-counter headache medications (Excedrin, Anacin). Caffeine acts by blocking inhibitory and excitatory adenosine receptors in the brain and vasculature, causing vasoconstriction and release of excitatory neurotransmitters. Some of the signaling pathways involved are important in modulating pain perception. Caffeine content in a cup of coffee prepared with is typically 115 mg, while a cup of Pepsi has 38 mg. Excedrin contains 65 mg of caffeine per tablets. Caffeine provides increased alertness, concentration, and energy in low to moderate doses (50-300 mg).

Magnesium:

Vitamins and other nutritional supplements to prevent migraines are Magnesium, an essential cation that plays an important role in several physiological processes. Magnesium may be involved in the development of migraine by counteracting vasospasm, inhibiting platelet aggregation, and stabilizing cell membranes. Its concentration affects serotonin receptors, nitric oxide synthesis and release, inflammatory mediators, and a variety of other migraine-related receptor neurotransmitters.

Feverfew:

Feverfew is an herbal preparation available as the dried leaves of the weedy plant *Tanacetum parthenium*. It was used centuries ago to treat headaches, inflammation, and fever, and was rediscovered in the late 20th century. The mechanism of action in migraine prevention may be related to parthenolide present in the leaves.



These inhibit serotonin release from platelets and leukocytes and inhibit platelet aggregation.

Petasites hybridus:

In recent years, the root extract of *Petasites hybridus*, also known as butterbur, has emerged as a potential new treatment for migraine prevention. Butterbur is a perennial shrub used in ancient times for its medicinal properties. *Petasites* is thought to function by modulating calcium channels and inhibiting peptide leukotriene biosynthesis.

Ginger:

Ginger has been used in China for its medicinal properties to treat pain, inflammation, and musculoskeletal conditions. It has anti-inflammatory properties that may be associated with thrombocytopenia. There are anecdotal and folklore accounts of its effectiveness in relieving headaches and nausea. [20]

CONCLUSION:

Chronic migraine is associated with a greater disease burden compared to episodic migraine, as evidenced by higher disability rates, higher healthcare costs, and increased rates of psychiatric comorbidity. The several genes or pathways have been linked to migraine pathophysiology. Several molecular mechanisms are involved in migraine like calcitonin gene related peptide [cgrp], serotonin[5-ht] and other environmental factors that stimulate the migraine. Different types of migraines are occurring and are different from each other, they include retinal migraine, ophthalmic migraine, vestibular migraine, episodic migraine, chronic migraine, menstrual migraine. Different food supplements, we consume in our food diet, having many vitamins minerals proteins. Maintaining the balanced diet and avoiding the contents in the food that stimulate the migraine reduces the number of headaches in a month like more caffeine intake, sodium nitrate foods.

REFERENCES:

1. T J Steiner, L J Stovner, G L Birbeck
Migraine: the seventh disabler DOI:
10.1177/0333102412473843
2. Roger K Cady 1, Curtis P Schreiber, John A
H Porter, Andrew M Blumenfeld, Kathleen U
Farmer- A Multi-Center Double-Blind Pilot
Comparison of OnabotulinumtoxinA and
Topiramate for the Prophylactic Treatment of
Chronic Migraineheadache doi:
10.1111/j.1526-4610.2010.01796.x
3. Marcelo E Bigal 1, Richard B Lipton-
Migraine Chronification DOI
10.1007/s11910-010-0175-6
4. Daniela Pietrobon 1, Jörg Striessnig-
NEUROBIOLOGY OF MIGRAINE
doi:10.1038/nrn1102
5. Min Su , Shengyuan Yu - Chronic migraine:
A process of dysmodulation and sensitization
DOI:10.1177/1744806918767697
6. Todd J Schwedt - Chronic migraine doi:
10.1136/bmj.g1416
7. Robert W Baloh – Vestibular Migraine I:
Mechanisms, Diagnosis, and Clinical
Features DOI; 10.1055/s-0039-3402735.
8. David Curfman 1, Michael Chilungu, Robert
B Daroff, Amer Alsheklee, Gisela
Chelimsky, Thomas C Chelimsky –Syncope
migrain DOI 10.1007/s10286-011-0141-7
9. Anne Donnet , Anne Ducros , Françoise Radat
, Bashar Allaf , Isabelle Chouette , Michel
Lanteri-Minet – Severe migraine and its
control: A proposal for definitions and
consequences for care
doi:10.1016/j.neurol.2020.11.012
10. Joseph M Furman 1, Dawn A Marcus –
Migraine and Motion Sensitivity
Doi:10.1212/01.CON.0000421621.18407.96.
11. Christoph J Schankin , Michele Viana , Peter
J Goadsby – Persistent and Repetitive Visual
Disturbances in Migraine : A Review doi:
10.1111/head.12946



12. Antonio Russo 1, Alessandro Tessitore, Gioacchino Tedeschi – Migraine and Trigeminal System—I can Feel it Coming... DOI :10.1007/s11916-013-0367-2
13. S Solomon - Migraine Variants doi: 10.1007/s11916-001-0084-0
14. M von Brevern , T Lempert - Vestibular migraine doi:10.1016/B978-0-444-63437-5.00022-4
15. Brian M Grosberg 1, Seymour Solomon, Richard B Lipton -Retinal Migraine DOI:10.1007/s11916-005-0035-2.
16. Hannelore Neuhauser 1, Thomas Lempert – Vestibular Migraine doi:10.1016/j.ncl.2008.11.004
17. Barbara L Nye 1, Vijay M Thadani – Migraine and Epilepsy: Review of the Literature doi: 10.1111/head.12536
18. Vasilios Tanos , Elissa Abi Raad , Kelsey Elizabeth Berry , Zara Abigail Toney - Review of migraine incidence and management in obstetrics and gynaecology doi:10.1016/j.ejogrb.2019.07.021
19. Pasquale Montagna 1 – Migraine: a genetic disease? DOI :10.1007/s10072-008-0886-5
20. Christina Sun-Edelstein 1, Alexander Mauskop- Foods and Supplements in the Management of Migraine Headaches doi:10.1097/AJP.0b013e31819a6f65.

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