

Research Articles on Anti-Migraine

Mr. Satish Rajendra Borse, Dr. Manoj Dilip Patil

(M. Pharm), Sumantai Institute of Pharmacy, Bambrud

(M. Pharm PhD), Sumantai Institute of Pharmacy, Bambrud

Submitted: 20-09-2022

Accepted: 30-09-2022

ABSTRACT

Migraine is a common disabling condition mostly in adult population and shows female predominance. Unilateral throbbing type moderate to severe intensity headache is a common manifestation of the migraine though it may present with varied presentation. Even though there is rapid advancement in the knowledge of pathophysiology leading to development of novel treatment, evidence-based treatment for migraine specially in developing nations is still unmet needs. This article reviews the pathophysiology, diagnosis and evidence-based approach for management of migraine.

Keywords: Anti-Migraine, Management of Migraine

I. INTRODUCTION

Headache disorders, these are characterized by the recurrent episodes of headache and are the most common nervous system disorders. Headache itself is the painful and also disabling feature of few numbers of primary headaches, like migraine, cluster head-ache, tension type headache. Among these, the migraine headache is ubiquitous, prevailing, disabling and essentially treatable, But still under-estimated and under-treated.

Migraine is the second most cause of headache and the most common headache related and neurologic cause of disability in the world. The name 'migraine' comes originally from the Greek word 'hemicrania', it means 'half of the head', it represents one of the most important features of the condition, that in many of the cases, the pain will affect half of the head only. However, some times the pain is felt bilaterally, either at back or front of the head and sometimes rarely all over the body and face ('migrainous corpalgia'). The pain is generally throbbing, and sometimes pulsatile in nature and it typically increases by any form of movements made by the body or head. Migraine is a common chronic headache disorder which is characterized by the recurrent attacks which lasts from 4-72 hours, with a pulsating quality. Migraine

is the commonest cause of head ache and its intensity includes as mild, moderate and severe, it is aggravated by any of the routine physical activity. Migraine has some associated features like, vomiting, nausea, photophobia and/or phonophobia and migraine is attributed to the activation of meningeal perivascular pain fibers and also increased sensitization of central pain neurons that process information from intra cranial structures and extra cranial skin and muscles.⁽¹⁾

II. MATERIALS & METHODS: PATHOPHYSIOLOGY

1. Vascular and Neurogenic theories

The cause of migraine headache is still not completely understood. Historically, two independent theories, the vascular theory and the neuronal theory, explaining the etiology of migraine headache were proposed. The vascular theory was introduced by Thomas Willis where he argued the pain from headache is caused by vasodilatation of the cerebral and meningeal arteries. The alternative neurogenic theory focuses on the cause of migraine pain and is currently linked to activation of the trigeminovascular system.⁽²⁾

2. Cortical Spreading Depression

The alternative and widely accepted theory suggests that cortical spreading depression (CSD), a wave of neuronal hyperactivity followed by an area of cortical depression, accounts for the aura and that the headache depends on activation of the trigeminovascular pain pathway.

In Chronic Migraine (CM), atypical pain processing, central and peripheral sensitization, cortical hyper excitability, and neurogenic inflammation all have a role to play. Cortical hyper excitability is thought to be another major factor participating in transformation of EM to CM.

3. Cortical hyperexcitability in migraine

As is the case for many episodic disorders, the trigger for migraine attacks has not been precisely identified. Many clinical factors such as

diet, alterations in sleep and stress are known to predispose individuals to attacks. It is particularly intriguing that photic stimulation can trigger both migraine attacks and epileptic seizures. How these factors bring on a migraine attack is not known. However, there is evidence for enhanced cortical responsiveness to diverse stimuli in migraineurs. The techniques that have been used to generate this evidence include psychophysical studies, visual, auditory, and somato sensory evoked potentials; magneto encephalography; and transcranial magnetic stimulation of the motor cortex. In all cases, there is evidence of heightened reactivity

between migraine attacks. Results from transcranial magnetic stimulation of the occipital (visual) cortex have been particularly compelling. Most but not all studies have observed that migraineurs have a reduced threshold for induction of phosphenes (the experience of light with non-luminous stimulation) compared with controls. This phenomenon appears to be equally present in individuals who experience migraines with and without aura. Thus, a pathologically low threshold for activation of cortical hyper excitability may characterize migraine."⁽³⁾

Figure 1

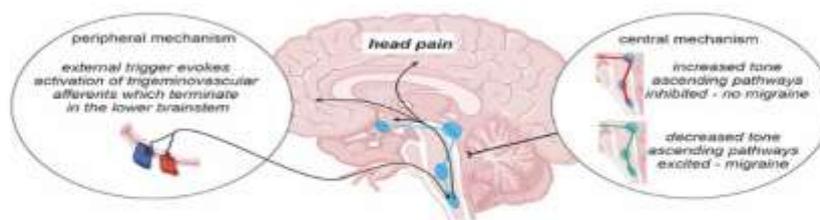


FIGURE 1. Central vs. peripheral mechanism of migraine. The prevailing theories of migraine initiation include the existence of a peripheral trigger and/or central nervous system changes including oscillations in the sensitivity of descending pain modulatory pathways across the migraine cycle. Such changes in brainstem "tone" are proposed to either prevent or allow an external trigger or basal brainstem activity from evoking activity changes in ascending pathways which are ultimately responsible for the presence of a migraine event.

Classification of migraine

The International Classification of Headache Disorders, 3rd Edition (Beta Version)

Part 1: The Primary Headaches

1. Migraine
2. Tension-type headache
3. Trigeminal autonomic cephalgia
4. Other primary headache disorders

Part 2: The Secondary Headaches-Headache (or Facial Pain) Attributed to:

5. Trauma or injury to the head and/or neck
6. Cranial or cervical vascular disease
7. Nonvascular intracranial disorder
8. A substance or its withdrawal
9. Infection
10. Disorder of homeostasis
11. Disorder of the cranium, neck eyes, ears, nose, sinuses, teeth, mouth, or other facial or cervical structure.
12. Psychiatric disorder

Part 3: Painful Cranial Neuropathies, Other Facial Pains, and Other Headaches

13. Painful cranial neuropathies and other facial pain
14. Other headache disorders⁽⁵⁾

Causes

Experts think that migraine episodes may stem from changes in the brain that affect the

- way nerves communicate
- balance of chemicals
- blood vessels

Genetic features may also play a role-having a family history Trusted Source of migraine is a common risk factor Migraine triggers vary from person to person. They commonly include:hormonal changes, such as those related to menstruation emotional triggers, such as stress, depression, anxiety, and excitement dietary factors, including alcohol, caffeine, chocolate, nuts, cheese, citrus fruits, and foods containing the additives tyramine and monosodium glutamate (MSG) medications such as sleeping pills, hormone replacement therapy (HRT), and some birth control pills environmental factors, including flickering screens, strong smells, second-hand smoke, loud noises, humidity, stuffy rooms, temperature changes, and bright lights

Some other possible triggers include.

- tiredness
- a lack of sleep

- shoulder and neck tension
- poor posture
- physical overexertion
- low blood sugar
- jet lag
- irregular mealtimes
- dehydration

Avoiding triggers, when possible, may help reduce the frequency of migraine episodes.⁽⁶⁾

Research data on race and ethnicity

Migraine can be a debilitating condition that is underdiagnosed and challenging to treat. This can be seen especially in BIPOC (Black, Indigenous, and People of Colour) populations. People of Colour are less likely to receive the diagnosis of migraine and the treatment than white people in fact, only 47% of African Americans Trusted Source have an official migraine diagnosis, compared with 70% of white people in the country. And other research found that Latino people are 50% less likely to receive a formal migraine diagnosis than white people. These disparities can impact treatment and therapies.

While these figures could lead to the conclusion that white people experience more migraine episodes than other groups, an analysis of nine studies Trusted Source looking at the average prevalence of severe headache or migraine from 2005 to 2012 in the U.S. found that the prevalence rates of episodes across all groups were similar

- 17.7% of Native American people
- 15.5% of white people
- 14.5% of Hispanic people
- 14.45% of Black people.
- 9.2% of Asian people⁽⁷⁾

Migraine treatment

Migraine can't be cured, but your doctor can help you manage migraine attacks by giving you the tools to treat symptoms when they occur, which may lead to fewer attacks in general. Treatment can also help make migraine less severe

Your treatment plan depends on

- your age.
- how often you have migraines attacks
- the type of migraine you have
- how severe they are based on how long they last. how much pain you have, and how often they

- keep you from going to school or work
- whether they include nausea or vomiting, as well as other symptoms
- other health conditions you may have
- and other medications you may take

Your treatment plan may include a combination of

- lifestyle adjustments, including stress management and avoiding migraine triggers
- OTC pain or migraine medications, like Nonsteroidal anti-inflammatory drugs (NSAIDs) or acetaminophen (Tylenol)
- prescription migraine medications that you take every day to help prevent migraine headaches and reduce how often you have headaches
- prescription migraine medications that you take as soon as an attack starts to keep it from becoming severe and to ease symptoms
- prescription medications to help with nausea or vomiting
- hormone therapy if migraines seem to occur in relation to your menstrual cycle
- counselling
- alternative care, which may include meditation, acupressure or acupuncture⁽⁸⁾

Medication

Medications can be used to either prevent a migraine attack from happening or treat it once it occurs. You may be able to get relief with OTC medication but if OTC medications aren't effective, your doctor may decide to prescribe other medications.⁽⁹⁾

The severity of your migraine and any other health conditions you have will determine which treatment is right for you.

Acute medications - taken as soon as you suspect a migraine attack is coming — include

- **NSAIDs:** These medications, like ibuprofen or aspirin, are typically used in mild-to-moderate attacks that don't include nausea or vomiting
- **Triptans.** These medications, like sumatriptan, eletriptan, and rizatriptan, are typically the first line of defense for individuals who have nerve pain as a symptom of their migraine attacks.
- **Antiemetics:** These medications, like metoclopramide, chlorpromazine, and prochlorperazine, are typically used with NSAIDs to help decrease nausea

- **Ergot alkaloids:** These medications like Migranal and Ergomar aren't prescribed that often and are usually reserved for individuals who don't respond to triptans or analgesics

Preventative medications-prescribed to people whose migraine attacks can be debilitating or happen more than four times a month are taken once a day, or every 3 months via injection. These medications include

- **Antihypertensives:** These drugs are prescribed for high blood pressure and can also help with migraine attacks. Beta-blockers and angiotensin receptor blockers (candesartan) are some examples of antihypertensive drugs used for migraine prevention.
- **Anticonvulsants:** Certain anti-seizure medications may also be able to prevent migraine attacks.
- **Antidepressants:** Some antidepressants, like amitriptyline and venlafaxine, may also be able to prevent migraine attacks.
- **Botox:** Botox injections are administered to the head and neck muscles every 3 months.
- **Calcitonin gene-related peptide treatments:** These treatments are administered either via injection or through an IV and work to prevent a migraine attack from developing.⁽¹⁰⁾

III. CONCLUSION

Headache, a condition that has been described almost since the beginning of recorded history, is now an area of increasingly intense interest and focus. Fundamental improvements in our understanding of this common and, at times debilitating condition are emerging. A flexible system of categorization of the various headaches allows for proper management in the present and sets the stage for advancement of future discoveries.⁽¹¹⁾

REFERENCE:

- [1]. GBD 2015 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015.
- [2]. The Subcutaneous Sumatriptan International Study Group. Treatment of migraine attacks with sumatriptan.



- [3]. World Health Organization. Neurological disorders: a public health approach neural disorder public health challenges, 2006.
- [4]. Rogowski MA. Common Pathophysiologic Mechanisms in Migraine and Epilepsy. Arch Neural, 2008
- [5]. Headache Classification Committee of the International Headache Society. The international classification of headache disorders, Third edition Cephalalgia, 2013.
- [6]. Parsekian D. Medicine cabinet. West J Med. 2000.
- [7]. Andreus AP, Goadsby PJ. Therapeutic potential of novel glutamate receptor antagonists in migraine. Expert Opin Investing Drugs 2019.
- [8]. Marin JC, Goadsby PJ. Glutamatergic fine tuning with ADX-10059- a novel therapeutic approach for migraine? Expert Opin investing drugs 2010.
- [9]. Amrut Mahajan's Essentials of Anatomy Arihant Excel Medical Book House, Aurangabad.
- [10]. Ross & Wilson Anatomy & Physiology in Health and Illness Third Edition (International Edition)
- [11]. Diener H-C, Holle D, Sobach K, Gaul C Medication- Overuse headache risk factors, pathophysiology and management Nat Rev Neural, 2016