



Headache

BLUEPRINT NEUROLOGY 5E

BMJ BEST PRACTICE

Headache

- Is a pain or discomfort in the head, scalp, or neck region
- The most prevalent medical problems worldwide
- headache disorders are the third highest cause of years lost to disability worldwide.
- headaches are one of the most common reasons patients present to physicians in primary care settings, in the emergency department (ED), or in neurologists' clinics

Headache disorders fall into two categories, **primary headache disorders** (those caused by the headache disorder itself, not due to other causes) and **secondary headache disorders** (those caused by another underlying medical problem).

Primary headache

1-MIGRAINE: Unilateral headache lasts 4-72H. Describe as throbbing pain . It associated with nausea, vomiting and photophobia, phonophobia.

And may preceded by reversible neurological symptoms called **Aura**.

2- TENSION-TYPE HEADACHE: Bilateral (encircles the entire headache).lasts under an Hour
For several days. Describe as tightness or pressure around the head. And Is NOT associated with N/V and photophobia and phonophobia.

3- CLUSTER HEADACHE: Unilateral pain around and behind the orbit. Lasts 15-20m several times a day .describe as stabbing ,burning pain. **IT ASSOCIATED WITH AUTONOMIC FEATURES**

Secondary headache

1-VASCULAR CAUSES : subarachnoid hemorrhage (SAH), intraparenchymal hemorrhage, and subdural and epidural hematomas

2-INFECTIOUS OR INFLAMMATORY CAUSES : encephalitis and meningitis

3-NEOPLASTIC CAUSES

4-INTRACEREBRAL PRESSURE DISORDERS : Intracerebral hypertension. Intracerebral hypotension.

5-MEDICATION CAUSES.

Red flags of headache

Sudden onset within seconds to minutes.

Worst headache of the patient's life.

New-onset headache that the patient has never experienced before, especially over the age of 50 years.

Headache pattern: increase in severity and frequency over time, worse after lying down.

Mental status change or any focal neurologic symptoms/signs.

New-onset headache associated with heavy exertion or head trauma.

New-onset headache associated with fever, stiff neck, or rash.

New-onset headache in a patient with HIV infection or cancer

DIAGNOSIS(history taking)

.patient Profile

.Chief Complaints

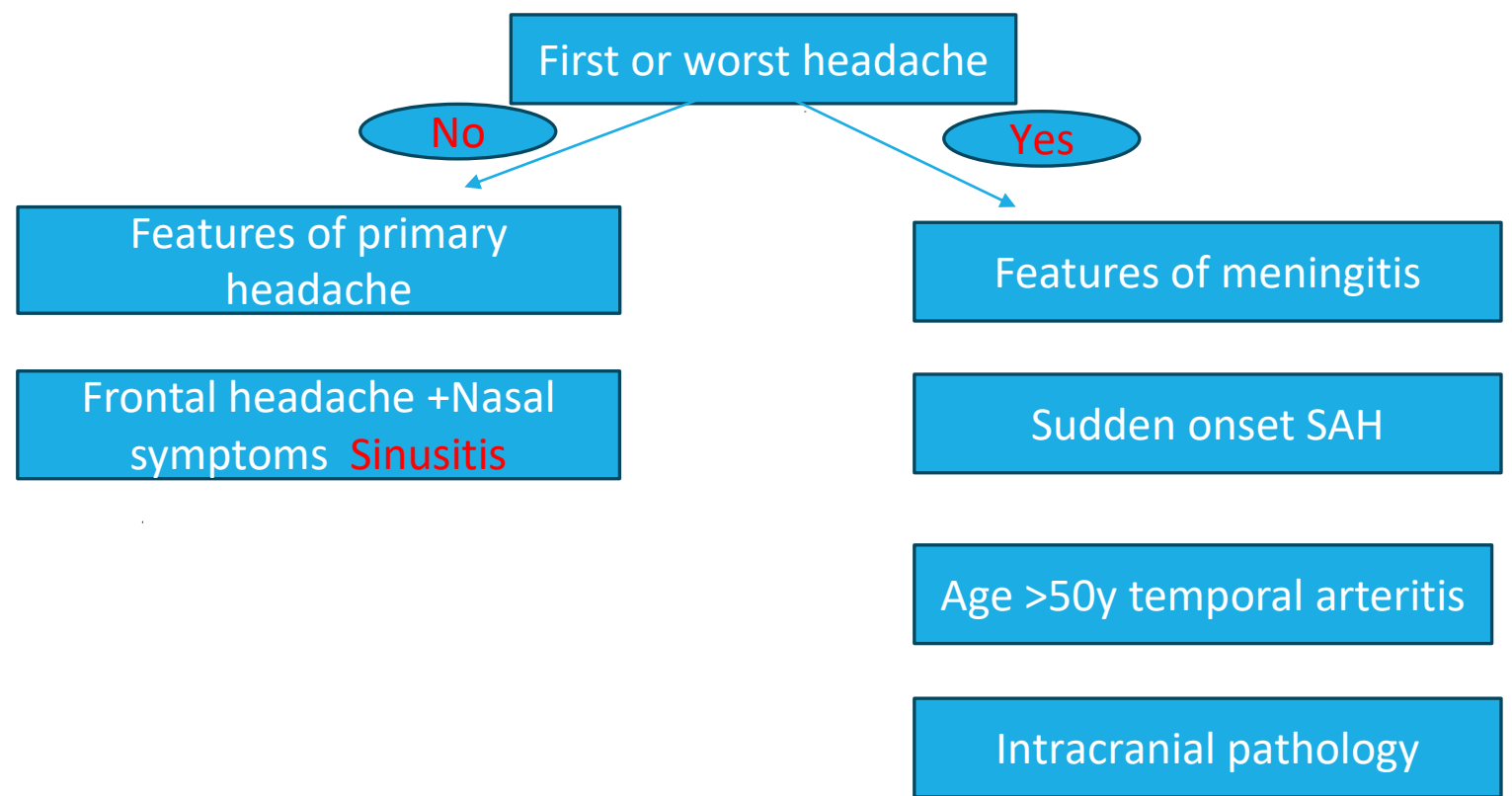
.History of Present illness (socrats)

.Past medical and surgical history

.drug history

.Family history

. Social history



DIAGNOSIS(physical examination)

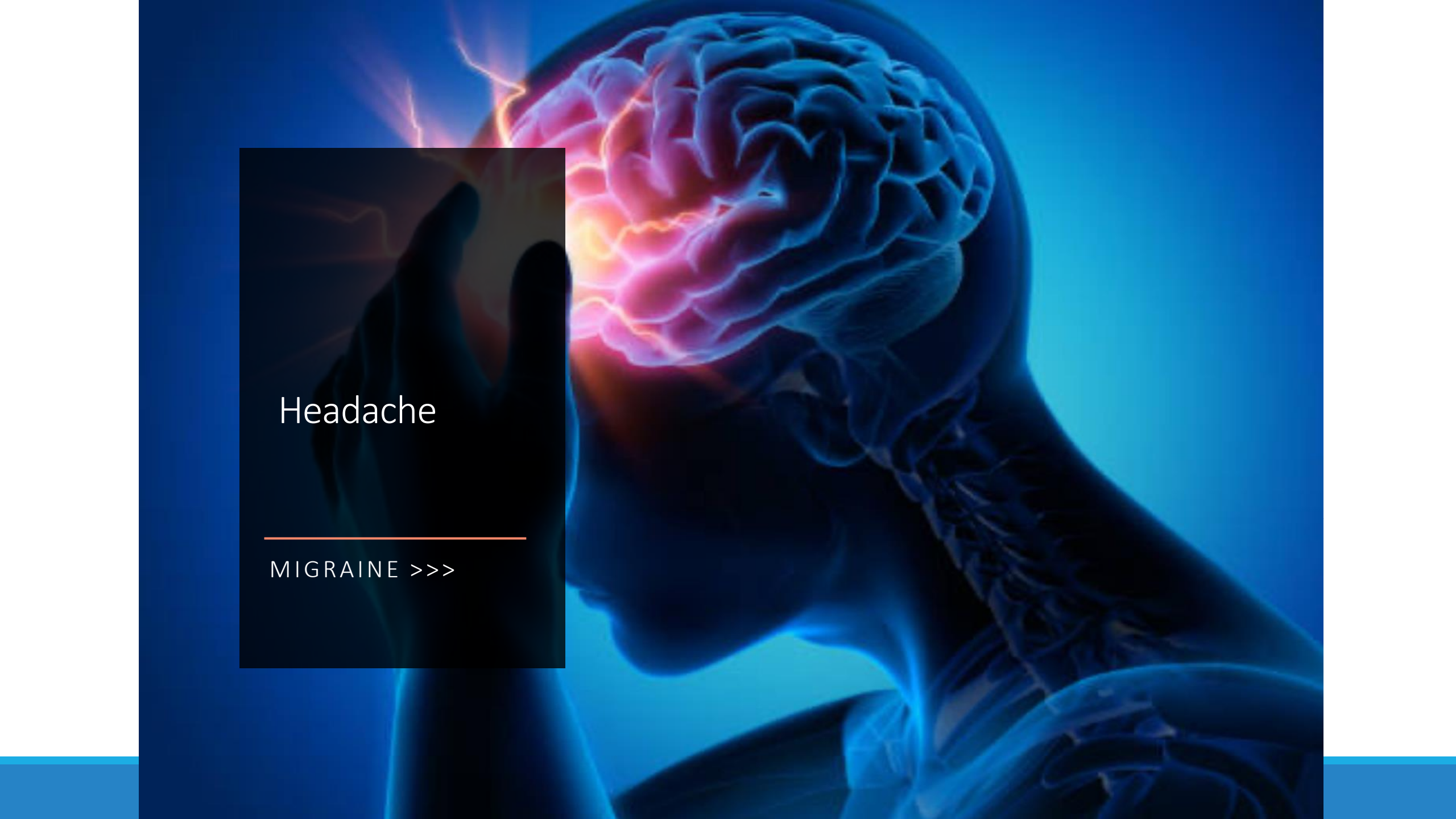
Patients with significant hypertension may be susceptible to developing certain secondary headaches

Fever may suggest an underlying infection (**CNS**) infection.

Palpation of the TMJ, assessment of dental wearing or chipping to suggest bruxism

Foldscope exam to assess for **papilledema**. Suggest **Increase ICP**

Patients with primary headache disorders usually have normal general medical and neurologic examinations



Headache

MIGRAINE >>>



Summary

Migraine is a chronic, episodic, neurological disorder that has a strong genetic component and usually presents in early-to-mid life. It can have a severe effect on quality of life, but it is often under-diagnosed and under-treated.

Patients complain of intermittent headache and associated symptoms, such as visual disturbance, nausea, vomiting, and sensitivity to light or noise (photophobia and phonophobia).

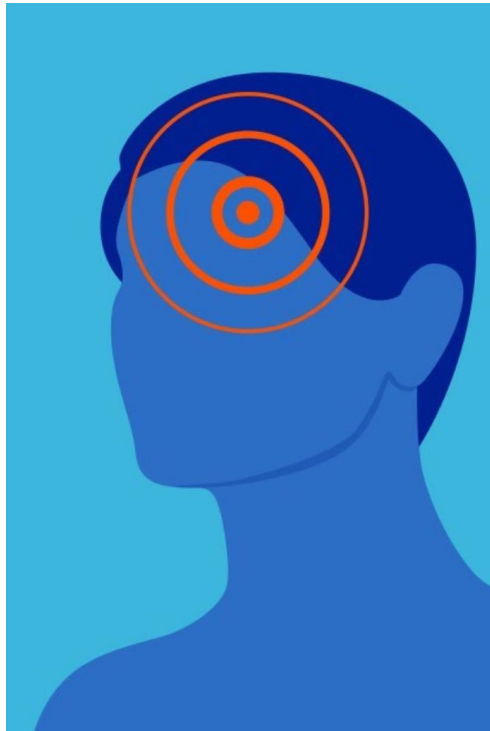
Some women experience menstrual migraine, which is most likely to occur in the 2 days leading up to a period and in the first 3 days of a period.

Diagnosis is based on history and physical examination. No laboratory or imaging tests are essential for diagnosis.

Treatment approaches involve identifying and avoiding trigger factors, and the use of medication to treat the acute attack and to prevent future attacks. Triptans are preferred over non-specific treatments.

Typical migraine aura (a complex of reversible visual, sensory, or speech symptoms), which precedes or occurs during headache, is pathognomonic of migraine but occurs only in 15% to 30% of patients.

Migraine.



Epidemiology :

Before puberty, migraine prevalence is higher in boys than in girls, but following adolescence it becomes three times more prevalent among women than men. Prevalence declines with age in both sexes.

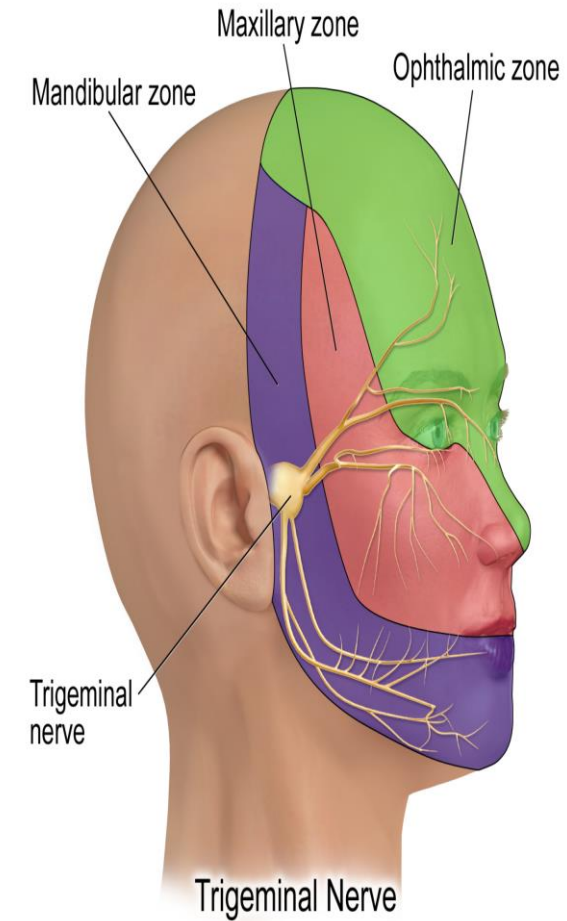
Aetiology:

Migraine has a strong genetic component. Genes associated with migraine increase or alter neuronal excitability through a variety of mechanisms so migraine is a disorder of excitability that is characterised by deficient regulation of the(cortical excitatory–inhibitory balance).

Migraine Pathophysiology

Migraine is a neurovascular disorder, in which neurological events precede and initiate headache. The headache of migraine results from neurogenic inflammation of first-division trigeminal sensory neurons that innervate the large vessels and meninges of the brain.

When activated, the trigeminal neurons release substances that cause dilation of meningeal blood vessels, leakage of plasma proteins into surrounding tissue, and platelet activation, substance P (has a role in the regulation of pain), and bradykinin are released, resulting in sensitisation of peripheral nociceptors. This peripheral sensitisation results in increased nociceptive inputs into the trigeminal sensory nucleus, and ultimately in central sensitisation, so that non-painful stimuli (for example, light touch) are interpreted as pain.





Classification of migraine

1) Migraine with aura: (classical) recurrent attacks, lasting minutes, of unilateral fully reversible visual, sensory, or other central nervous system symptoms that usually develop gradually and are usually followed by headache and associated migraine symptoms.....

2) Migraine without aura: (common) recurrent headache disorder manifesting in attacks lasting 4 to 72 hours. Typical characteristics of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity, and association with nausea and/or photophobia and phonophobia.

[aura]:

*Migraine with typical aura: migraine with aura, in which aura consists of visual and/or sensory and/or speech/language symptoms, # but no motor weakness#, and is characterised by gradual development, duration of each symptom no longer than 1 hour, a mix of positive and negative features, and complete reversibility. *there is typical aura with a headache and a typical aura without headache.

**Migraine with brainstem aura: migraine with aura symptoms clearly originating from the brain stem, but no motor weakness.

***Hemiplegic migraine: migraine with aura including motor weakness.



What does aura mean?

Typical migraine aura (a complex of reversible visual, sensory, or speech symptoms) is pathognomonic of migraine, but occurs in only 15% to 30% of patients. Aura typically occurs just before or at onset of headache, developing over 5 to 20 minutes and lasting less than 60 minutes, but it may not occur every time. Aura can also occur during the headache. Aura includes positive phenomena (visual sparkles, flashing lights, tingling) and negative phenomena (visual loss or scotomata, numbness, speech disturbance)

Migraine with aura

Pathophysiology

Aura is caused by neuronal dysfunction, A wave of neuronal excitation spreads anteriorly in the cortex, at a rate of 3-5 mm/minute (which correlates temporally with the reported rate of change in visual symptoms). This is followed by a prolonged period of decreased neuronal activity, and finally neuronal recovery. Cortical depression causes release of excitatory amino acids and other mediators of excitation, resulting in activation of nociceptors in adjacent dura and blood vessels, leading in turn to activation of the trigeminal sensory nucleus. How these neurons are triggered in migraine without aura is unknown, but one hypothesis is that cortical spreading depression in migraine without aura occurs in 'silent' areas of the brain that do not produce recognisable symptoms of aura.

International classification of headache disorders, 3rd edition(ICHD-3)

**Migraine with aura:

A. At least 2 attacks fulfilling criteria B and C

B. One or more of the following fully reversible aura symptoms:

(Visual• Sensory• Speech and/or language • Motor• Brainstem • Retinal)

C. At least 3 of the following 6 characteristics:

(At least one aura symptom spreads gradually over 5 minutes • Two or more aura symptoms occur in succession• Each individual aura symptom lasts 5 to 60 minutes• At least one aura symptom is unilateral• At least one aura symptom is positive• The aura is accompanied, or followed within 60 minutes, by headache).

D. Not better accounted for by another ICHD-3 diagnosis.



Diagnosis of migraine

The key to diagnosis of common forms of migraine **is history and physical examination**. Before starting treatment for migraine, it is important to ensure that the patient meets International Classification of Headache Disorders, third edition (ICHD-3) criteria for migraine or probable migraine. A single patient can have more than one headache diagnosis. The diagnosis of migraine does not preclude the diagnosis of another headache disorder if criteria are met.

* Diagnostic test:

Diagnostic testing for people with headache is primarily to exclude concerning or problematic causes for headache, as there is no specific test revealing the causes of pain in the head. In many cases where the history is compatible with a diagnosis of migraine and the neurological examination is normal, **no testing is necessary**.



Red Flags:

Features that should increase suspicion of a dangerous underlying headache or migraine mimic can be summarised using the mnemonic 'SNOOP4':

- Systemic symptoms: fever, weight loss
- Neurological symptoms or abnormal signs: confusion, impaired alertness or consciousness
- Onset: sudden, abrupt, or split-second
- Older: new-onset and progressive headache, especially in patients aged >50 years
- 4 'P's:
- Pattern change (increased frequency)
- Papilloedema
- Precipitating factors (Valsalva, etc)
- Positional aggravation.



History and exam

Key diagnostic factors, presence of risk factors (common):

- family history of migraine; female sex; menstruation; stressful life events; obesity; sleep disorders; medication overuse.
- prolonged headache (common) A headache that lasts 4 to 72 hours if untreated is suggestive of migraine.
- Nausea and vomiting are more common in younger patients and become less common as patients age. decreased ability to function (common)
- Migraine is commonly made worse with routine daily activities as well as exercise, A useful feature when trying to distinguish between migraine and tension-type headache, as the latter is not made worse by exertion.
- sensitivity to light (common).
- unilateral (uncommon), Migraine is often unilateral. However, it is bilateral in more than 40% of patients, so location does not confirm the diagnosis or rule it out.
- Headache may shift from side to side during or within attacks; some experts suggest that when this does not occur (side-locked headache), the likelihood of a fixed structural lesion is increased.



MIGRAINE WITHOUT AURA

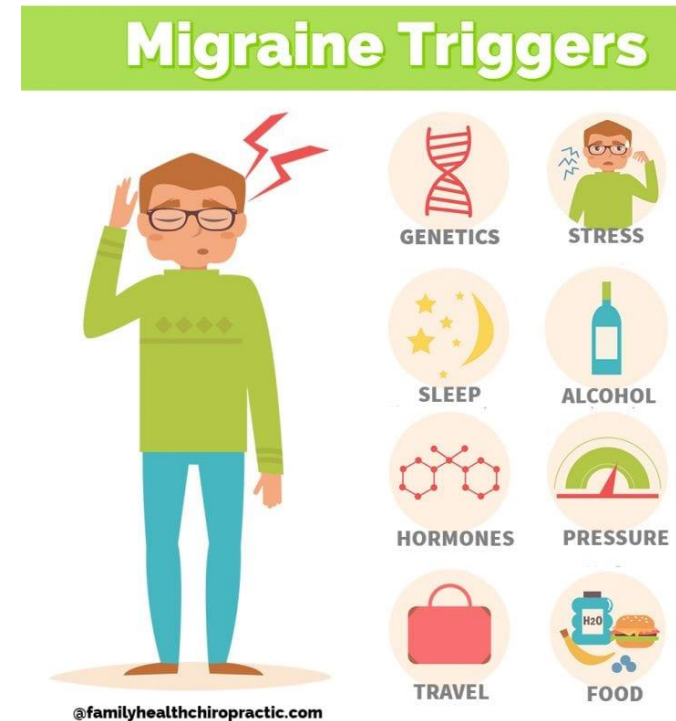


MIGRAINE WITHOUT AURA

- **Common** migraine 80% it has no Aura .
- Migraine Without Aura It is estimated that one in seven adults worldwide has migraine.
- It impacts **women** more than men in a 2:1 ratio.
- Migraine may start in childhood and manifest occasionally with abdominal symptoms (“abdominal migraine”).
- Motion sickness in children is a risk factor for the development of migraine
- Typically begins in adolescence or early adulthood.
- They can be episodic or chronic

Triggers

- Migraine has numerous identified triggers, including weather changes, menses, and caffeine (both withdrawal and overuse).
- Many patients identify foods and drinks such as alcohol (most commonly red wine), soft cheeses, and nitrite-heavy foods, such as processed meats, as precipitants, but data are sparse in this area and many migraine attacks occur without identifiable triggers.



Symptoms

Throbbing or pulsing headache, often unilateral.

Duration: 4 to 72 hours.

Associated symptoms: nausea, vomiting, sensitivity to light and sound.

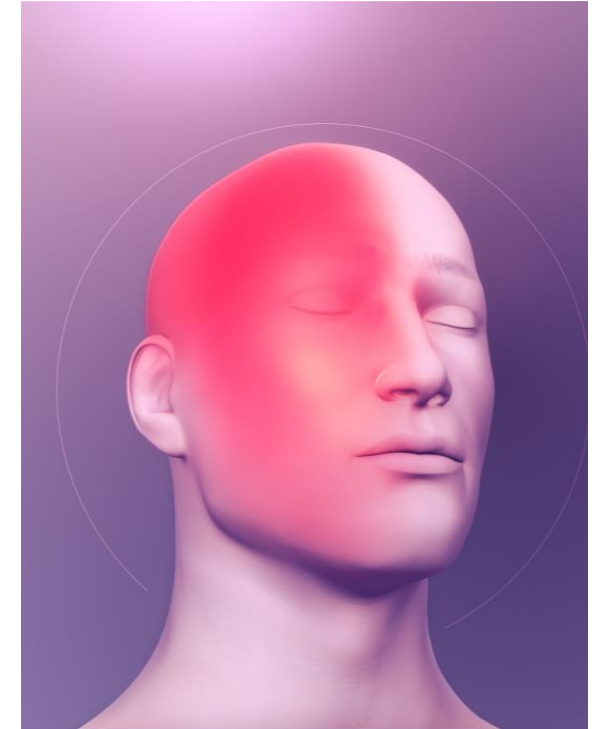
DIAGNOSIS

To diagnose migraine, a patient must have at least five attacks with the following characteristics:

- 1. The headache lasts for 4 to 72 hours if untreated.
- 2. It must include at least two of the following features:
 - Throbbing
 - Unilateral headaches
 - Worsening with activity, such as (walking or climbing stairs)
 - Moderate to severe pain

3. It must be associated with at least one of the following:

- a. Nausea, vomiting, or both
- b. Photophobia and phonophobia



Simplified Diagnostic Criteria for MIGRAINE

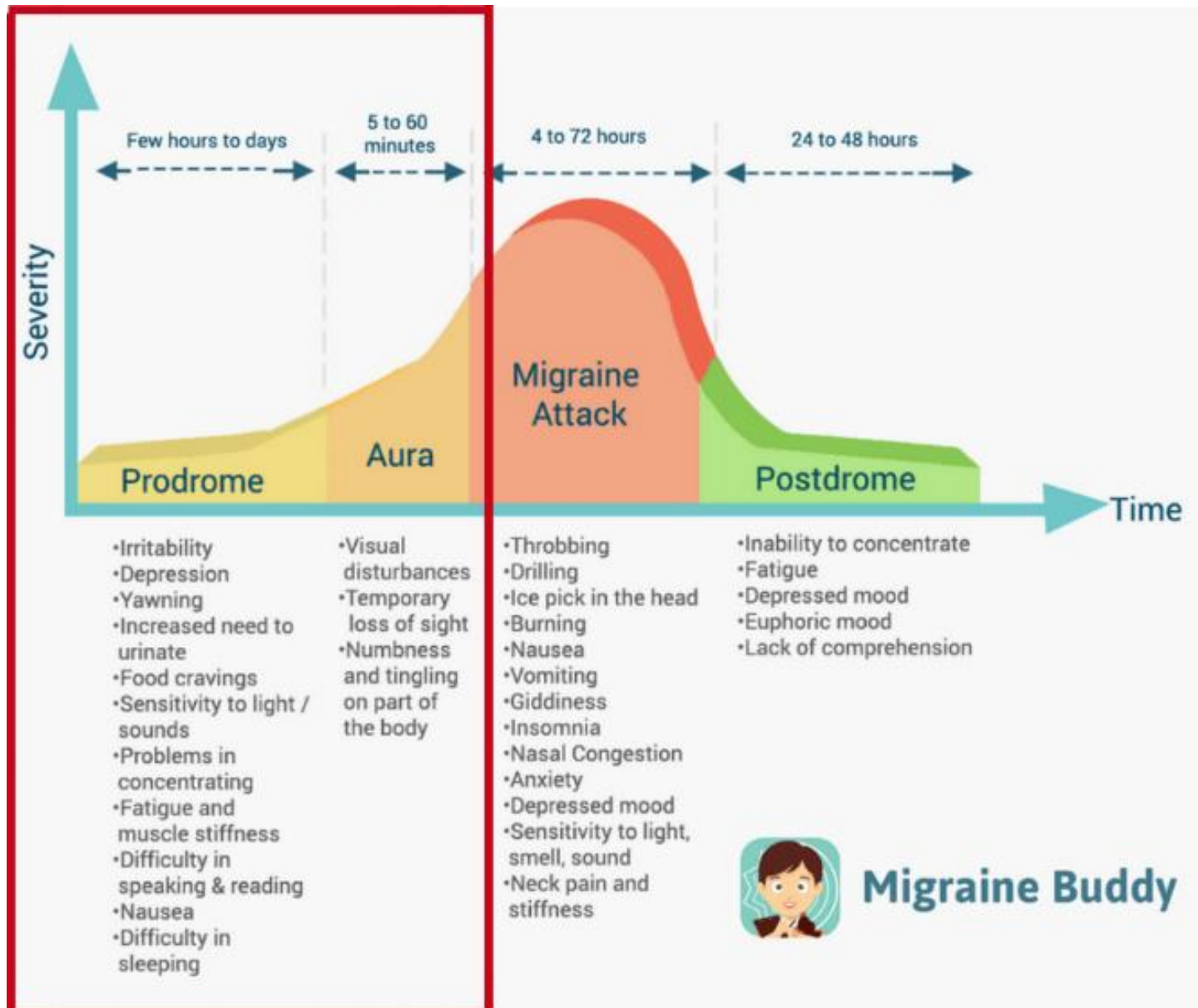
<i><u>At least 2 of the following</u></i>	<i><u>+ At least 1 of the following</u></i>
○ Unilateral pain ○ Throbbing pain ○ Aggravation by movement ○ Moderate to severe intensity	○ Nausea/vomiting ○ Photophobia and phonophobia

With this type of migraine attack, you don't experience any sensory disturbances.

Instead, you might experience symptoms during what's called the **premonitory or prodrome** phase before the migraine attack. These symptoms may include food cravings, constipation, fatigue, and sensitivity to light or sound.

A moderate to severe migraine headache then follows, accompanied by pain on one side of the head or throbbing pain.

In a postdrome phase — sometimes called a migraine hangover — you may feel depressed and fatigued and have difficulty concentrating and comprehending.



Phases of migraine attack

COMPLICATIONS ASSOCIATED WITH MIGRAINE

Status Migrainosus

When migraine lasts for more than 72 hours, the condition is known as status migrainosus. This is often caused by abortive medication overuse (often referred to as rebound headache) and frequently requires intravenous (IV) treatment or a brief course of oral steroids to break the headache cycle.

Stroke Risk Associated with Migraine Patients with migraine with aura have an increased cardiovascular risk when compared to healthy controls. The use of estrogen-based contraceptives is therefore contraindicated in patients with migraine with aura, as the combination results in a substantially increased stroke risk.

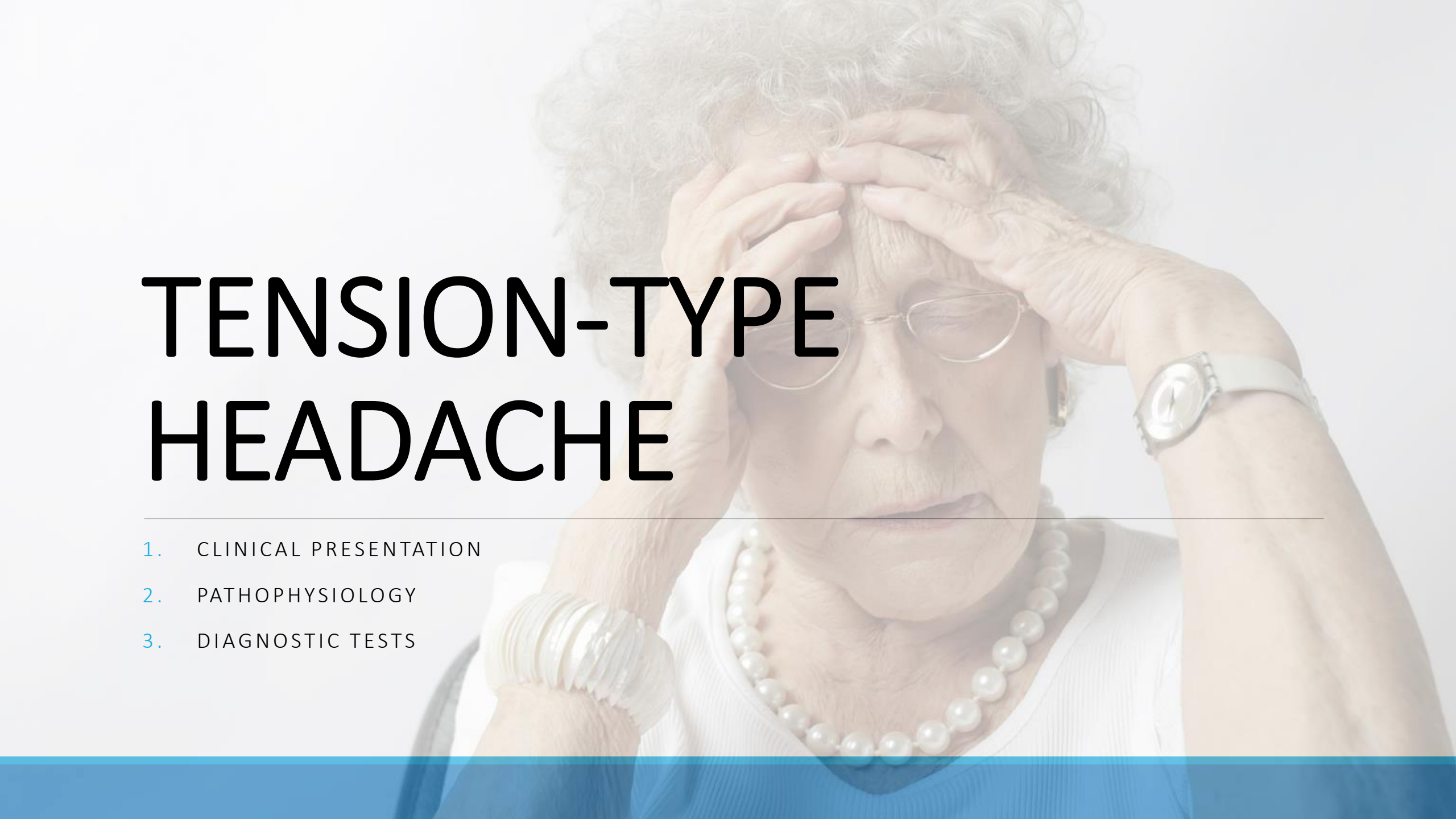
Cont,

Migraine and Menses Women of reproductive age frequently have exacerbations of migraine during menses, most commonly 1 to 2 days prior to the onset of bleeding, often persisting for up to 3 days into bleeding. This is thought due to the withdrawal of estrogen that occurs with menses. Some women have migraine at the time of menstruation only, a condition known as pure menstrual migraine. Most, however, have a few episodic headaches at other times of the month, or menstrually related migraine. It is important to identify the relationship of menses to migraine because there are specific treatments that may be helpful for patients with a clear exacerbation around their menses.

Cont,

Chronic Migraine Patients who have a headache more than 15 days/month for more than 3 months are diagnosed with chronic migraine. Some patients with chronic migraine do not have typical features of migraine with all headaches, but they must have at least 8 days of headache consistent with migraine to be diagnosed with chronic migraine. If the headaches are not consistent with migraine, other diagnoses must be considered.

Patients often describe a history of gradually progressive episodic migraines that increase in frequency to the point of meeting criteria for chronic migraine. With frequent headaches, many patients with chronic migraine have some component of medication overuse headache (MOH). Importantly, patients with chronic migraine can revert to episodic migraine after effective treatment.

A photograph of an elderly woman with short, curly white hair, wearing glasses, a white pearl necklace, a silver watch, and a white bangle. She is holding her hands to her forehead, suggesting a headache. The image is semi-transparent, allowing the text to be overlaid.

TENSION-TYPE HEADACHE

-
1. CLINICAL PRESENTATION
 2. PATHOPHYSIOLOGY
 3. DIAGNOSTIC TESTS

TENSION-TYPE HEADACHE

Are the next most prevalent primary headache disorders, occurring in 30% to 70% of adults worldwide.

Typically present with the following clinical features:

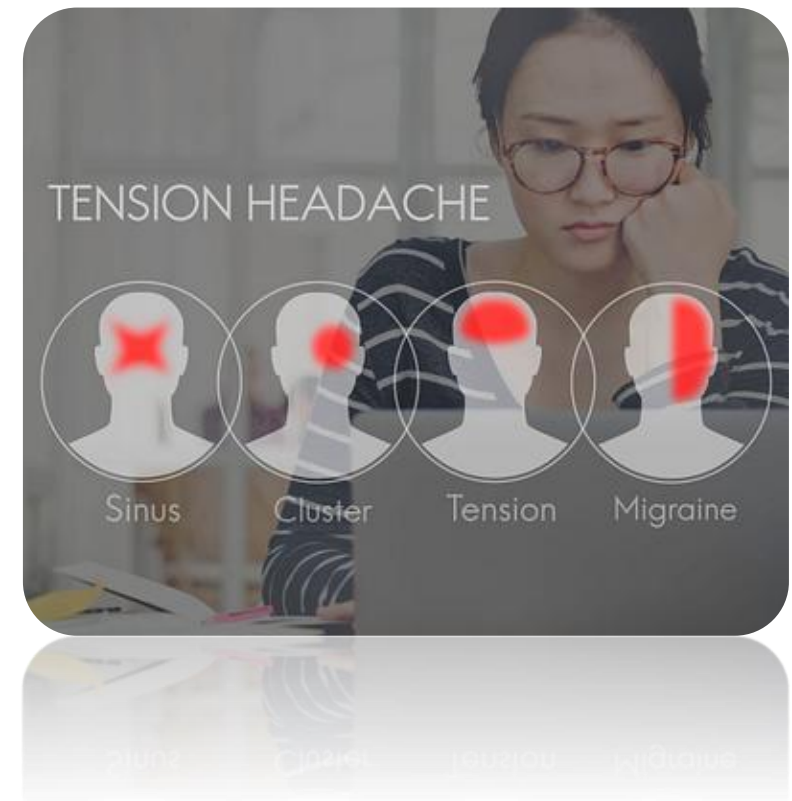
❑ Pain characteristics:

Location: bilateral, often described as a band – like or pressure sensation around the head (like a “tight band”).

Intensity: mild to moderate intensity .

Duration: can last from 30 min to several hours or even days .

Unlike migraine, it is not associated with aura, photophobia, phonophobia, nausea, or vomiting



TENSION-TYPE HEADACHE

❑ Examination findings :

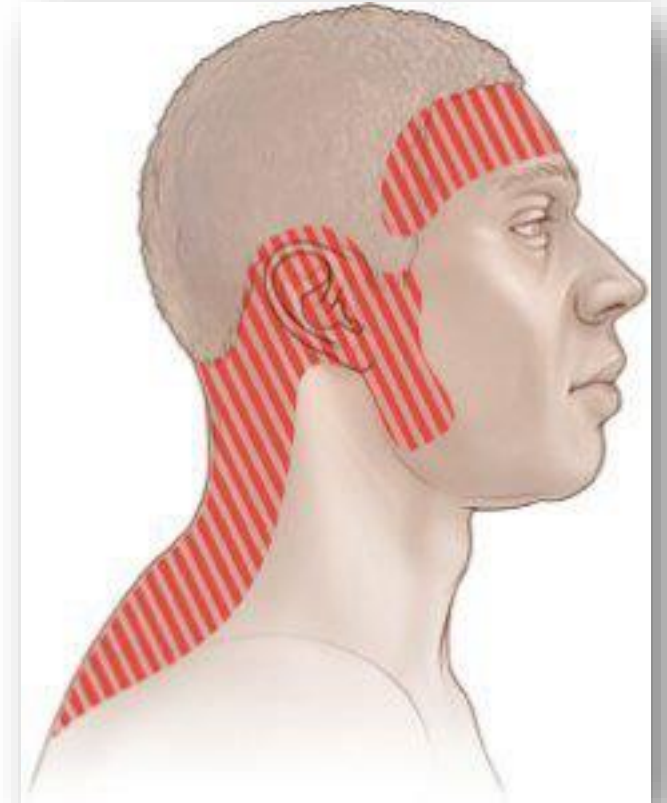
The neurological examination is generally normal, but some patients have [peri-cranial tenderness to palpation](#) of the scalp, neck, or shoulder muscles, indicating muscle tension.

❑ Frequency:

(TTH) classified based on frequency into the following types:

[episodic tension headache](#) : less than 15 day per month.

[chronic tension headache](#) : more than 15 days per month.



TENSION-TYPE HEADACHE



- **the pathophysiology of tension type headache** is not fully understood , but several triggers and aggravating Factors can contribute... Like:

- Stress, Anxiety.
- Fatigue.
- Depression.
- sleep disturbances.
- poor posture or long period of sitting in one position (like working on computer).



TENSION-TYPE HEADACHE

There are no specific diagnostic tests that definitively confirm tension-type headaches (TTH). Instead, the diagnosis is largely clinical and based on a **medical history and physical examination**. **Diagnostic tests are generally used to rule out other causes of headaches** or to confirm there are no underlying conditions...

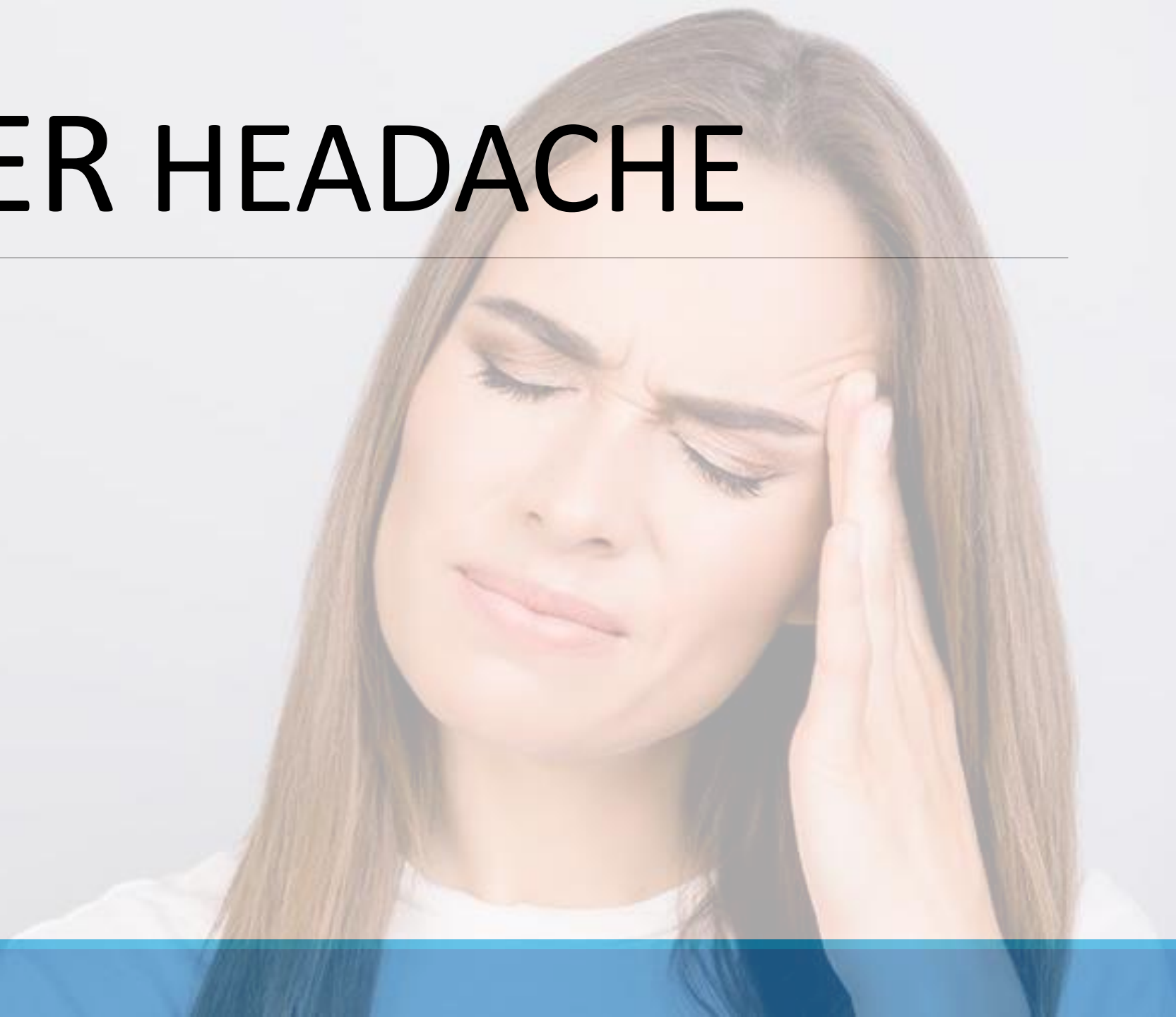
some of these **diagnostic tests:**

- **CT scan or MRI** : These are generally **not necessary** for typical tension-type headaches, but they may be ordered **if there are atypical features (e.g., sudden onset, neurological symptoms, or progressive worsening of headaches)** to exclude structural brain abnormalities (tumors, hemorrhage, stroke).
- **Blood tests** : to check if the patient has **metabolic disorder** like ...(thyroid dysfunction , anemia).



CLUSTER HEADACHE

1. Clinical presentation
2. Pathophysiology
3. Diagnostic tests





CLUSTER HEADACHE

Clinical presentation of cluster headache :

1. Severe unilateral headache:

- Location : The pain is typically located around or behind one eye, the temple, or forehead, but always on one side of the head (**unilateral**).
- Pain intensity : Described as **excruciating or stabbing** and is often regarded as one of the most painful types of headache.
- Duration : Cluster headaches typically last between **15 and 120 minutes**.
- Frequency: Attacks occur in clusters, typically **1 to 8 times per day**. Cluster periods can last weeks to months, followed by **remission periods** (with no headache) that **can last months to years**.

Cluster headaches

Symptoms of cluster headaches include:



Pain on one side of your head.



Watery eyes.



Eye redness.



A runny or stuffy nose.



Droopy eyelid.



Flushing or sweating.

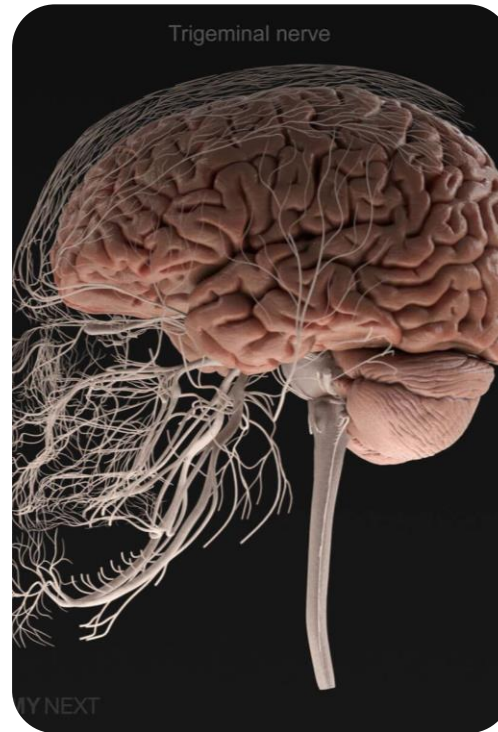
CLUSTER HEADACHE

2. Autonomic Symptoms (Ipsilateral to the Pain):

✓ Autonomic symptoms occur on the same side as the headache and include:

- ❖ Lacrimation: Excessive tearing of the eye.
- ❖ Conjunctival injection: Redness of the eye.
- ❖ Rhinorrhea: Runny nose.
- ❖ Nasal congestion: Blocked nostrils.
- ❖ Ptosis: Drooping of the eyelid.
- ❖ Miosis: Constriction of the pupil.
- ❖ Forehead and Facial sweating or flushing.

CLUSTER HEADACHE



Pathophysiology of cluster headache :

The pathophysiology of cluster headaches is not fully understood, but it involves several key mechanisms, including:

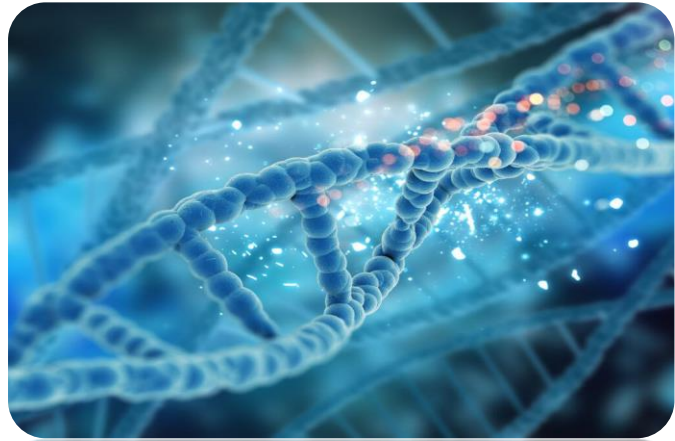
1. Hypothalamic Dysfunction:

- Central Role of the Hypothalamus: The hypothalamus, specifically the [posterior hypothalamus](#), is believed to be the key driver of cluster headaches. This region is associated with the [body's circadian rhythms](#), explaining why cluster headaches often occur at the same time of day and during specific times of the year.
- Functional imaging studies have shown increased activity in the posterior hypothalamus during cluster headache attacks, which is believed to drive imbalances in parasympathetic and sympathetic outflow.
- the overactivity of the parasympathetic system and the suppression of the sympathetic system lead to array of autonomic symptoms that accompany the intense pain of cluster headaches.

2. Trigeminal-Autonomic Reflex:

Trigeminal Nerve Involvement: Activation of the trigeminal nerve (particularly the first division, which innervates the forehead and eyes) is a hallmark of cluster headaches. This leads to [the release of neuropeptides](#) like calcitonin gene-related peptide ([CGRP](#)) and [substance P](#), which cause [vasodilation of blood vessels around the brain](#), contributing to the severe pain.

CLUSTER HEADACHE



3. Genetic Factors:

Although cluster headaches are typically sporadic, there is some evidence suggesting a genetic component. **First-degree relatives of individuals with cluster headaches** have a **higher risk** of developing the disorder.

Diagnostic tests of cluster headache:

The diagnosis of cluster headache is primarily clinical, based **on patient history and physical examination(ptosis , miosis , lacrimation...)** . While there are no specific tests for diagnosing cluster headaches, MRI and other imaging techniques are often used to rule out structural or vascular abnormalities. Blood tests, lumbar puncture, and other specialized tests are used to exclude secondary causes of headache when there are atypical features or "red flags."

SUMMARY:

TABLE 10-1. Key Features of Primary Headache Disorders

	Episodic Migraine	Episodic Tension	Episodic Cluster
Sex	Female > male	Female > male	Male > female
Location	Unilateral > bilateral	Bilateral (band around the head)	Unilateral (behind or around the eye)
Quality	Throbbing, pulsatile	Dull pressure or tightening (vice- like)	Stabbing, burning, boring
Severity	Moderate to severe	Moderate	Severe
Attack duration	4–72 h	30 min–7 d	15–180 min
Attack frequency	Variable	Variable	From 1 every other day to 8/d

Secondary headache

Overview

Secondary headache disorders are headaches caused by a medical condition or medication. We have some warning signs and symptoms for secondary headache and can be remembered by the acronym SNOOPPP, this acronym says that the headache is abnormal and it identifies if it's serious or not. Treatment is based on addressing the underlying disorder. There are six major categories of secondary headaches that may present with headache only and must be considered.

S → Systemic symptoms ex: fever / myalgia / weight loss
→ secondary risk factor ex: HIV / malignancy / immuno-suppression

N → neurological deficit

O → old ≥ 80

O → onset is abrupt or acute "thunder clap"

P → papillary edema

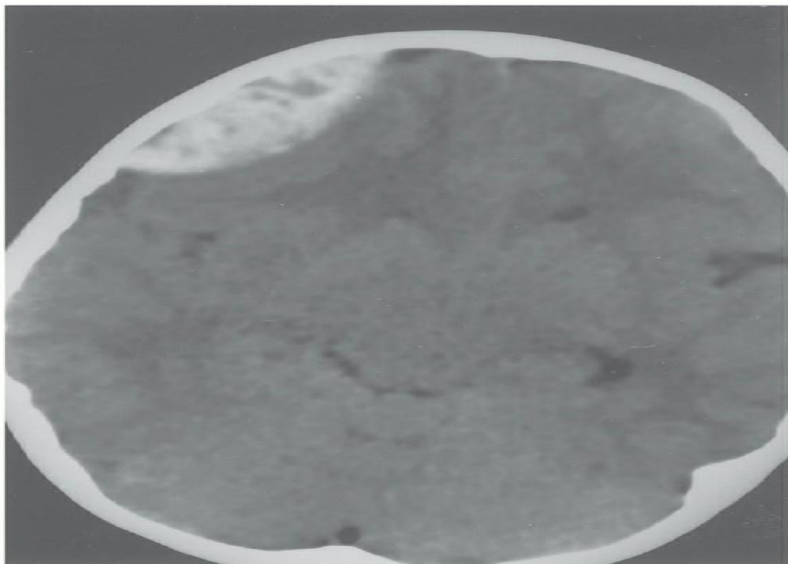
→ positional

→ Precipitation by Valsalva maneuver

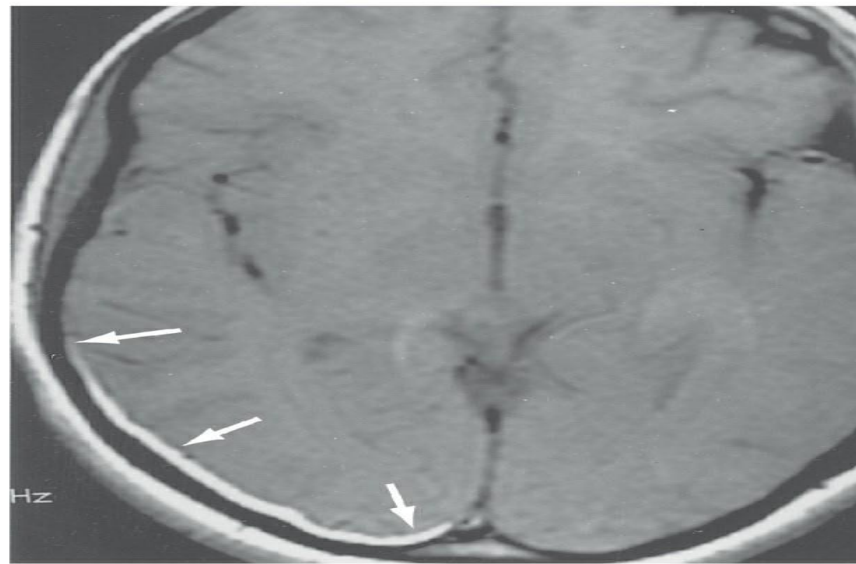
Vascular causes

There are numerous vascular causes of headache. All cerebral hemorrhages can cause headache. This includes subarachnoid hemorrhage (SAH), intraparenchymal hemorrhage, and subdural and epidural hematomas. These hemorrhages may be spontaneous (associated with stroke or hypertension) or traumatic. Patients with intracerebral hemorrhages typically present with what is referred to as a **thunderclap headache**; the onset is abrupt and severe. Emergency imaging, usually with a noncontrast head computed tomography (CT), is needed to evaluate any abrupt-onset headache. Cerebral vessel imaging is also warranted if a SAH is identified, to assess for an aneurysm.

Ischemic strokes are often associated with headaches. Their semiology is nonspecific, but typically abrupt in onset. Patients may have focal neurologic deficits which aid in the diagnosis. Cerebral thrombosis, either arterial or venous, can also cause headache. Patients with venous sinus thrombosis often have headaches with features of increased intracranial pressure (ICP). Thrombosis should be considered particularly in patients with hypercoagulability states, including pregnancy. The diagnosis is made on imaging, including that of cerebral vessels.

A

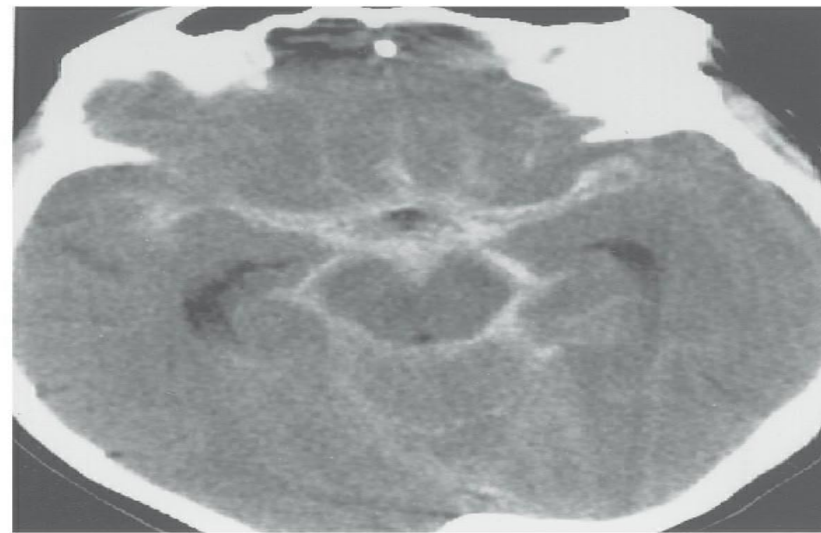
Epidural

B

Subdural

C

Intraparenchymal

D

Subarachnoid

Continued...

Cerebral vasculitis frequently causes a nonspecific headache. When part of a systemic vasculitis, it is considered a **secondary angiitis**. If the vasculitis occurs in the cerebral vessels alone, it is referred to as **primary CNS angiitis**. In addition to headache, patients often have paroxysmal focal neurologic deficits. Cerebral arterial vessel imaging and lumbar puncture (LP) are often required to make this diagnosis; peripheral vessel biopsies may be necessary.

Giant cell arteritis (GCA), also called temporal arteritis, is a peripheral cranial arterial vasculitis that often presents with a unilateral headache. Patients are generally above the age of 50 and report additional symptoms including vision changes (amaurosis fugax), jaw claudication, fever, and scalp tenderness. Involvement of the branches of the external carotid artery, including the ophthalmic artery, can result in blindness if not readily identified and treated promptly. Patients usually have elevated inflammatory markers (erythrocyte sedimentation rate and C-reactive protein). Empiric steroids should be started in any patient with a high clinical concern. Temporal artery biopsy is the gold standard, but GCA can cause “skip lesions” and may require serial biopsies to identify the pathology.

Infectious and inflammatory causes

Intracranial infections, such as encephalitis and meningitis, usually present with headache and often with fever. They may also have nuchal rigidity and altered mental status. As the infection progresses, seizures and focal neurologic deficits may occur. Infections can be bacterial, viral, fungal, or parasitic; the headache semiology does not help to differentiate the underlying cause. LP is the crucial diagnostic test and necessary in any patient for whom there is concern for a CNS infection. There are often other signs of infection, and the headache has a temporal correlation to the infection. CNS inflammatory and autoimmune conditions such as sarcoidosis and lupus frequently present with headache.

Neoplastic causes

Intracranial neoplasms may present with headaches, especially when there is significant mass effect. The headache semiology may be nonspecific but may have features of intracranial hypertension, including waking the patient from sleep, being worse when supine, and worse with Valsalva maneuver. The headache may occur early or late with neoplasms and with any type of primary cancer.

Traumatic causes

Head and neck injuries often result in headache. To be attributed to trauma, the headache must develop with a temporal association to the injury. The severity of the injury does not necessarily correlate with the severity of the headache; even minor head injuries or whiplash may cause headaches. There is no specific headache semiology that helps with the diagnosis. In patients with a history of significant head or neck injury, it is important to assess for an intracranial hemorrhage or dissection of cervical vessels— which may require additional treatment.

Intracerebral pressure disorder

Intracranial hypertension and hypotension can cause headaches, but with markedly different semiologies.

Intracerebral hypertension may be “idiopathic” (most common in obese young women) or due to medications or systemic disorders. The headache is often described as worse when supine or sleeping (awakening the patient from sleep), or with Valsalva maneuver. It improves with standing. Patients often have other associated features, including papilledema, pulsatile tinnitus, or visual symptoms. Patients should have imaging to exclude a mass lesion or venous sinus thrombosis. If the imaging is unrevealing, the diagnosis is made with an LP when the patient is in the lateral decubitus position with legs extended. Intracerebral pressure (ICP) is elevated if above 200 mm cerebrospinal fluid (CSF). Acetazolamide is the first-line treatment for idiopathic intracranial hypertension (IIH). Patients require monitoring of their visual fields, and treatment for obesity is warranted when present.

Intracerebral hypotension may be spontaneous or traumatic. This headache improves when the patient is supine but worsens with standing. The pain is often most severe at the vertex and can be associated with neck pain or tinnitus. It is frequently traumatic—occurring after an attempted epidural puncture or LP. Generally, the dural leak causing the headache heals gradually, without intervention, but when symptoms persist or when the headache etiology is unclear, brain MRI may be helpful; . The definitive diagnosis is made with an LP showing an opening pressure below 60 mm CSF. If a patient remains symptomatic from intracranial hypotension, a blood patch may be attempted to cover the dural leak (if it can be found).

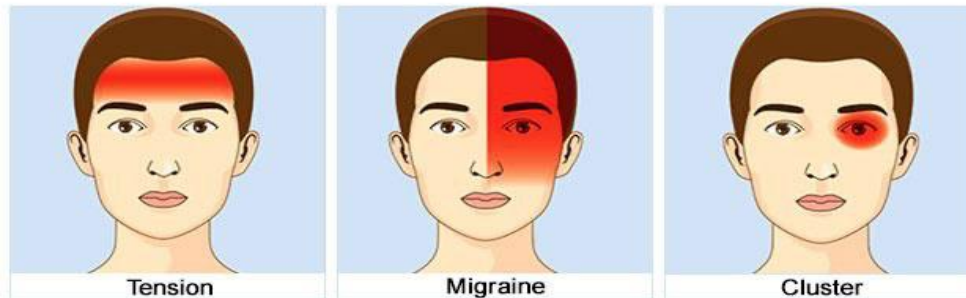
Medication

Medications associated with headaches are numerous. They range from hormonal therapies, including contraceptives, to nitric oxide. Withdrawal of a medication or other treatment may also cause a headache; the most common example is a caffeine withdrawal headache. Headache semiology is nonspecific, but the temporal association to medication change helps establish the diagnosis.

Medication overuse headache (MOH), also referred to as rebound headache or drug-induced headache, is a chronic headache occurring in patients with a primary headache disorder. The baseline headache disorder is typically markedly exacerbated (i.e., more frequent, severe, or both) when the medication is overused. MOH is diagnosed when patients use an abortive therapy 10 or more times per month for more than 3 months with an increase in headache

Treatment principles of headache.

Headache Types



Important definitions:

A) Abortive treatments

Also called rescue medication, these medications used to stop a headache at the onset and, all abortive treatments are the most effective if the patient is treated at the onset of headache.

B) Preventive treatments

Also called prophylactic treatments, are used for patients with chronic headache or frequent headaches that don't respond to abortive treatment, and they aim to reduce the frequency and severity of headache.

C) Adjuvant treatments

These are treatments used to reduce nausea and emesis associated with headaches.

NOTE : the treatment is dependent on the diagnosis

Migraine treatments

1. The abortive treatments of migraine:

NSAIDs and Triptans are the mainstay of abortive treatments.

Many patients respond well to NSAIDs alone.

Some patients have **contraindications** to use NSAIDs . In these cases , Triptans can be highly effective.

Triptans and NSAIDs can be combined when needed and may have a synergistic effect to treat migraines pain .

Using abortive treatments on chronic basis more than twice a week can result in **Medication-overuse headaches (MOH)**

- NSAIDs

- Triptans
(serotonin 1b/1d agonist)

CONT.....

2. Adjuvant treatments :

Prochlorperazine and Metoclopramide use to **decrease nausea and severity of the pain**

they useful in **preventing patients from vomiting their abortive drugs.**

- **Prochlorperazine**
- **Metoclopramide**

CONT

3. Preventive treatments:

• Oral drugs

All preventive treatments take some time to have an effect .

Selecting the right prophylactic medication requires consideration of the patient's **Co-morbidities, concomitant medications, cost and family planning goals.**

TABLE 10-2. Migraine Prophylaxis Oral Medications

Antihypertensives

Metoprolol

Propranolol

Timolol

Antiseizure Drugs

Sodium valproate

Topiramate

Antidepressants

Amitriptyline

Venlafaxine

Medications in **bold** have level A evidence for efficacy. Medications in italics have level B evidence for efficacy.

Cluster Headaches Treatments

1. Abortive treatments:

A first line abortive treatment for cluster headaches is 100% Oxygen.

For patients who do not respond, or who do not have access to home oxygen, **triptans are prescribed**.

Note : patients with cluster headaches very susceptible to (**MOH**)

- 100% oxygen

- **Triptans**
Sumatriptan
Zolmitriptan

2. Preventive treatments :

Verapamil is the first-line therapy for cluster headache prophylaxis.

Preventive treatments for cluster headache are **similar to those used for migraine and include** antihypertensive, antiseizure, and psychiatric medications

Tension-Type headache treatment.

1. Abortive treatments :

Many patients with tension headaches don't require abortive treatments because the pain is **generally mild** and not affects on patient's functioning.

Doesn't use for more than twice a week for long periods (**MOH**)

2. Adjuvant treatments:

Tension headaches are often reported to be triggered by **stress (physical or emotional)**; if chronic, is important. Biofeedback (a mind-body technique used to teach patients greater body awareness and how to control some physical reactions to pain and stress) can be effective.

Poor posture and neck muscle spasm are also frequent contributors to chronic tension-type headaches, and **physical therapy can help**

▪ **NSAIDs**
(moderate to severe)

▪ **Aspirin and Acetaminophen**
(mild)

Tension-Type headache treatment.

3. Preventive treatments :

A) **Antidepressants** are the **best** therapy for chronic tension headaches.

- **Tricyclic amitriptyline** is the **1st line** treatment
- **Mitrazapine** and **Venlafaxin** are the **2nd line** treatments

B) **Muscle relaxants** such as **Tizanidine** are helpful sometimes.

Other approaches to headaches treatment :

1. Lifestyle Modifications:

- Regular exercise, adequate sleep, and stress management practices

2. Monitoring and Follow-Up:

- Regular check-ins to assess treatment effectiveness and adjust as necessary.

3. Patient Education

- Teach patients about potential triggers, lifestyle changes, and self-management techniques.

Note: These principles should be individualized based on the patient's specific needs and headache characteristics.

* 14y old girl came to neuro clinic with severe headache for years.

Patient complain of severe headache all over the head and both eyes . And the pain starts both suddenly and gradually depending on the day . The patient Described it as tension pain . It's constantly and Not relived by medication. she has photophobia and phonophobia. The pain increases when speaking or using the phone. She prefer being in a Quite room .

It associated with nausea start with headache and associated with blurred eyes
No vomiting No cough No SOB She doesn't smoking . And has no allergies .

DONE BY :



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Thank You
