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Review Article

Headache, its Type & Migraine: A Clinical Review of Diagnosis and Management

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Abstract

Headache is a common clinical presentation in medical practice, both as an outpatient and an inpatient. It may manifest an underlying serious condition or be related to common but non-sinister causes. It usually becomes a headache for clinicians for diagnostic and management purposes. It's also an economic burden for the patient and the healthcare system. It also has many physical and psychological implications for patients, especially if not diagnosed or treated.

Migraine is a common type of headache that can be confused with many conditions, such as stroke. It's especially important for our trainees and junior clinicians, who often encounter such cases in the emergency department and OPD. It has many subtypes and varies a lot in severity and clinical presentation.

Therefore, this review article focuses on this important issue to summarize salient clinical points of headache and migraine to make the presentation understandable and clinically manageable. The management of migraine is elaborated in detail, including the latest advances in its treatment.

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Headache

The first & most important thing to be excluded is any intracranial cause of a headache by excluding three aspects of it:

1. Signs or symptoms of meningeal irritation: If these signs are present with inflammatory symptoms, they indicate meningitis. Sudden onset and meningeal irritation signs present without inflammatory symptoms indicate subarachnoid hemorrhage (SAH). The patient needs immediate CT / MRI and then an LP.
2. Signs of Raised ICP; if present, it indicates hydrocephalus, space-occupying lesions (SOL), or cerebral edema. It is usually worse after lying or sleeping. These patients may have papilledema on fundoscopy. The patient needs immediate brain CT or MRI.
3. If focal neurological signs of any type are present, they indicate CNS pathology that needs immediate brain imaging. This could be due to hemorrhage, stroke, encephalitis, tumor, or space-occupying lesions—weakness, numbness, visual, etc.
4. Loss of consciousness, syncope, fainting, seizures, or brainstem signs (vertigo, diplopia, nystagmus, etc
5. Carotico-cavernous fistula is a rare cause of a headache due to the fistula b/w the carotid artery and cavernous sinus, presenting with a headache, a bruit over the orbital area, cranial neuropathy affecting 3,4,5, 6 & ophthalmic branch of the 5th cranial nerve as they travel through the cavernous sinus. CT angiography detects it.[1,2]

If it's not due to the above, look at each structure in the head and neck, including the skull.

C-spine, eyes, ears, nose, teeth, throat, TM Joint, temporal arteries, trigeminal nerves, etc, can cause headaches. The nerve supply for most of these structures is from the trigeminal nerve or C2 and C3 on the neck and occipital area, so pathology in any of these areas can present with a headache. So, a

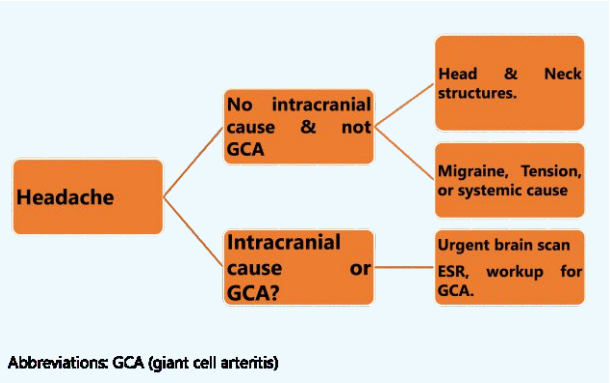
detailed, thorough assessment of head and neck anatomical structures, like ENT, visual assessment, teeth, cervical muscles, cervical spine, etc., is essential. Nerve pain can be hyperesthesia or hypoesthesia and may involve a neuronal deficit in the nerve territory. Trigeminal nerve-related issues are discussed separately.[3]

Trigeminal neuralgia pain is brief episodes lasting seconds to a few minutes and worsens with non-painful stimuli such as touching the skin, blowing air on the cheeks, brushing the teeth, chewing, etc. Hyperaesthesia is an important finding that differentiates it from other local causes. Aneurysm irritating the trigeminal nerve is an imprudent cause that MRI and MR Angiography should exclude.[4]

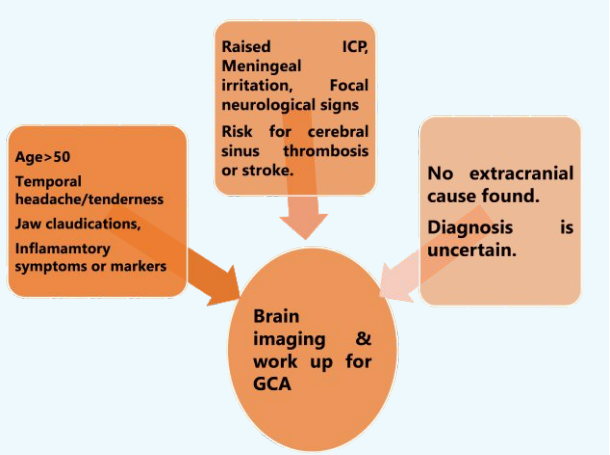
Temporal arteritis is an important cause to keep in mind in anyone who is 50 or above and has a headache with any of the following clues: constitutional symptoms, polymyalgia rheumatica, visual or neurological loss (TIA or stroke), temporal arterial tenderness or cord-like feel on palpation, raised ESR / CRP. Even if all of these clues are normal, it can still be temporal arteritis if the patient has a headache of 50 or above. It may have polymyalgia rheumatica, which is stiffness of the shoulder muscles and pelvic muscles & joints.[3,4]

If none of the above, think of systemic causes like fever, constipation, cough, and drugs. Think of depression/stress, migraine, cluster headache, etc. Sometimes, a migraine or a stress headache is a diagnosis of exclusion.

Migraine can also be associated with focal neurological s/s due to vasospastic elements, but these deficits are reversible. Migraine can present just with neurological deficits without headache. It is commonly confused with TIA/ Stroke. The patient may have the first episode of complex migraine presenting with a neurological deficit. So, an absent history of migraine doesn't exclude migraine as a possibility, but of course, this is a diagnosis of exclusion. A typical migraine may have an aura (warning symptoms due to vascular/chemical brain dysfunction). Usually unilateral, throbbing, associated with nausea/vomiting, worse with activity, and lasts 2-48 hours. However, chronic migraine is a well-known entity that may last for a week or more. It can be occipital or retro-orbital as well.[5]



Abbreviations: GCA (giant cell arteritis)



Abbreviations: GCA (giant cell arteritis), ICP (intracranial pressure)

Psychiatric/Psychogenic causes like Tension headaches, depression, etc., are important and very common, especially in long-standing headaches, which are more like constant (24/7) pressure-like headaches, more common in females. It's often occipital or band-like, like encircling the whole head.[6]

Who needs brain imaging?

1. Focal neurological signs, raised ICP, or meningeal irritation.
2. Clues for a secondary cause such as cancer, autoimmune, infection, vascular, etc.
3. New onset of headache or change in the headache pattern.
4. Old age, demented cases, those at high risk for malignancies, or vascular events.
5. Treatment-resistant or worsening headache.

Migraine: Migraine is a common cause of headaches, and they present in a wide variety of ways, both geographically and characteristically. Due to some neurochemical (such as serotonin) disturbances of the blood vessels or the nerves controlling these vessels, migraines cause abnormal vascular spasms and/or dilatation.[6,7]

Pathophysiology of migraine: Genetic predisposition leads to hypersensitization of the brain to various internal or external stimuli. The following are stepwise changes that start and maintain headaches.

1.These triggers stimulate the trigeminovascular system, which releases mediators such as CGRP (calcitonin gene-related protein) and other peptides.

2.These proteins & peptides cause inflammation, stimulation of the pain system & trigeminal nucleus activation in the brainstem, and changes in vascular tone (vasodilation).

3.The trigeminal nucleus in the brainstem activates the thalamus and sensory cortex and inhibits the descending pathways that normally suppress pain.

4.Trigeminal sensory fibers also stimulate histamine release from the meningeal mast cells, causing meningeal inflammation and irritation.

Thus, these mechanisms lead to the persistence of pain mediated by changes in the vascular tone, some degree of inflammation, and meningeal irritation. These CGRP and other peptides are a new target for drugs (antibodies) that neutralize or block their receptors.[8,9]

Clinical Manifestations & types of migraine

Classic migraine: It is a unilateral throbbing headache that lasts at least 2 hours & a maximum of 72 hours (unless it's chronic migraine). Two diagnostic hallmarks in the history are:

1.It is almost always worsened by physical activity, and the patient feels better or refers to staying in a dark, quiet room without doing any physical activity.

2.It's associated with nausea, vomiting, and & photophobia.

A classic example of a migraine case is a young female with a unilateral throbbing headache, nausea, vomiting, and photophobia who feels better in a quiet, dark place and hurts more with physical activity. Aura may or may not be present. However, classic cases are 20-30%, and most present with different patterns. Patients often recognize their headache and may predict when it's about to start.[10-12]

Aura: It is present in only 50-60% of cases and classically starts from the occipital lobe and marches anteriorly to the parietal lobe. It may also involve the frontal lobe.

Classic aura is visual symptoms followed by sensory symptoms. Symptoms appear in succession, and each symptom may last 1-5 minutes, followed by the next.

Rarely, frontal lobe symptoms such as aphasia, hemiplegia, or transient cognitive decline occur.

Rarely, the aura involves the brainstem, especially when the patient has migraine due to the basilar arteries. This basilar aura can cause vertigo, nausea, vomiting, dizziness, or impaired consciousness.

Aura symptoms last for 5 minutes to an hour.

How is it confused with TIA or stroke?

An MRI may also show some ischemic changes due to migraine. Patients with migraine often have small infarcts on MRI, which can look like chronic small vessel ischemia or a small old stroke. Spasm of the intracranial vessel leads to the aura or neurological deficits, such as TIA or, rarely, stroke-like manifestations.

Sometimes, spasm is the only phase without a dilatation phase, so the headache is not there. This is Migraine sine qua non. This is a reason any young person without risk factors for stroke presenting with acute neurological symptoms, suspecting stroke with headache, one D/D is hemiplegic migraine, which often creates confusion about thrombolysis.

Sometimes, these patients with hemiplegic migraine can get thrombolysis and make us think that the tPA has worked. So, stroke with headache, after ruling out dissection, migraine is another possibility.

Even if the patient is known to have migraine, it may still be a genuine stroke on top of migraine. So, a detailed clinical assessment and discussion with a neurologist are needed in such cases.

If the patient is at high risk of stroke, treat it as a stroke unless clinically confident that it's migraine.

Occipital or retro-orbital, the latter two are the basilar types of migraine that involve basilarovertebral circulation.[13-15]

Autonomic disturbances in migraine

Autonomic symptoms are common with migraine and especially affect the GIT, which makes patients feel nauseous, vomit, or may also have abdominal discomfort.

Drug absorption is impaired once migraine has started, so that's why oral drugs either take longer to work or don't work adequately. Patients should take medication at the warning or early stage.[15-17]

Variations in classic migraine: Variations from the

classic pattern are common and include the following:

Location: It may also be global, bilateral, occipital, or retro-orbital. A migraine can be frontal, on one side of the scalp, on both sides of the head, or occipital or retro-orbital. Later, two seem to be in the basilar type of migraine, which involves basilo-vertebral circulation and can also present with posterior circulation, such as the brainstem and cerebellum.

It may be a continuous severe headache or, rarely, continuous dullness.

Aura: Many patients with migraines don't have an aura; even if it is present, it may not be there before each episode.

Prodrome & / or postdrome: 50-60% of patients may have a prodrome of non-specific & temporary symptoms before the start of aura at the onset of migraine. This pre-prodrome includes fatigue, neck stiffness, depression, irritability, cognitive changes, euphoria, hyperexcitability, food cravings, etc. Similarly, some patients have a post-drome as well after the headache. This may be a feeling of rejuvenation and freshness or tiredness and exhaustion once the headache is over.

No headache- some patients have an aura, which is not followed by a headache.[18,19]

Cluster Headache / Eyes and nose symptoms: A cluster of headaches with symptoms such as teary eyes, runny nose, and an orbital/frontal headache is seen in a Cluster type of headache, which is common in males and often happens during sleep. The patient gets clusters of headaches and then is normal in b/w those clusters. A retro-orbital headache can also be present in a basilar migraine.[8,17,19]

Chronic migraine: It lasts for many days or weeks, or at least 15 days a month. Transition from acute migraine to chronic causes a loss of the normal pattern of migraine for the patient. This transformation may occur in months or years.

It's common in obese females who have pain in other body parts or overuse medications.

The majority of cases of migraine in a tertiary neurology clinic have chronic migraine.

Prophylaxis &/or treatment with more than one agent is often needed due to persistent or chronic pain.

Chronic migraine vs. Tension Headache: Chronic migraine can last as an ongoing pain for the week(s) & can be confused with tension headache. But migraines give headache-free days even if they are chronic, whereas tension headaches are like a partner (with you 24/7).[17-20]

Hemiplegic migraine: Migraine aura due to vasospasm can cause transient ischemia, presenting as aphasia or hemiplegia, etc. It can present as an acute stroke or TIA. It often resolves within a few minutes or hours. Many cases of code strokes turn out to be hemiplegic migraine. A history of migraine in the past or migraine-like headaches with neurological symptoms should raise this possibility. It's more difficult when the headache doesn't follow the aura (causing hemiplegia).

Neuro & vascular imaging is normal.

Patients with hemiplegic aura or migraine shouldn't be given non-selective beta-blockers for prophylaxis, as they can worsen vasospasm.[21-34]

Diagnosis of migraine: It's a clinical diagnosis, and the exclusion of other serious causes is needed when clinically appropriate. MRI of the brain may be required to exclude any stroke or any other cause of a headache, as clinically necessary.

Migraine treatment & prophylaxis [22-33]: Treatment: Pain medication should be taken immediately after an aura or headache starts. Delay reduces gut motility and absorption of the medicine, leading to reduced effectiveness and increasing the duration of onset of the drug's effect.

Panadol and antiemetics are often helpful.

Antiemetics control nausea and vomiting and may also prevent pain. Examples include metoclopramide, domperidone, chlorpromazine, and prochlorperazine. They also have a sedative effect.

NSAIDs are also effective, but GIT upset is a risk, especially as these patients are already nauseous or have vomiting. IV / IM can also be used, especially for patients having severe nausea or vomiting.
Corticosteroids are also used to abort acute attacks.

Serotonin agonists at 5-HT1b/d, such as triptan, are migraine-specific drugs and are effective.

All triptans cause vasoconstrictions & therefore, can cause chest tightness and fatigue or can worsen vascular pathologies (stroke, IHD, PVD, etc.). Thus, these should be avoided in patients with vascular diseases.

All triptans are effective, though their efficacy is not equal, and if one doesn't work,

another can be used.

Sumatriptan is the first drug in the group. It's available for oral, injectable, and nasal use. Eletriptan, zolmitriptan, etc., are among the seven drugs in the group.

Rizatriptan is the most effective, and Eletriptan is the second most effective drug in the group.

Ditans are 5-HT1f receptor agonists that don't cause vasoconstriction. Lasmiditan is available in 50, 100, or 200 mg strength. It's less effective than triptans and can also cause sedation.

Ergot derivatives (dihydroergotamine) can be taken orally, nasally, or IM. However, as they cause vasoconstriction, they should be avoided in hemiplegic migraine.

Opioids should be avoided due to dependency and the risk of rebound headaches. Opioids should be used only for a limited period in an acute setting when other medications are not effective.

Migraine Prophylaxis: It is recommended when a patient has 4 episodes/month or disabling or episodes/month or disabling or impairing work or quality of life.

Effectiveness of prophylaxis: Prophylaxis reduces the intensity and frequency of the attacks by at least 50%. Prophylactic drugs also work for chronic migraine.

The choice of prophylaxis depends on side effects, tolerance, affordability, and availability of the drugs. First-line prophylactic drugs include beta-blockers (propranolol, atenolol, metoprolol), topiramate, or candesartan. Second lines are antidepressants such as TCAs (tricyclic antidepressants), calcium channel blockers (flunarizine, Verapamil), or antiepileptics such as valproate. Other less commonly used drugs are gabapentin, venlafaxine, cyproheptadine, Verapamil, etc.

Pros & cons of prophylactic drugs:

Verapamil is good for Vestibular migraine but can cause bradycardia.

TCAs are effective but sedating.

Venlafaxine is non-sedative & usually tolerated well.

Cyproheptadine is also a sedative and good for children.

Topiramate and valproate are not safe in pregnancy.

Topiramate can cause nephrotoxicity, neuropathy, and weight loss.

Valproate is hepatotoxic, with vertebral column defects in the foetus and cytopenia.

Duration of prophylaxis: These prophylactic drugs are used for at least three months for the drugs to work properly before changing them if they are not effective. If effective, these should be given for 6-12 months & then we will try to assess if we can stop them.

Dose of prophylaxis drugs: Prophylactic drugs are started at low doses to avoid side effects, and the dose is increased gradually. All beta blockers are equally effective. Propranolol 40 mg BD can be increased to 80-160 mg BD or TDS based on pulse rate. Metoprolol can be started at 25-50 mg OD and then increased. Candesartan (an ARB) is effective, but other ARBs or ACE inhibitors are not recommended for prophylaxis. Amitriptyline (TCA) at a 10-100 mg / day dose is effective. SSRIs and SnRIs are not as effective as TCAs. Topiramate 200-400 mg /day or valproate 400-800 mg /day is routine.

CGRP blockers: These drugs are effective for prophylaxis, & Gepants are also used for aborting acute migraine.

Monoclonal antibodies against CGRP: These antibodies are prophylactic drugs only, as they are slow-acting medicines and ineffective for the acute phase.

Antibodies are against CGRP or its receptor. Eptinezumab, galcanezumab, & fremanezumab target CGRP, whereas erenumab is against its receptors.

Eptinezumab is a weekly IV infusion; the other three are monthly or weekly subcutaneous injections.

These antibodies are more effective than other non-CGRP options. Antibodies are effective prophylaxis against both episodic and chronic migraine.

They are used for treatment-resistant migraine. However, long-term safety is not yet established, especially for pregnant women, lactating women, children, and the elderly.

Gepants: These are oral drugs that work as CGRP receptor antagonists. Oral bioavailability and hepatoxicity are issues. In addition, these drugs interact with other medicines that are metabolized by the cytochrome P450. CGRP receptor blockers are

Gepant drugs (rimegepant, etc.) that can be used as prophylactic or acute treatment. However, they are not more effective than Triptans. These are options for patients who can't use triptans.

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Important clues for various types of headaches:

Headache Type	Main clues.	Additional clues may help if present.
Migraine	Worsens by any activity. Associated with nausea, vomiting, phonophobia &/or photophobia.	Aura Unilateral Throbbing/pulsating Migraine triggers are stress, the menstrual cycle, hypoglycemia, or insomnia.
Tension headache	Ongoing bilateral dull pressure / band-like pain of mild to moderate severity affects the whole head or the occipital region. Uneasiness or tenderness of the trapezius or neck muscles. It's not worsened by activity. No nausea, vomiting, photophobia, or phonophobia.	Depression, anxiety, fibromyalgia, and IBS.
Trigeminal autonomic cephalgia	Unilateral localized to the territory of the ophthalmic branch of the Trigeminal (orbit, forehead, or temple). **Localized autonomic clues:**Partial or complete Horner syndrome, conjunctival congestion, tearing, nasal stuffiness, and/or nasal discharge	Brief episodes. Cluster worsens with Alcohol, cigarettes, odour, heat, and GTN. Hemicrania worsens with neck movements or alcohol.
Trigeminal neuralgia	Sharp shock/cutting/burning pain. Aggravated by light touch or non-painful stimuli of the structures supplied by trigeminal nerves in >91-99% of cases (a pathognomonic feature of the trigeminal neuralgia). Triggers: touching or rubbing the skin, washing, drying, air blowing, talking, eating, facial shaving, brushing teeth, chewing, or jaw movements, etc. **No signs of trigeminal nerve deficiency.**	Triggers often cause a refractory period of seconds to minutes, during which the next trigger doesn't cause pain. 75% Maxillary or mandibular. Only 25% involve the ophthalmic or whole trigeminal nerve. Autonomic findings in the cranial area are rare and mild, do not have more than one symptom, and do not present with all the episodes of trigeminal neuralgia.
Trigeminal neuropathy	**Sensory &/or motor signs are there.** Pain in the distribution of the nerve or its branch(es) (same as trigeminal neuralgia). Non-noxious stimuli don't aggravate pain. Triggers are the same as those of trigeminal neuralgia.	Trigger zones in neuropathies are in the same location where the pain is, but trigeminal neuralgia has dissociation of the pain area from the trigger area.

Important clues for various types of headaches:

Headache Type	Main clues.	Additional clues may help if present.
Unilateral episodic headache with autonomic clues;	Episodes Duration <6 minutes & orbital or nasal congestion is SUNCT / SUBA. Episodes Duration <2 mins are trigeminal neuralgia. An episode duration of 2-30 minutes is likely paroxysmal hemicrania.	The episode duration is 14-180 minutes, limited to orbit, and improves with oxygen/triptan, which is Cluster. Episodes 4 to 72 hours with photophobia, phonophobia, nausea, vomiting, or worse with activity are migraine.
GCA (Giant cell arteritis)	Age 50 & above Temporal headache (unilateral, more common). Temporal tenderness or cord-like feeling. Raised both ESR & CRP (in 90%). ESR or CRP in 80%. Dramatic response to steroids.	PMR in 50% of cases. Biopsy yield is higher if biopsies are multiple and Doppler-guided. Ipsilateral visual or neurological deficits / TIA.

Authors' Contribution

AH, JA: Conception

AH, JA: Design of the work

AH, JA: Data acquisition, analysis, or interpretation

AH, JA: Draft the work

AH, JA: Review critically for important intellectual content

All authors approve the version to be published

All authors agree to be accountable for all aspects of the work

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