HEADACHE

APPROACH TO THE PATIENT

Headache: Introduction

- Headache is among the most common reasons patients seek medical attention.
- Primary headaches
  - Benign
  - Recurrent
  - No organic disease as their cause

- Secondary headaches
  - Underlying organic disease

- Primary headache often results in considerable disability and a decrease in the patient's quality of life.

Common Causes of Headache

Primary Headache	
Type	**%**
Tension-type	69
Migraine	16
Idiopathic stabbing	2
Exertional	1
Cluster	0.1

Secondary Headache	
Type	**%**
Systemic infection	63
Head injury	4
Vascular disorders	1
Subarachnoid hemorrhage	<1
Brain tumor	0.1

Headaches

Sinus: pain is usually behind the forehead and/or cheekbones

Cluster: pain is in and around one eye

Tension: pain is like a band squeezing the head

Migraine: pain, nausea and visual changes are typical of classic form

Primary Headache Disorders

- More common
  - Migraine, with or without aura
  - Tension type
  - Cluster

- Less common
  - Paroxysmal hemicrania
  - SUNCT (short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing)
  - Primary Stabbing Headache
  - Primary Cough Headache
  - Cold-stimulus
  - Benign cough
  - Benign exertional

Secondary Headache Disorders

- Associated with vascular disorders
  - Subarachnoid hemorrhage
  - Acute ischemic cerebrovascular disorder
  - Unruptured vascular malformation
  - Arteritis
  - Carotid or vertebral artery pain
  - Venous thrombosis
  - Arterial hypertension
- Associated with non-vascular intracranial disorder
  - Benign intracranial hypertension
  - Intracranial infection
  - Low CSF pressure
- Associated with noncepalic infection
  - Viral infection
  - Bacterial infection

Secondary Headache Disorders

- Associated with head trauma
  - Acute post-traumatic headache
- Associated with substance use or withdrawal
  - Acute use or exposure
  - Chronic use or exposure
- Associated with metabolic disorders
  - Hypoxia
  - Hypercapnia
  - Mixed hypoxia & hypercapnia
  - Dialysis

Anatomy and Physiology of Headache

- Relatively few cranial structures are pain-producing;
  - the scalp,
  - middle meningeal artery,
  - dural sinuses,
  - falx cerebri, and
  - proximal segments of the large pial arteries.
- The ventricular ependyma, choroid plexus, pial veins, and much of the brain parenchyma are not pain-producing.

Clinical Evaluation of Acute, New-Onset Headache

- In new-onset and severe headache, the probability of finding a potentially serious cause is considerably greater than in recurrent headache.
- Patients with recent onset of pain require prompt evaluation and appropriate treatment
- In most cases, CT or MRI study.
- In some circumstances, a lumbar puncture (LP)

eyes by funduscopy, intraocular pressure measurement, and refraction; cranial arteries by palpation is required

Headache Symptoms that Suggest a Serious Underlying Disorder

"Worst" headache ever

First severe headache

Subacute worsening over days or weeks

Abnormal neurologic examination

Fever or unexplained systemic signs

Vomiting that precedes headache

Pain induced by bending, lifting, cough

Pain that disturbs sleep or presents immediately upon awakening

Known systemic illness

Onset after age 55

Pain associated with local tenderness, e.g., region of temporal artery

Migraine

- Migraine is a neurovascular disease caused by neurogenic inflammation and characterized by severe, recurring headaches

- It usually characterized by the severe pain on one side of the head as compare to the pain in rest of the head second most common cause of headache,
- Women > men.

- It is usually an episodic headache associated with certain features such as sensitivity to light, sound, or movement; nausea and vomiting often accompany the headache.

Migraine contd...

- Headache can be initiated or amplified by various triggers, including

  glare, bright lights, sounds, or other afferent stimulation; hunger; excess stress; physical exertion; stormy weather or barometric pressure changes;

  hormonal fluctuations during menses;

  lack of or excess sleep; and alcohol or other chemical stimulation.

Classification of Migraine headache

1)Migraine without Aura or common migraine

Does not give any warning signs before the onset of headache.

It occurs in about 70 to 80% of migraine patients

2)Migraine with Aura

Give some warning signs " called aura" before the actual headache begins. Approximate, 20 to 30% migraine sufferers experience aura.

The most common aura is visual and may include both positive and negative (visual field defects) features.

Zigzag structure

Negative scotoma. Loss of local awareness of local structure

Positive Scotoma. Additional structures

One side loss of perception.

Classification of Migraine headache contd.

3)Retinal migraine- It involves attacks of monocular scotoma or even blindness of one eye for less than an hour and associated with headache.

4)Childhood periodic syndromes that involve *cyclical vomiting* (occasional intense periods of vomiting), *abdominal migraine* (abdominal pain, usually accompanied by nausea), and *benign paroxysmal vertigo of childhood* (occasional attacks of vertigo). They may be precursors or associated with migraine.

5)*Complications of migraine* describe migraine headaches and/or auras that are unusually long or unusually frequent, or associated with a seizure or brain lesion

Pathogenesis

Vascular theory-

- Intracerebral blood vessel constriction – aura
- Intracranial/extra cranial blood vessel vasodilatation-headache

Serotonin theory-

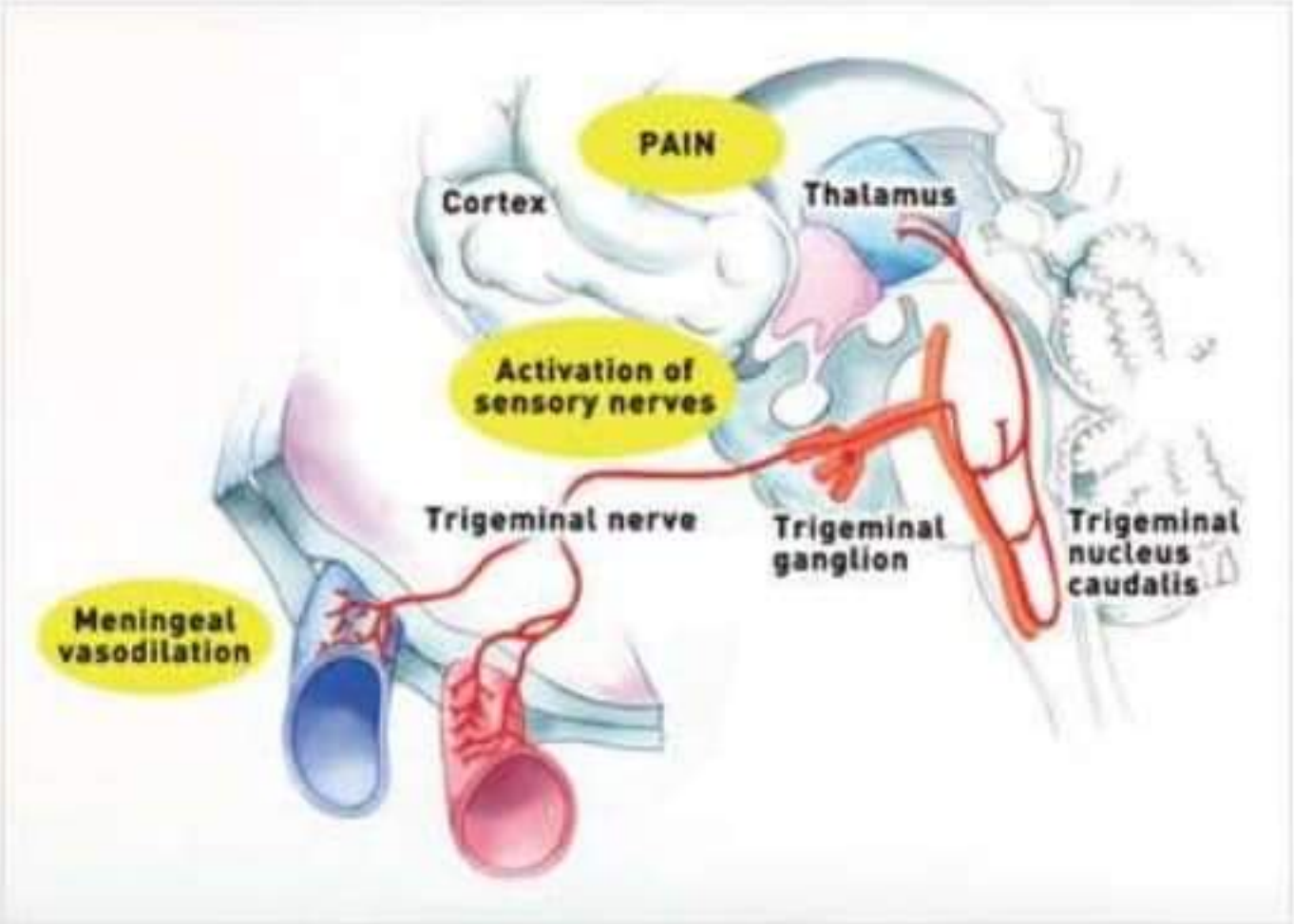
- Decreased serotonin levels linked with migraine
- Specific serotonin receptors found in blood vessels of brain

Pathogenesis

- Dysfunction of Activation of cells in the trigeminal nucleus results in the release of vasoactive neuropeptides, particularly calcitonin gene–related peptide (CGRP), at vascular terminations of the trigeminal nerve and within the trigeminal nucleus.
- Data also support a role for dopamine in the pathophysiology of migraine. Most migraine symptoms can be induced by dopaminergic stimulation. Moreover, there is dopamine receptor hypersensitivity in migraineurs.

- Mutations involving the $Ca_v2.1$ (P/Q)–type voltage-gated calcium channel *CACNA1A* gene are now known to cause FHM 1 **familial hemiplegic migraine**

Pathogenesis



Symptoms Accompanying Severe Migraine

Symptom	Patients Affected, %
Nausea	87
Photophobia	82
Lightheadedness	72
Scalp tenderness	65
Vomiting	56
Visual disturbances	36
Paresthesias	33
Vertigo	33
Photopsia	26
Alteration of consciousness	18
Diarrhea	16
Fortification spectra	10
Syncope	10
Seizure	4
Confusional state	4

TREATMENT GOALS FOR MIGRANT SUFFERER

Goals for Acute Migraine Treatment	Goals for Migraine Prophylaxis
Treat migraine attacks quickly and consistently	Reduction of migraine frequency by 50% per month
Improve quality of life	Reduction of migraine intensity and duration
Minimize the use of rescue medications	Increase response of acute attacks to abortive therapy
Optimize self-care for overall management	Eliminate or minimize adverse effects
Emphasize cost-effective drug therapy	Minimize the use of abortive therapy and rescue medications
Eliminate or minimize adverse effects	

Medscape

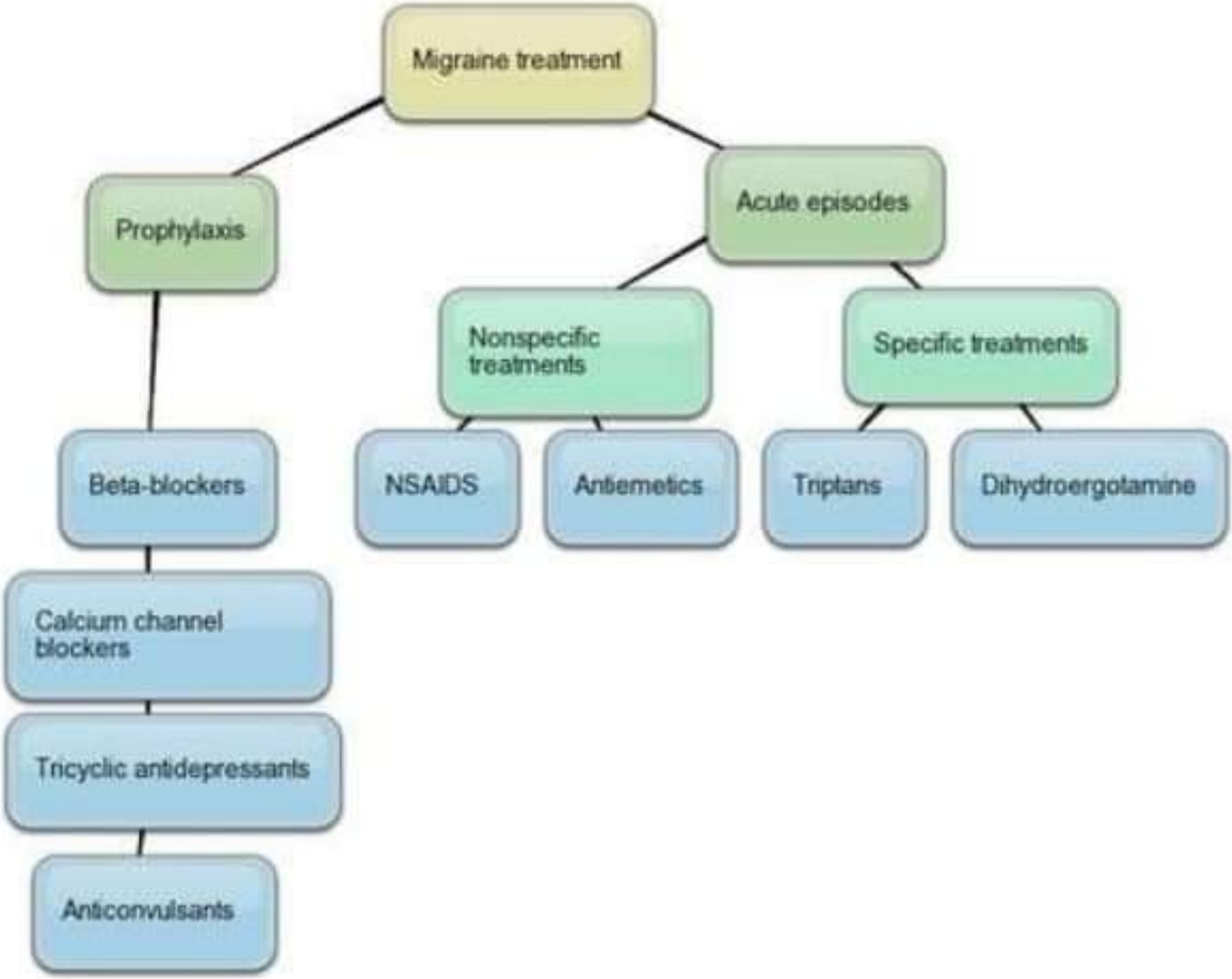
MIGRAINE MANAGMENT

Non pharmacological treatment

- Identification of triggers
- Meditation
- Relax techniques
- Psychotherapy

Pharmacological treatment

- Abortive treatment
- Preventive treatment


Migraine treatment
Prophylaxis
Acute episodes
Nonspecific treatments
Specific treatments
Beta-blockers
NSAIDS
Antiemetics
Triptans
Dihydroergotamine
Calcium channel blockers
Tricyclic antidepressants
Anticonvulsants

MODE OF ACTION

ERGOTAMINE

- Structurally similar to amines serotonin dopamine
- Causes constriction of blood vessels
- Wide range of effect

TRIPTANS

- Serotonin is involved in migraine attack
- Triptans mimic the action of serotonin
- Triptans act on receptors at smooth muscle cells of brain vessels
- They are an advance over ergots

PREVENTIVE THERAPY

- Beta blockers – e.g.. Propanolol
- Calcium channel blocker – eg.verapamil
- TCA3 – amitriptylene
- SSRI's –fluxetine
- anticonvulsant- - sodium valproate
- Anti histaminic - cyproheptadine

NEED FOR PROPHYLACTIC TREATMENT

- Abortive drugs should not be used for more than 2-3 times a week
- Long term prophylaxis improves quality of life by reducing frequency and severity of attacks

INDICATION OF PROPHYLAXIS

- Patients who have very frequent headaches (more than 2-3/week)
- Attack duration> 48hrs
- Headache severity is extreme
- Migraine attacks are accompanied by severe aura
- Contraindication to acute treatment
- Unacceptable adverse effects occur with acute migraine treatment
- Patients preference

Tension-Type Headache

- The term *tension-type headache* (TTH) is commonly used to describe a chronic head-pain syndrome characterized by bilateral tight, band like discomfort.

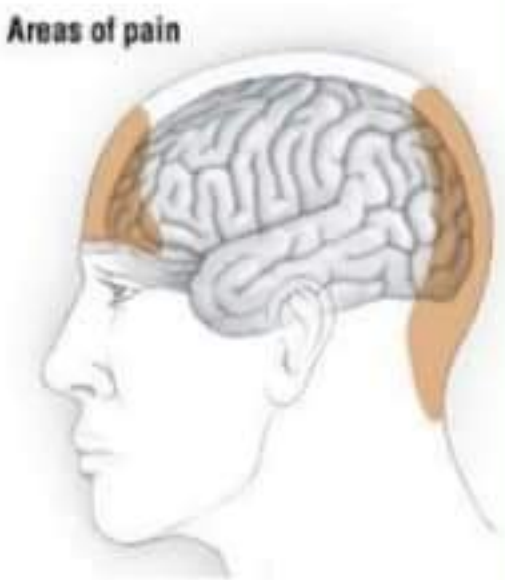
- The pain typically builds slowly, fluctuates in severity, and may persist more or less continuously for many days.

- The headache may be episodic or chronic (present >15 days per month).

Clinical Features

- Completely without accompanying features such as nausea, vomiting, photophobia, phonophobia, osmophobia, throbbing, and aggravation with movement.

- In clinical practice, dichotomizing patients on the basis of the presence of associated features (migraine) and the absence of associated features (TTH) is highly recommended.



Pathophysiology

- Primary disorder of CNS pain modulation alone, unlike migraine, which involves a more generalized disturbance of sensory modulation.

- The name *tension-type headache* implies that pain is a product of *nervous tension*, but there is no clear evidence for tension as an etiology.

- Muscle contraction has been considered to be a feature that distinguishes TTH from migraine, but there appear to be no differences in contraction between the two headache types.

Treatment: Tension-Type Headache

- The pain of TTH can generally be managed with simple analgesics such as acetaminophen, aspirin, or NSAIDs.
- Behavioral approaches including relaxation can also be effective.
- TRIPTANS in pure TTH are NOT HELPFUL, although triptans are effective in TTH when the patient also has migraine.
- For chronic TTH , AMITRIPTYLINE is the only proven treatment Other TCA, SSRI and the benzodiazepines have not been shown to be effective.

Cluster Headache Rare INCIDENCE only 0.1%.

THE PAIN IS

- often excruciating in intensity,
- nonfluctuating, explosive in quality.
- periodic
- onset nocturnal
- recurs at about the same hour

The typical cluster headache patient has daily bouts of 1-2 attacks of short-duration unilateral pain for 8 to 10 weeks a year; usually followed by a pain-free interval little less than 1 year.

- Patients tend to move about during attacks, WHILE PATIENTS WITH MIGRAINE REMAIN SILENT

Cluster Headache Symptoms

A cluster headache is one of the most painful types of headache. A striking feature of cluster headache is that the attacks occur in cyclical patterns or clusters. The pain of a cluster headache is often described as sharp, penetrating or burning.

Excruciating pain in or around one eye

Drooping eyelid

Excessive tearing

Reduced pupil size

Stuffy or runny nostril on the affected side

Restlessness

Sensitivity to light and sound

Aura

Paleness of the face

Exhaustion afterwards

Nausea

Cluster Headache

Gender Pain	M > F 3:1
Type	Stabbing, boring
Severity	Excruciating
Site	Orbit, temple
Attack frequency	1/alternate day–8/d
Duration of attack	15–180 min
Autonomic features	**Yes**
Migrainous features[b]	**Yes** ; Nausea, photophobia, or phonophobia; photophobia and phonophobia are typically unilateral on the side of the pain
Alcohol trigger	**Yes**
Cutaneous triggers	No

Treatment: Cluster Headache

- The most satisfactory treatment is the administration of drugs to prevent cluster attacks until the bout is over
- **ACUTE ATTACK TREATMENT**
  - Many patients respond very well to oxygen inhalation. This should be given as 100% oxygen at 10–12 L/min for 15–20 min.
  - Sumatriptan 6 mg SC is rapid in onset and will usually shorten an attack to 10–15 min; there is no evidence of tachyphylaxis.
  - Sumatriptan (20 mg) and zolmitriptan (5 mg) nasal sprays are both effective
- **ORAL SUMATRIPTAN IS NOT EFFECTIVE**

Preventive Management of Cluster Headache

Short-Term Prevention	Long-Term Prevention
Episodic Cluster Headache	**Episodic Cluster Headache & Prolonged Chronic Cluster Headache**
Prednisone 1 mg/kg up to 60 mg qd, tapering over 21 days	Verapamil 160–960 mg/d
	Lithium 400–800 mg/d
Methysergide 3–12 mg/d	Methysergide 3–12 mg/d
Verapamil 160–960 mg/d	Topiramate[a] 100–400 mg/d
Greater occipital nerve injection	Gabapentin[a] 1200–3600 mg/d
	Melatonin[a] 9–12 mg/d

Fb/Nurse-Info

THANK YOU