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CLINICAL PROFILE OF HEADACHE IN PATIENTS ATTENDING TERTIARY CARE CENTER

A DISSERTATION SUBMITTED

FOR THE DEGREE OF M.D. (BRANCH-I)

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In partial fulfillment of the requirements

For The Degree Of

M.D. (BRANCH-I) GENERAL MEDICINE

BY

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Under The Guidance of

DR. N. R. RATHOD,

Certificate

This is to certify that

DR. MANSI P. SHAH

Has carried out the work to prepare

This dissertation on

CLINICAL PROFILE OF HEADACHE IN PATIENTS

ATTENDING TERTIARY CARE CENTER

(A study of 100 cases)

Under my guidance, supervision and

To utmost satisfaction

For the degree of M.D.

(Bra nch -I, General Medicine)

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CONTENTS

INTRODUCTION

HEADACHE is a very common complaint of the patients visiting doctors. Headache occurs in all age groups. On a global basis headache is responsible for more disability than any other neurological problem.

The prevalence of chronic frequent headache in general population is around 4% worldwide[4]. Around 76% of the women and 57% of the men report at least one significant headache per month, and more than 90% of population experience at least one noteworthy headache episode in their lifetime. The relatively high prevalence of headache together with a low quality of life indicates that chronic headache is a serious health problem. Almost all patients start with episodic headaches which are of tension type or migraine type headaches, which gradually become more frequent until their headaches are almost daily. Psychological factors may also play an important role in chronification of headaches.

The International Headache Society classifies Headache as PRIMARY or SECONDARY. Primary headaches are those in which headache and its associated features are the disorder in itself. Secondary headaches are those caused by exogenous disorders. Common types of primary headache include Tension type headache, Migraine headache, Cluster headache, Idiopathic stabbing headache, Exertional headache, others. Common causes of secondary headache include systemic infections, head injury, vascular disorders, subarachnoid hemorrhage, brain tumor, others.

This study is conducted to evaluate the clinical profile of headache in the patients attending tertiary care center.

AIMS AND OBJECTIVES

- To assess the clinical profile of headache in patients presenting at the tertiary care center.
- To reveal the identifiable cause of headache (primary or secondary headache).
- To identify various risk factors that may affect prognosis and guide treatment.
- Management.
- Complications and outcome.

REVIEW OF LITERATURE

Headache is among the most common reasons patients seek medical attention, on a global basis being responsible for more disability than any other neurologic problem. Diagnosis and management are based on a careful clinical approach augmented by an understanding of the anatomy, physiology and pharmacology of the nervous system pathways mediating the various headache syndromes.

A classification system[1] developed by the International Headache Society[5] characterizes headache as primary or secondary.

Primary headaches[1] are those in which headache and its associated features are the disorder in itself, where as secondary headaches are those caused by exogenous disorders. Primary headache often results in considerable disability and a decrease in the patient's quality of life. Mild secondary headache, such as that seen in association with upper respiratory tract infections, is common but rarely worrisome. Lifethreatening headache is relatively uncommon, but vigilance is required in order to recognize and appropriately treat such patients.

ANATOMY AND PHYSIOLOGY OF HEADACHE[1]

Pain usually occurs when peripheral nociceptors are stimulated in response to tissue injury, visceral distension, or other factors. In such situations, pain perception is a normal physiologic response mediated by a healthy nervous system. Pain can also result when pain producing pathways of the peripheral or central nervous system are damaged or activated inappropriately. Headache may originate from either or both mechanisms.

Relatively few cranial structures are pain-producing; these include 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.

The key structures involved in primary headache appear to be thefollowing[2]:

- The large intracranial vessels and dura matter and the peripheral terminals of the trigeminal nerve that innervate these structures

- The caudal portion of the trigeminal nucleus, which extends into the dorsal horns of the upper cervical spinal cord and receives input from the first and second cervical nerve roots (the trigemino-cervical complex)

- Rostral pain-processing regions, such as the ventro-postero-medialthalamus and the cortex

- The pain-modulatory systems in the brain that modulate input from trigeminal nociceptors at all levels of the pain-processing pathways and influence vegetative functions, such as hypothalamus and brainstem structures.

The innervation of the large intracranial vessels and dura matter by the trigeminal nerve is known as the trigemino-vascular system. Cranial autonomic symptoms, such as lacrimation, conjunctival injection, nasal congestion, rhinorrhea, peri-orbital swelling, aural fullness, and ptosis, are prominent in the trigeminal autonomic cephalalgias, including cluster headache and paroxysmal hemicrania, and may also be seen in migraine, even in children. These autonomic symptoms reflect activation of cranial parasympathetic pathways, and functional imaging studies indicate that vascular changes in migraine and cluster headache, when present, are similarly driven by these cranial autonomic systems. Moreover, they can often be mistaken for symptoms or signs of cranial sinus inflammation, which is thus over-diagnosed and inappropriately managed. Migraine and other primary headache types are not ―vascular headaches‖; these disorders do not reliably manifest vascular changes, and treatment outcomes cannot be predicted by vascular effects. Migraine is a brain disorder and is best understood and managed as such.

Common Causes of Headache[1]

Primary Headache		**Secondary Headache**	
Type	%	Type	%
Tension-type	69	Systemic infection	63
Migraine	16	Head injury	4
Idiopathic stabbing	2	Vascular disorders	1
Exertional	1	Subarachnoid hemorrhage	<1
Cluster	0.1	Brain tumor	0.1

CLINICAL EVALUATION OF ACUTE, NEW-ONSET HEADACHE

The patient who presents with a new, severe headache has a differential diagnosis that is quite different from the patient with recurrent headaches over many years. 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. Serious causes to be considered include meningitis, subarachnoid hemorrhage, epidural or subdural

hematoma, glaucoma, tumor, and purulent sinusitis. When worrisome symptoms and signs are present, rapid diagnosis and management are critical.

A careful neurologic examination is an essential first step in the evaluation. In most cases, patients with an abnormal examination or a history of recent-onset headache should be evaluated by a computed tomography (CT) or magnetic resonance imaging (MRI) study. As an initial screening procedure for intracranial pathology in this setting, CT and MRI methods appear to be equally sensitive. In some circumstances, a lumbar puncture (LP) is also required, unless a benign etiology can be otherwise established.

A general evaluation of acute headache might include cranial arteries by palpation; cervical spine by the effect of passive movement of the head and by imaging; the investigation of cardiovascular and renal status by blood pressure monitoring and urine examination; and eyes by funduscopy, intraocular pressure measurement, and refraction. The psychological state of the patient should also be evaluated because a relationship exists between head pain and depression. This is intended to identify comorbidity rather than provide an explanation for the headache, because troublesome headache is seldom simply caused by mood change. Although it is notable that medicines with antidepressant actions are also effective in the prophylactic treatment of both tension-type headache and migraine, each symptom must be treated optimally. Underlying recurrent headache disorders may be activated by pain that follows otologic or endodontic surgical procedures. Thus, pain about the head as the result of diseased tissue or trauma may reawaken an otherwise quiescent migraine syndrome. Treatment of the headache is largely ineffective until the cause of the primary problem is addressed. Serious underlying conditions that are associated with headache are described below. Brain tumor is a rare cause of headache and even less commonly a cause of severe pain. The vast majority of patients presenting with severe headache have a benign cause[3].

HEADACHE SYMPTOMS THAT SUGGEST A SERIOUS UNDERLYING DISORDER[1]

- Sudden-onset headache
- First severe headache
- —Worst‖ headache ever[2]
- Vomiting that precedes headache
- Subacute worsening over days or weeks
- Pain induced by bending, lifting, cough
- Pain that disturbs sleep or presents immediately upon awakening
- Known systemic illness
- Onset after age [55]
- Fever or unexplained systemic signs
- Abnormal neurologic examination
- Pain associated with local tenderness, e.g., region of temporal artery

CHRONIC DAILY HEADACHE

The broad diagnosis of chronic daily headache (CDH) can be applied when a patient experiences headache on 15 days or more per month[1]. CDH is not a single entity; it encompasses a number of different headache syndromes, both primary and secondary[9]. In aggregate, this group presents considerable disability. Population-based estimates suggest that about 4% of adults have daily or near-daily headache[3].

Classification of Chronic Daily Headache[1]

Primary		**Secondary**
>4 h Daily	<4 h Daily	Posttraumatic • Head injury • Iatrogenic • Post-infectious

Chronic migraine	Chronic cluster headache	Inflammatory • Giant cell arteritis • Sarcoidosis • Behcet's syndrome
Chronic tension type headache	Chronic paroxysmal hemicrania	Chronic CNS infection
Hemicrania continua	SUNCT/SUNA	Medication overuse headache
New daily persistent headache	Hypnic headache	

PRIMARY HEADACHE DISORDERS[1]

Primary headaches are disorders in which headache and associated features occur in the absence of any exogenous cause. The most common are migraine, tension-type headache, and the trigeminal autonomic cephalalgias, notably cluster headache.

Primary Headache Disorders classification[5] (Modified from International Classification of Headache Disorders-III-Beta, Committee of the

International Headache Society, 2013)

1. Migraine headache

- Migraine without aura Migraine with aura
  - ➢ Migraine with typical aura
    - Typical aura with headache
    - Typical aura without headache
  - ➢ Migraine with brainstem aura
  - ➢ Hemiplegic migraine

- Familial hemiplegic migraine (FHM)
  - Familial hemiplegic migraine type 1
  - Familial hemiplegic migraine type 2
  - Familial hemiplegic migraine type 3
- Sporadic hemiplegic migraine
- Retinal migraine

- Chronic migraine
- Complications of migraine
  - Status migrainosus
  - Persistent aura without infarction
  - Migrainous infarction
  - Migraine aura-triggered seizure
- Probable migraine
  - Probable migraine without aura
  - Probable migraine with aura
- Episodic syndromes that may be associated with migraine ➢Recurrent gastrointestinal disturbance
  - Cyclical vomiting syndrome
  - Abdominal migraine ➢Benign paroxysmal vertigo
  - Benign paroxysmal torticollis

2. Tension-type headache

- Infrequent episodic tension-type headache
- Frequent episodic tension-type headache
- Chronic tension-type headache

3. Trigeminal autonomic cephalalgias

- Cluster headache

- Episodic cluster headache
- Chronic cluster headache

Paroxysmal hemicrania

- Episodic paroxysmal hemicrania
- Chronic paroxysmal hemicrania

Short-lasting unilateral neuralgiform headache attacks

- Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)
- Short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)

Hemicrania continua

4. Other primary Headache disorders

- Primary cough headache
- Primary exercise headache
- Primary headache associated with sexual activity
- Primary thunderclap headache Cold-stimulus headache
  - Headache attributed to external application of a cold stimulus
  - Headache attributed to ingestion or inhalation of a cold stimulus
- External-pressure headache
  - External-compression headache
  - External-traction headache
- Primary stabbing headache
- Nummular headache
- Hypnic headache

- New daily persistent headache (NDPH)

TENSION TYPE HEADACHE[1]

Clinical Features:

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[3] (present >15 days per month).

A useful clinical approach is to diagnose TTH in patients whose headaches are completely without accompanying features such as nausea, vomiting, photophobia, phonophobia, osmophobia, throbbing, and aggravation with movement. Such an approach neatly separates migraine, which has one or more of these features and is the main differential diagnosis, from TTH.

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. Indeed patients whose headaches fit the TTH phenotype and who have migraine at other times, along with a family history of migraine, migrainous illnesses of childhood, or typical migraine triggers to their migraine attacks, may be biologically different from those who have TTH headache with none of the features.

TTH may be infrequent (episodic) or occur on 15 days or more a 3 .
month (chronic)

Pathophysiology:

The pathophysiology of TTH is incompletely understood. It seems likely that TTH is due to a primary disorder of central nervous system pain modulation alone, unlike migraine, which involves a more generalized disturbance of sensory modulation. Data suggest a genetic contribution to TTH, but this may not be a valid finding.

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[2].

Treatment[1]:

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. Clinical studies have demonstrated that 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 tricyclics, selective serotonin reuptake inhibitors, and the benzodiazepines have not been shown to be effective. There is no evidence for the efficacy of acupuncture. Placebo-controlled trials of onabotulinum toxin type A in chronic TTH were negative.

MIGRAINE HEADACHE

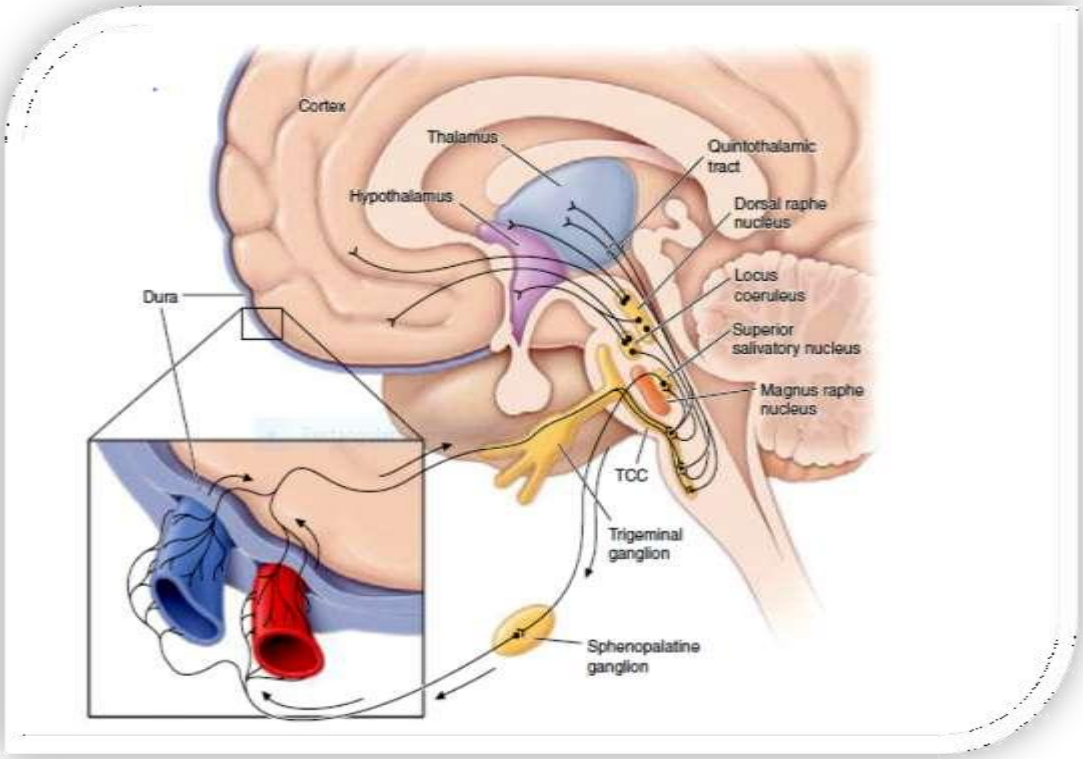
Migraine, the second most common cause of headache, and the most common headache-related and indeed neurologic, cause of disability in the world[8], afflicts approximately 15% of women and 6% of men[10,11] over a 1-year period[1]. It is usually an episodic headache associated with certain features such as sensitivity to light, sound, or movement; nausea and vomiting often accompany the headache.

Migraine is a recurring syndrome of headache associated with other symptoms of neurologic dysfunction in varying admixtures. Migraine can often be recognized by its activators, referred to as triggers. The brain of the migraineur[12] is particularly sensitive to environmental and sensory stimuli; migraine-prone patients do not habituate easily to sensory stimuli. This sensitivity is amplified in females during the menstrual cycle. Headache can be initiated or amplified by various triggers[13,14,15], including glare, bright lights, sounds, or other afferent stimulation;hunger[16]; let-down from stress; physical exertion; stormy weather or barometric pressure changes; hormonal fluctuations during menses; lack of or excess sleep; and alcohol or other chemical stimulation, such as with nitrates. Knowledge of a patient's susceptibility to specific triggers can be useful in management strategies involving lifestyle adjustments.

Pathogenesis:

The sensory sensitivity that is characteristic of migraine is probably due to dysfunction of monoaminergic sensory control systems located in the brainstem and hypothalamus. Activation of cells in the trigeminal nucleus results in the release of vasoactive neuropeptides, particularly calcitonin gene–related peptide (CGRP) [3], at vascular terminations of the trigeminal nerve and within the trigeminal nucleus. CGRP receptor antagonists, gepants, have now been shown to be effective in the acute treatment of migraine, and monoclonal antibodies to CGRP have been shown effective in two early phase clinical trials. Centrally, the secondorder trigeminal neurons cross the midline and project to ventrobasal and posterior nuclei of the thalamus for further processing. Additionally, there are projections to the periaqueductal gray and hypothalamus, from which reciprocal descending systems have established anti-nociceptive effects. Other brainstem regions likely to be involved in descending modulation of trigeminal pain include the nucleus locus coeruleus in the pons and the rostroventromedial medulla.

Pharmacologic and other data point to the involvement of the neurotransmitter5-hydroxytryptamine (5-HT; also known as serotonin) in migraines. Approximately 60 years ago,



methysergide was found to antagonize certain peripheral actions of 5-HT and was introduced as the first drug capable of preventing migraine attacks. The triptans were designed to stimulate selectively subpopulations of 5-HT receptors; at least 14 different 5-HT receptors exist in humans. The triptans are potent agonists of 5-HT1B and 5-HT1D receptors, and some are active at the 5-

HT1F receptors; the latter's exclusive agonists are called ditans. Triptans arrest nerve signaling in the nociceptive pathways of the trigeminovascular system, at least in the trigeminal nucleus caudalis and trigeminal sensory thalamus, in addition to cranial vasoconstriction, while ditans, now shown conclusively to be effective in acute migraine, act only at neural targets. An interesting range of neural targets is now being actively pursed for the acute and preventive management of migraine. 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, as demonstrated by the induction of yawning, nausea, vomiting, hypotension and other symptoms of a migraine attack by dopaminergic agonists at doses that do not affect non-migraineurs. Dopamine receptor antagonists are effective therapeutic agents in migraine, especially when given parenterally or concurrently with other anti-migraine agents. Moreover, hypothalamic activation, anterior to that seen in cluster headache, has now been shown in the premonitory phase of migraine using functional imaging.

Figure 1: Brainstem pathways that modulate sensory input.

The key pathway for pain in migraine is the trigemino-vascular input from the meningeal vessels, which passes through the trigeminal ganglion and synapses on second-order neurons in the trigeminocervical complex (TCC). These neurons in turn project in the quintothalamic tract and, after decussating in the

brainstem, synapse on neurons in the thalamus. Important modulation of the trigemino-vascular nociceptive input comes from the dorsal raphe nucleus, locus coeruleus, and nucleus raphemagnus, understanding some part of the role of dopamine in the disorder.

Migraine genes identified by studying families with familial hemiplegic migraine (FHM) reveal involvement of ion channels, suggesting that alterations in membrane excitability can predispose to migraine. Mutations involving the Cav2.1 (P/Q)–type voltage-gated calcium channel CACNA1A gene are now known to cause FHM 1;this mutation is responsible for about 50% of FHMs. Mutations in the Na+K+ATPase ATP1A2 gene, designated FHM 2, are responsible for about 20% of FHMs. Mutations in the neuronal voltage-gated sodium channel SCN1A cause FHM 3. Functional neuroimaging has suggested that brainstem regions in migraine and the posterior hypothalamic gray matter region close to the human circadian pacemaker cells of the suprachiasmatic nucleus in cluster headache are good candidates for specific involvement in primary headache.

Diagnosis and clinical features:

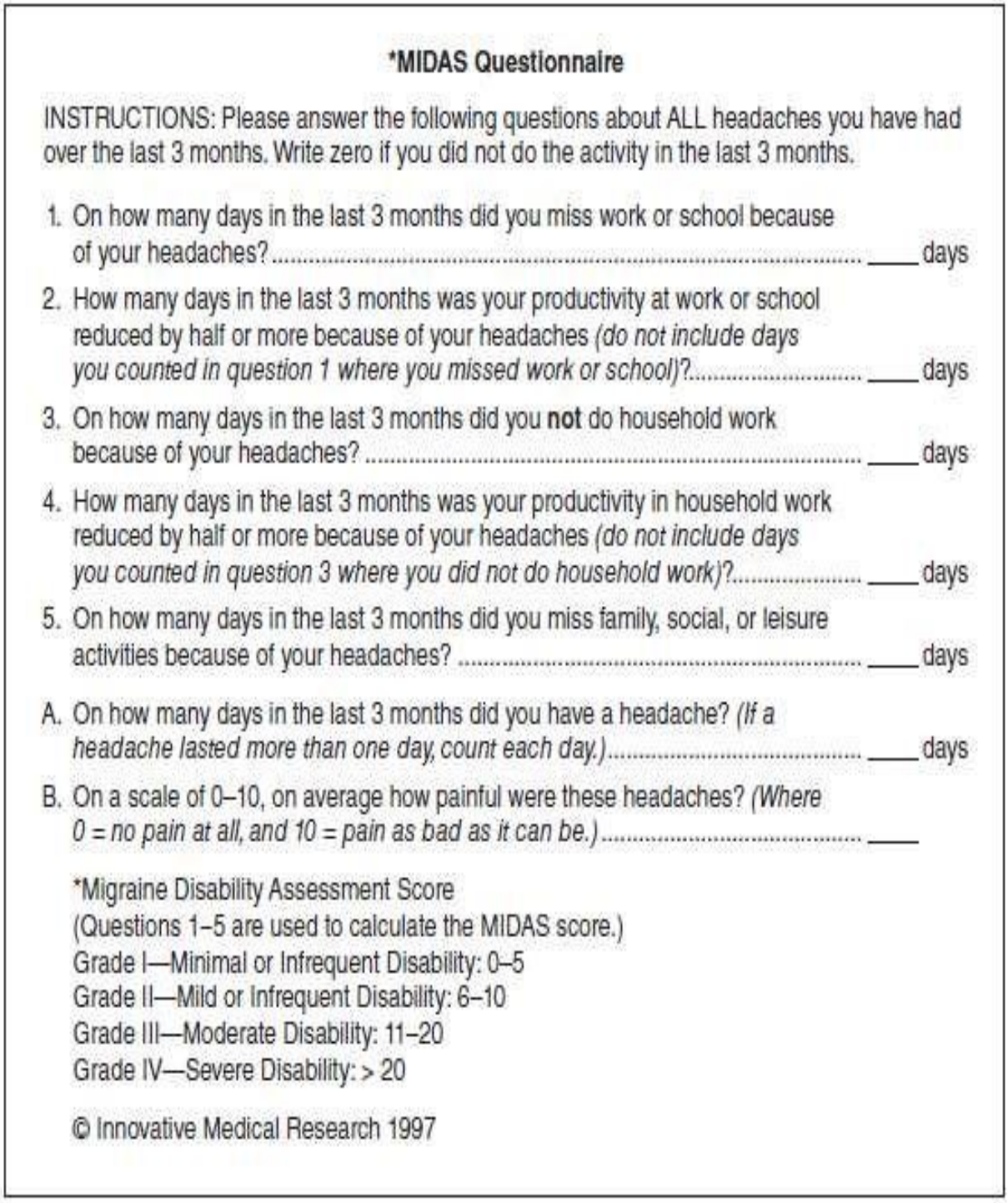
A high index of suspicion is required to diagnose migraine. The migraine aura, consisting of visual disturbances with flashing lights or zigzag lines moving across the visual field or of other neurologic symptoms[24], is reported in only 20–25% of patients. A headache diary can often be helpful in making the diagnosis; this is also helpful in assessing disability and the frequency of treatment for acute attacks. Patients with episodes of migraine that occur daily or near-daily are considered to have chronic migraine. Migraine must be differentiated from tension-type headache[22], the most common primary headache syndrome seen in the population[28].

Migraine has several forms that have been defined: migraine with and without aura and chronic migraine, the latter occurring 15 days or more a month, as the most important. Migraine at its most basic level is headache with associated features, and tension-type headache is headache that is featureless. Most patients with disabling headache probably have migraine. Patients with acephalgic migraine (typical aura without headache, experience recurrent neurologic symptoms, often with nausea or vomiting, but with little or no headache. Vertigo can be prominent; it has been estimated that one-third of patients referred for vertigo or dizziness have a primary diagnosis of migraine. Migraine aura can have prominent brainstem symptoms, and the terms basilar artery and basilartype migraine have now been replaced by migraine with brainstem aura.

Treatment:

Once a diagnosis of migraine has been established, it is important to assess the extent of a patient's disease and disability. The Migraine Disability Assessment Score (MIDAS) is a well-validated[25], easy-to-use tool1, 2.

Patient education is an important aspect of migraine management. Information for patients is available at sites such as www.achenet.org, the website of the American Council for Headache Education



***MIDAS Questionnaire**

INSTRUCTIONS: Please answer the following questions about ALL headaches you have had over the last 3 months. Write zero if you did not do the activity in the last 3 months.

1. On how many days in the last 3 months did you miss work or school because of your headaches? ..... ____ days
2. How many days in the last 3 months was your productivity at work or school reduced by half or more because of your headaches *(do not include days you counted in question 1 where you missed work or school)*? ..... ____ days
3. On how many days in the last 3 months did you **not** do household work because of your headaches? ..... ____ days
4. How many days in the last 3 months was your productivity in household work reduced by half or more because of your headaches *(do not include days you counted in question 3 where you did not do household work)*? ..... ____ days
5. On how many days in the last 3 months did you miss family, social, or leisure activities because of your headaches? ..... ____ days

A. On how many days in the last 3 months did you have a headache? *(If a headache lasted more than one day, count each day.)* ..... ____ days

B. On a scale of 0–10, on average how painful were these headaches? *(Where 0 = no pain at all, and 10 = pain as bad as it can be.)* ..... ____

*Migraine Disability Assessment Score
(Questions 1–5 are used to calculate the MIDAS score.)
Grade I—Minimal or Infrequent Disability: 0–5
Grade II—Mild or Infrequent Disability: 6–10
Grade III—Moderate Disability: 11–20
Grade IV—Severe Disability: > 20

(ACHE). It is helpful for patients to understand that migraine is an inherited tendency to headache; that migraine can be modified and controlled by lifestyle adjustments and medications, but it cannot be eradicated; and that, except in some occasions in women on oral estrogens or contraceptives, migraine is not associated with serious or life-threatening illnesses.

Figure 2: The Migraine Disability Assessment Score (MIDAS) Questionnaire

NONPHARMACOLOGIC MANAGEMENT:

Migraine can often be managed to some degree by a variety of nonpharmacologic approaches. Most patients benefit by the identification and avoidance of specific headache triggers. A regulated lifestyle is helpful, including a healthy diet, regular exercise, regular sleep patterns, avoidance of excess caffeine and alcohol, and avoidance of acute changes in stress levels, being particularly wary of the let-down effect.

The measures that benefit a given individual should be used routinely because they provide a simple, cost-effective approach to migraine management. Patients with migraine do not encounter more stress than headache-free individuals; over-responsiveness to changes in stress appears to be the issue. Because the stresses of everyday living cannot be eliminated, lessening one's response to stress by various techniques is helpful for many patients[91, 92]. These may include yoga, transcendental meditation, hypnosis, and conditioning techniques such as biofeedback. For most patients, this approach is, at best, an adjunct to pharmacotherapy.

Non-pharmacologic measures are unlikely to prevent all migraine attacks. If these measures fail to prevent an attack, pharmacologic approaches are then needed to abort an attack.

ACUTE ATTACK THERAPIES FOR MIGRAINE:

The mainstay of pharmacologic therapy is the judicious use of one or more of the many medicines that are effective in migraine. The selection of the optimal regimen[58] for a given patient depends on a number of factors, the most important of which is the severity of the attack.

Mild migraine attacks can usually be managed by oral agents; the average efficacy rate is 50–70%.Severe migraine attacks may require parenteral therapy. Most drugs effective in the treatment of migraine are members of one of three major pharmacologic classes: nonsteroidal antiinflammatory drugs, 5-HT1B/1D receptor agonists, and dopamine receptor antagonists.

In general, an adequate dose of whichever agent is chosen should be used as soon as possible after the onset of an attack. If additional medication is required within 60 min because symptoms return or have not abated, the initial dose should be increased for subsequent attacks or a different class of drug tried as first-line treatment.

Migraine therapy must be individualized; a standard approach for all patients is not possible. A therapeutic regimen may need to be constantly refined until one is identified that provides the patient with rapid, complete and consistent relief with minimal side effects.

Nonsteroidal Anti-Inflammatory Drugs (NSAIDs):

Both the severity and duration of a migraine attack can be reduced significantly by NSAIDs. Indeed, many undiagnosed migraineurs selftreat with nonprescription NSAIDs. A general consensus is that NSAIDs are most effective when taken early in the migraine attack. However, the effectiveness of these agents in migraine is usually less than optimal in moderate or severe migraine attacks. The combination of acetaminophen, aspirin, and caffeine has been approved for use by the U.S. Food and Drug Administration (FDA) for the treatment of mild to moderate migraine. The combination of aspirin and metoclopramide has been shown to be comparable to a single dose of oral sumatriptan. Important side effects of NSAIDs include dyspepsia and gastrointestinal irritation. **5-HT1B/1D RECEPTOR AGONISTS:**

Oral Stimulation of 5-HT1B/1D receptors can stop an acute migraine attack. Ergotamine and dihydroergotamine are nonselective receptor agonists, whereas the triptans are selective 5-HT1B/1D receptor agonists. A variety of triptans, 5-HT1B/1D receptor agonists—

sumatriptan, almotriptan, eletriptan, frovatriptan, naratriptan, rizatriptan, and zolmitriptan—are now available for the treatment of migraine. Each drug in the triptan class has similar pharmacologic properties but varies slightly in terms of clinical efficacy. Rizatriptan andeletriptan are the most efficacious of the triptans currently available in the United States. Sumatriptan and zolmitriptan have similar rates of efficacy as well as time to onset, with an advantage of having multiple formulations, whereas almotriptan has a similar rate of efficacy to sumatriptan and is better tolerated, and frovatriptan and naratriptanare somewhat slower in onset and are better tolerated. Clinical efficacy appears to be related more to the tmax (time to peak plasma level) than to the potency, half-life, or bioavailability. This observation is consistent with a large body of data indicating that faster acting analgesics are more effective than sloweracting agents.

Monotherapy with a selective oral 5-HT1B/1D receptor agonist does not result in rapid, consistent, and complete relief of migraine in all patients. Triptans are generally not effective in migraine with aura unless given after the aura is completed and the headache initiated. Side effects are common, although often mild and transient. Moreover, 5-HT1B/1D receptor agonists are contraindicated in individuals with a history of cardiovascular and cerebrovascular disease. Recurrence of headache, within usual time course of an attack, is another important limitation of triptan use and occurs at least occasionally in most patients. Evidence from randomized controlled trials show that co-administration of a longer-acting NSAID, naproxen 500 mg, with sumatriptan will augment the initial effect of sumatriptan and, importantly, reduce rates of headache recurrence. Ergotamine preparations offer a nonselective means of stimulating5-HT1 receptors. A non-nauseating dose of ergotamine should besought because a dose that provokes nausea is too high and may intensify head pain. Except for a sublingual formulation of ergotamine, oral formulations of ergotamine also contain 100 mg caffeine (theoretically to enhance ergotamine absorption and possibly to add additional analgesic activity). The average oral ergotamine dose for a migraine attack is 2 mg. Because the clinical studies demonstrating efficacy of ergotamine in migraine predated the clinical trial methodologies used with the triptans, it is difficult to assess the

clinical efficacy of ergotamine versus the triptans. In general, ergotamine appears to have a much higher incidence of nausea than triptans but less headache recurrence.

Nasal formulations of dihydroergotamine[61] (Migranal), zolmitriptan (Zomig nasal) or sumatriptan can be useful in patients requiring a non oral route of administration. The nasal sprays result in substantial blood levels within 30–60 min. Although in theory nasal sprays might provide faster and more effective relief of a migraine attack than oral formulations, their reported efficacy is only approximately 50– 60%. Studies with a new inhalational formulation of dihydroergotamine indicate that its absorption problems can be overcome to produce rapid onset of action with good tolerability.

Parenteral Administration of drugs by injection, such as dihydroergotamine and sumatriptan, is approved by the FDA for the rapid relief of a migraine attack. Peak plasma levels of dihydroergotamine are achieved 3 min after IV dosing, 30 min after IM dosing, and 45 min after SC dosing. If an attack has not already peaked, SC or IM administration of 1 mg of dihydroergotamine suffices for about80–90% of patients. Sumatriptan, 4–6 mg SC, is effective in ~50–80% of patients and can now be administered by a needle-free device.

DOPAMINE RECEPTOR ANTAGONISTS:

Oral dopamine receptor antagonists can be considered as adjunctive therapy in migraine. Drug absorption is impaired during migraine because of reduced gastrointestinal motility. Delayed absorption occurs even in the absence of nausea and is related to the severity of the attack and not its duration. Therefore, when oral NSAIDs and/or triptan agents fail, the addition of a dopamine receptor antagonist, such as metoclopramide 10 mg or domperidone 10 mg, should be considered to enhance gastric absorption. In addition, dopamine receptor antagonists decrease nausea/vomiting and restore normal gastric motility.

Parenteral Dopamine receptor antagonists (e.g., chlorpromazine, prochlorperazine, metoclopramide) by injection can also provide significant acute relief of migraine; they can be used in combination with parenteral 5-HT1B/1D receptor agonists. A common IV protocol used for the treatment of severe migraine is the administration over 2 min of a mixture of 5 mg of prochlorperazine and 0.5 mg of dihydroergotamine.

OTHER MEDICATIONS FOR ACUTE MIGRAINE:

Oral: The combination of acetaminophen, dichloralphenazone, and isometheptene, one to two capsules, has been classified by the FDAas

—possibly‖ effective in the treatment of migraine. Because the clinical studies demonstrating the efficacy of this combination analgesic in migraine predated the clinical trial methodologies used with the triptans, it is difficult to compare the efficacy of this sympathomimetic compound to other agents.

Nasal preparation of butorphanol is available for the treatment of acute pain. As with all opioids, the use of nasal butorphanol has little role in migraine treatment.

Parenteral Opioids are modestly effective in the acute treatment of migraine. For example, IV meperidine (50–100 mg) is given frequently in the emergency room. This regimen —works‖ in the sense that the pain of migraine is eliminated. However, this regimen is clearly suboptimal for patients with recurrent headache. Opioids do not treat the underlying headache mechanism; rather, they act to alter the pain sensation, and there is evidence their use may decrease the likelihood of a response to triptans in the future. Moreover, in patients taking oral opioids, such asoxycodone or hydrocodone, habituation or addiction can greatly confuse the treatment of migraine. Opioid craving and/or withdrawal can aggravate and accentuate migraine. Therefore, it is recommended that opioid use in migraine be limited to patients with severe, but infrequent, headaches that are unresponsive to other pharmacologic approaches or who have contraindications to other therapies.

MEDICATION-OVERUSE HEADACHE[63, 64, 66]:

Acute attack medications, particularly opioid or barbituratecontaining compound analgesics, have a propensity to aggravate headache frequency and induce a state of refractory daily or near-daily headache called medication-overuse headache. This condition is likely not a separate headache entity but a reaction of the migraine patient to a particular medicine[70]. Migraine patients who have two or more headache days a week should be cautioned about frequent analgesic use.

PREVENTIVE TREATMENTS FOR MIGRAINE:

Patients with an increasing frequency of migraine attacks or with attacks that are either unresponsive or poorly responsive to abortive treatments are good candidates for preventive agents[62] In general, a preventive medication should be considered in the subset of patients with four or more attacks a month. Significant side effects are associated with the use of many of these agents; furthermore, determination of dose can be difficult because the recommended doses have been derived for conditions other than migraine. The mechanism of action of these drugs is unclear; it seems likely that the brain sensitivity that underlies migraine is modified. Patients are usually started on a low dose of a chosen treatment; the dose is then gradually increased, up to a reasonable maximum, to achieve clinical benefit. Drugs must be taken daily, and there is usually a lag of between 2 to 12 weeks before an effect is seen. The drugs that have been approved by the FDA for the prophylactic treatment of migraine include propranolol, timolol, sodium valproate, topiramate, and methysergide (not available). In addition, a number of other drugs appear to display prophylactic efficacy. This group includes amitriptyline, nortriptyline, flunarizine, phenelzine, gabapentin, and cyproheptadine. Placebo-controlled

trials of onabotulinum toxin type A in episodic migraine were negative, whereas, overall, placebo-controlled trials in chronic migraine were positive. Phenelzine and methysergide are usually reserved for recalcitrant cases because of their serious potential side effects. Phenelzine is a mono-amine oxidase inhibitor (MAOI); therefore, tyramine-containing foods, decongestants, and meperidine are contraindicated. Methysergide may cause retroperitoneal or cardiac valvular fibrosis when it is used for >6 months, and thus monitoring is required for patients using this drug; the risk of fibrosis is about 1:1500 and is likely to reverse after the drug is stopped. The probability of success with any one of the anti-migraine drugs is 50–75%. Many patients are managed adequately with low-dose amitriptyline, propranolol, candesartan, topiramate, or valproate. If these agents fail or lead to unacceptable side effects, second-line agents such as methysergide or phenelzine can be used. Once effective stabilization is achieved, the drug is continued for ~6 months and then slowly tapered to assess the continued need. Many patients are able to discontinue medication and experience fewer and milder attacks for long periods, suggesting that these drugs may alter the natural history of migraine.

TRIGEMINAL AUTONOMIC CEPHALALGIAS, INCLUDING CLUSTER HEADACHE

The trigeminal autonomic cephalalgias (TACs) describe a grouping of primary headaches including cluster headache, paroxysmal hemicrania, SUNCT (short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing)/SUNA (short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms), and hemicrania continua. TACs are characterized by relatively short-lasting attacks of head pain associated with cranial autonomic symptoms, such as lacrimation, conjunctival injection, or nasal congestion.

Pain is usually severe and may occur more than once a day. Because of the associated nasal congestion or rhinorrhea, patients are often misdiagnosed with ―sinus headache‖ and treated with decongestants, which are ineffective. TACs must be differentiated from short-lasting headaches that do not have prominent cranial autonomic syndromes, notably trigeminal neuralgia, primary stabbing headache, and Hypnic headache. The cycling pattern and length, frequency, and timing of attacks are useful in classifying patients. Patients with TACs should undergo pituitary imaging and pituitary function tests because there is an excess of TAC presentations in patients with pituitary tumor–related headache.

CLUSTER HEADACHE:

Cluster headache is a relatively rare form of primary headache with a population frequency of approximately 0.1%. The pain is deep, usually retro-orbital, often excruciating in intensity, non fluctuating and explosive in quality. A core feature of cluster headache is periodicity. At least one of the daily attacks of pain recurs at about the same hour each day for the duration of a cluster bout.

The typical cluster headache patient has daily bouts of one to two attacks of relatively short-duration unilateral pain for 8 to 10 weeks a year; this is usually followed by a pain-free interval that

averages a little less than1 year. Cluster headache is characterized as chronic when there is less than 1 month of sustained remission without treatment. Patients are generally perfectly well between episodes. Onset is nocturnal in about50% of patients and men are affected three times more often than women. Patients with cluster headache tend to move about during attacks, pacing, rocking, or rubbing their head for relief; some may even become aggressive during attacks. This is in sharp contrast to patients with migraine, who prefer to remain motionless during attacks.

Cluster headache is associated with ipsilateral symptoms of cranial parasympathetic autonomic activation: conjunctival injection or lacrimation, rhinorrhea or nasal congestion or cranial sympathetic dysfunction such as ptosis. The sympathetic deficit is peripheral and likely to be due to parasympathetic activation with injury to ascending sympathetic fibers surrounding a dilated carotid artery as it passes into the cranial cavity. When present photophobia and phonophobia are far more likely to be unilateral and on the same side of the pain, rather than bilateral, as is seen in migraine. This phenomenon of unilateral photophobia/phonophobia is characteristic of TACs.

Cluster headache is likely to be a disorder involving central pacemaker neurons in the posterior hypothalamic region.

Treatment:

The most satisfactory treatment is the administration of drugs to prevent cluster attacks until the bout is over. However, treatment of acute attacks is required for all cluster headache patients at some time.

ACUTE ATTACK TREATMENT:

Cluster headache attacks peak rapidly, and thus a treatment with quick onset is required. Many patients with acute cluster headache respond very well to oxygen inhalation. This should be given as100% oxygen at 10–12 L/min for 15–20 min. It appears that high flow and high oxygen content are important. 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 in acute cluster headache, offering a useful option for patients who may not wish to self-inject daily. Oral sumatriptan is not effective for prevention or for acute treatment of cluster headache.

PREVENTIVE TREATMENTS:

The choice of a preventive treatment in cluster headache depends in part on the length of the bout. Patients with long bouts or those with chronic cluster headache require medicines that are safe when taken for long periods. For patients with relatively short bouts, limited courses of oral

glucocorticoids or methysergide can be very useful. A 10-day course of prednisone, beginning at 60 mg daily for 7 days and followed by a rapid taper, may interrupt the pain bout for many patients. Lithium (400– 800mg/d) appears to be particularly useful for the chronic form of the disorder. Many experts favor verapamil as the first-line preventive treatment for patients with chronic cluster headache or prolonged bouts. While verapamil compares favorably with lithium in practice, some patients require verapamil doses far in excess of those administered for cardiac disorders. The initial dose range is 40–80 mg twice daily; effective doses may be as high as 960 mg/d. Side effects such as constipation and leg swelling can be problematic. Of paramount concern, however, is the cardiovascular safety of verapamil, particularly at high doses. Verapamil can cause heart block by slowing conduction in the atrioventricular node, a condition that can be monitored by following the PR interval on a standard electrocardiogram (ECG). Approximately 20% of patients treated with verapamil develop ECG abnormalities, which can be observed with doses as low as 240 mg/d; these abnormalities can worsen over time in patients on stable doses. A baseline ECG is recommended for all patients. The ECG is repeated 10 days after a dose change in patients whose dose is being increased above 240 mg daily. Dose increases are usually made in 80-mg increments. For patients on long-term verapamil, ECG monitoring every 6 months is advised.

NEUROSTIMULATION THERAPY:

When medical therapies fail in chronic cluster headache, neurostimulation strategies can be used. Deep-brain stimulation of the region of the posterior hypothalamic gray matter has proven successful in a substantial proportion of patients, although its risk-benefit ratio makes it inappropriate with so many other options now available. Favorable results have also been reported with the less-invasive approach of occipital nerve stimulation, with sphenopalatine ganglion stimulation and with a noninvasive vagal nerve stimulator.

PAROXYSMAL HEMICRANIA

Paroxysmal hemicrania (PH) is characterized by frequent unilateral, severe, short-lasting episodes of headache. Like cluster headache, the pain tends to be retro-orbital but may be experienced all over the head and is associated with autonomic phenomena such as lacrimation and nasal congestion. Patients with remissions are said to have episodic PH, whereas those with the non-remitting form are said to have chronic PH. The essential features of PH are unilateral, very severe pain; short lasting attacks (2–45 min); very frequent attacks (usually more than five a day); marked autonomic features ipsilateral to the pain; rapid course (<72 h); and excellent response to indomethacin. In contrast to cluster headache, which predominantly affects males, the male-to female ratio[1] in PH is close to 1:1.

Indomethacin (25–75 mg tid), which can completely suppress attacks of PH, is the treatment of choice. Although therapy may be complicated by indomethacin-induced gastrointestinal side effects, currently there are no consistently effective alternatives. Topiramate is helpful in some cases. Piroxicam has been used, although it is not as effective as indomethacin. Verapamil, an effective treatment for

cluster headache, does not appear to be useful for PH. In occasional patients, PH can coexist with trigeminal neuralgia (PH-tic syndrome); similar to cluster-tic syndrome, each component may require separate treatment.

Secondary PH has been reported with lesions in the region of the sella turcica, including arterio-venous malformation, cavernous sinus meningioma, pituitary pathology and epidermoid tumors. Secondary PH is more likely if the patient requires high doses (>200 mg/d) of indomethacin. In patients with apparent bilateral PH, raised cerebrospinal fluid (CSF) pressure should be suspected. It is important to note that indomethacin reduces CSF pressure. When a diagnosis of PH is considered, magnetic resonance imaging (MRI) is indicated to exclude a pituitary lesion.

SUNCT/SUNA

SUNCT (short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing) is a rare primary headache syndrome characterized by severe, unilateral orbital or temporal pain that is stabbing or throbbing in quality.

Diagnosis requires at least 20 attacks, lasting for 5–240 s; ipsilateral conjunctival injection and lacrimation should be present. In some patients, conjunctival injection or lacrimation is missing, and the diagnosis of SUNA (short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms) can be made.

Diagnosis[1, 2]:

The pain of SUNCT/SUNA is unilateral and may be located anywhere in the head. Three basic patterns can be seen: single stabs, which are usually short-lived; groups of stabs; or a longer attack comprising many stabs between which the pain does not completely resolve, thus giving a —saw-tooth‖ phenomenon with attacks lasting many minutes. Each pattern may be seen in the context of an underlying continuous head pain. Characteristics that lead to a suspected diagnosis of SUNCT are the cutaneous (or other) triggers of attacks, a lack of refractory period to triggering between attacks, and the lack of a response to indomethacin. Apart from trigeminal sensory disturbance, the neurologic examination is normal in primary SUNCT.

The diagnosis of SUNCT/SUNA is often confused with trigeminal neuralgia (TN) particularly in first-division TN. Minimal or no cranial autonomic symptoms and a clear refractory period to triggering indicate a diagnosis of TN.

Secondary (symptomatic) SUNCT: SUNCT can be seen with posterior fossa or pituitary lesions. All patients with SUNCT/SUNA should be evaluated with pituitary function tests and a brain MRI with pituitary views.

TREATMENT OF SUNCT /SUNA:

ABORTIVE THERAPY

Therapy of acute attacks is not a useful concept in SUNCT/SUNA because the attacks are of such short duration. However, IV lidocaine, which arrests the symptoms, can be used in hospitalized patients.

PREVENTIVE THERAPY

Long-term prevention to minimize disability and hospitalization is the goal of treatment. The most effective treatment for prevention is lamotrigine, 200–400 mg/d. Topiramate and gabapentin may also be effective. Carbamazepine, 400–500 mg/d, has been reported by patients to offer modest benefit.

Surgical approaches such as micro-vascular decompression or destructive trigeminal procedures are seldom useful and often produce long-term complications. Greater occipital nerve injection has produced limited benefit in some patients. Occipital nerve stimulation is probably helpful in a subgroup of these patients. Complete control with deep-brain stimulation of the posterior hypothalamic region was reported in a single patient. For intractable cases, short-term prevention with IV lidocaine can be effective, as can occipital nerve stimulation.

Hemicrania Continua

The essential features of hemicrania continua are moderate and continuous unilateral pain associated with fluctuations of severe pain; complete resolution of pain with indomethacin; and exacerbations that may be associated with autonomic features, including conjunctival injection, lacrimation and photophobia on the affected side.

The age of onset ranges from 11 to 58 years; women are affected twice as often as men. The cause is unknown.

TREATMENT:

Treatment consists of indomethacin; other NSAIDs appear to be of little or no benefit. The IM injection of 100 mg of indomethacin has been proposed as a diagnostic tool, and administration with a placebo injection in a blinded fashion can be very useful diagnostically. Alternatively, a trial of oral indomethacin, starting with 25 mg tid, then 50 mg tid, and then 75 mg tid, can be given. Up to 2 weeks at the maximal dose may be necessary to assess whether a dose has a useful effect. Topiramate can be helpful in some patients. Occipital nerve stimulation probably has a role in patients with hemicrania continua who are unable to tolerate indomethacin.

OTHER PRIMARY HEADACHES

Primary Cough Headache[1, 2,3]:

Primary cough headache is a generalized headache that begins suddenly, lasts for several minutes, sometimes up to a few hours, and is precipitated by coughing; it is preventable by avoiding coughing or other precipitating events, which can include sneezing, straining, laughing, or stooping. In all patients with this syndrome, serious etiologies must be excluded before a diagnosis of —benign‖ primary cough headache can be established. A Chiari malformation or any lesion causing obstruction of CSF pathways or displacing cerebral structures can be the cause of the head pain. Other conditions that can present with cough or Exertional headache as the initial symptom include cerebral aneurysm, carotid stenosis, and vertebro-basilar disease. Benign cough headache can resemble benign exertional headache (below), but patients with the former condition are typically older.

TREATMENT:

Indomethacin 25–50 mg two to three times daily is the treatment of choice. Some patients with cough headache obtain complete cessation of their attacks with lumbar puncture; this is a simple option when compared to prolonged use of indomethacin, and it is effective in about one-third of patients. The mechanism of this response is unclear.

Primary Exercise Headache:

Primary Exertional headache[2] has features resembling both cough headache and migraine. It may be precipitated by any form of exercise; it often has the pulsatile quality of migraine. The pain, which can last from 5 min to 24 h, is bilateral and throbbing at onset; migrainous features may develop in patients susceptible to migraine. The duration tends to be shorter in adolescents than in older adults. Primary Exertional headache can be prevented by avoiding excessive exertion, particularly in hot weather or at high altitude.

The mechanism of primary Exertional headache is unclear. Acute venous distension likely explains one syndrome—the acute onset of headache with straining and breath holding, as in weightlifter's headache. Because exertion can result in headache in a number of serious underlying conditions, these must be considered in patients with Exertional headache. Pain from angina may be referred to the head, probably by central connections of vagal afferents and may present as Exertional headache (cardiac cephalgia). The link to exercise is the main clinical clue that headache is of cardiac origin. Pheochromocytoma may occasionally cause Exertional headache. Intracranial lesions and stenosis of the carotid arteries are other possible etiologies.

TREATMENT:

Exercise regimens should begin modestly and progress gradually to higher levels of intensity. Indomethacin at daily doses from25 to 150 mg is generally effective in benign Exertional headache. Indomethacin (50 mg), ergotamine (1 mg orally), dihydroergotamine (2 mg by nasal spray), and methysergide (1–2 mg orally given30–45 min before exercise) are useful prophylactic measures.

Primary Headache Associated with Sexual Activity:

Three types of sex headache are reported[1]: a dull bilateral ache in the head and neck that intensifies as sexual excitement increases; a sudden, severe, explosive headache occurring at orgasm; and a postural headache developing after coitus that resembles the headache of low CSF pressure. The last arises from vigorous sexual activity and is a form of low CSF pressure headache. Headaches developing at the time of orgasm are not always benign; 5–12% of cases of subarachnoid hemorrhage are precipitated by sexual intercourse. Sex headache is reported by men more often than women and may occur at any time during the years of sexual activity. It may develop on several occasions in succession and then not trouble the patient again, even without an obvious change in sexual activity. In patients who stop sexual activity when headache is first noticed, the pain may subside within a period of 5 min to 2 h.

In about half of patients, sex headache will subside within 6 months. About half of patients with sex headache have a history of Exertional headaches, but there is no excess of cough headache. Migraine is probably more common in patients with sex headache.

TREATMENT:

Benign sex headaches recur irregularly and infrequently. Management can often be limited to reassurance and advice about ceasing sexual activity if a mild, warning headache develops. Propranolol can be used to prevent headache that recurs regularly or frequently, but the dosage required varies from 40 to 200 mg/d. An alternative is the calcium channel–blocking agent diltiazem, 60 mg tid. Ergotamine (1 mg) or indomethacin (25–50 mg) taken 30–45 min prior to sexual activity can also be helpful.

Primary Thunderclap Headache

Sudden onset of severe headache may occur in the absence of any known provocation[1]. The differential diagnosis includes the sentinel bleed of an intracranial aneurysm, cervicocephalic arterial dissection, and cerebral venous thrombosis. Headaches of explosive onset may also be caused by the ingestion of sympathomimetic drugs or of tyraminecontaining foods in a patient who is taking MAOIs, or they may be a symptom of pheochromocytoma. Whether thunderclap headache can be the presentation of an unruptured cerebral aneurysm is uncertain. When neuroimaging studies and lumbar puncture exclude subarachnoid hemorrhage, patients with thunderclap headache usually do very well over the long term. In one study of patients whose computed tomography (CT) scans and CSF findings were negative, ~15% had recurrent episodes of thunderclap headache and nearly half subsequently developed migraine or TTH.

The first presentation of any sudden-onset severe headache should be diligently investigated with neuroimaging (CT or, when possible, MRI with MR angiography) and CSF examination. Formal cerebral angiography should be reserved for those cases in which no primary diagnosis is forthcoming and for clinical situations that are particularly suggestive of intracranial aneurysm. Reversible segmental cerebral vasoconstriction may be seen in primary thunderclap headache without an intracranial aneurysm. In the presence of posterior leukoencephalopathy, the differential diagnosis includes cerebral angiitis, drug toxicity (cyclosporine, intrathecal methotrexate/cytarabine, pseudoephedrine, or cocaine), post-transfusion effects and postpartum angiopathy. Treatment with nimodipine may be helpful, although by definition, the vasoconstriction of primary thunderclap headache resolves spontaneously.

Cold-Stimulus Headache

This refers to head pain triggered by application or ingestion/inhalation of something cold. It is bought on quickly and typically resolves within 10–30 min of the stimulus being removed. It is best recognized as ―brain-freeze‖ headache or ice-cream headache when due to ingestion. Although cold may be uncomfortable at some level for many people, it is the reliable, severe, and somewhat prolonged nature of these pains that set them apart. The transient receptor potential cation subfamily M member 8 (TRPM8) channel, a known cold temperature sensor, may be a mediator of this syndrome.

External Pressure Headache

External pressure from compression or traction on the head can produce a pain that may have some generalized component, although the pain is largely focused around the site of the pressure. It typically resolves within an hour of the stimulus being removed. Examples of stimuli include helmets, swimming goggles, or very long ponytails.

Treatment is to recognize the problem and remove the stimulus.

Primary Stabbing Headache

The essential features of primary stabbing headache are stabbing pain confined to the head or, rarely, the face, lasting from 1 to many seconds or minutes and occurring as a single stab or a series of stabs; absence of associated cranial autonomic features; absence of cutaneous triggering of attacks; and a pattern of recurrence at irregular intervals

(hours to days). The pains have been variously described as —ice-pick pains‖ or —jabs and jolts.‖ They are more common in patients with other primary headaches, such as migraine, the TACs and hemicrania continua.

TREATMENT

The response of primary stabbing headache to indomethacin (25– 50 mg two to three times daily) is usually excellent. As a general rule, the symptoms wax and wane, and after a period of control on indomethacin, it is appropriate to withdraw treatment and observe the outcome.

Nummular Headache

Nummular headache is felt as a round or elliptical discomfort that is fixed in place, ranges in size from 1–6 cm, and may be continuous or intermittent[1]. Uncommonly it may be multifocal. It may be episodic but is more often continuous during exacerbations. Accompanying the pain there may be a local sensory disturbance, such as allodynia or hypesthesia. Local dermatologic or bony lesions need to be excluded by examination and investigation. This condition can be difficult to treat; tricyclics, such as amitriptyline, or anticonvulsants, such as topiramate or valproate, are most often tried.

Hypnic Headache

This headache syndrome typically begins a few hours after sleep onset. The headaches last from 15 to 30 min and are typically moderately severe and generalized; although they may be unilateral and can be throbbing. Patients may report falling back to sleep only to be awakened by a further attack a few hours later; up to three repetitions of this pattern occur through the night. Daytime naps can also precipitate head pain. Most patients are female and the onset is usually after age 60 years[1]. Headaches are bilateral in most, but may be unilateral. Photophobia, phonophobia, and nausea are usually absent. The major secondary consideration in this headache type is poorly controlled hypertension; 24h blood pressure monitoring is recommended to detect this treatable condition.

TREATMENT:

Patients with Hypnic headache generally respond to a bedtime dose of lithium carbonate (200–600 mg). For those intolerant of lithium, verapamil (160 mg) or methysergide (1–4 mg at bedtime) may be alternative strategies. One to two cups of coffee or caffeine, 60 mg orally, at bedtime may be

effective in approximately one-third of patients. Case reports also suggest that flunarizine, 5 mg nightly, can be effective.

NEW DAILY PERSISTENT HEADACHE

Primary new daily persistent headache (NDPH) occurs in both males and females[1]. It can be of the migrainous type, with features of migraine, or it can be featureless, appearing as new-onset TTH. Migrainous features are common and include unilateral headache and throbbing pain; each feature is present in about one-third of patients. Nausea, photophobia, and/or phonophobia occur in about half of patients. Some patients have a previous history of migraine; however, the proportion of NDPH sufferers with preexisting migraine is no greater than the frequency of migraine in the general population. At 24 months, ~86% of patients are headache-free. Treatment of migrainous-type primary NDPH consists of using the preventive therapies effective in migraine. Featureless NDPH is one of the primary headache forms most refractory to treatment. Standard preventive therapies can be offered but are often ineffective.

SECONDARY HEADACHE

The management of secondary headache focuses on diagnosis and treatment of the underlying condition.

MENINGITIS

Acute, severe headache with stiff neck and fever suggests meningitis. Lumbar Puncture is mandatory. Often there is striking accentuation of pain with eye movement. Meningitis can be easily mistaken for migraine in that the cardinal symptoms of pounding headache, photophobia, nausea and vomiting are frequently present, perhaps reflecting the underlying biology of some of the patient.

INTRACRANIAL HEMORRHAGE

Acute, severe headache with stiff neck but without fever suggests subarachnoid hemorrhage. A ruptured aneurysm, arterio-venous malformation, or intra-parenchymal hemorrhage may also present with headache alone. Rarely, if the hemorrhage is small or below the foramen magnum, the head CT scan can be normal. Therefore, LP may be required to definitively diagnose subarachnoid hemorrhage.

BRAIN TUMOR

Approximately 30% of patients with brain tumors consider headache to be their chief complaint. The head pain is usually nondescript—an intermittent deep, dull aching of moderate intensity, which may worsen with exertion or change in position and may be associated with nausea and vomiting. This pattern of symptoms results from migraine far more often than from brain tumor.

The headache of brain tumor disturbs sleep in about 10% of patients. Vomiting that precedes the appearance of headache by weeks is highly characteristic of posterior fossa brain tumors. A history of amenorrhea or galactorrhea should leads one to question whether a prolactin-secreting pituitary adenoma (or the polycystic ovary syndrome) is the source of headache.

Headache arising de novo in a patient with known malignancy suggests either cerebral metastases or carcinomatous meningitis, or both. Head pain appearing abruptly after bending, lifting, or coughing can be due to a posterior fossa mass, a Chiari malformation, or low cerebrospinal fluid (CSF) volume.

TEMPORAL ARTERITIS

Temporal (giant cell) arteritis is an inflammatory disorder of arteries that frequently involves the extracranial carotid circulation. It is a common disorder of the elderly; its annual incidence is 77 per 100,000 individuals age 50 and older. The average age of onset is 70 years and women account for 65%

of cases. About half of patients with untreated temporal arteritis develop blindness due to involvement of the ophthalmic artery and its branches; indeed, the ischemic optic neuropathy induced by giant cell arteritis is the major cause of rapidly developing bilateral blindness in patients>60 years. Because treatment with glucocorticoids is effective in preventing this complication, prompt recognition of the disorder is important.

Typical presenting symptoms include headache, polymyalgia rheumatica, jaw claudication, fever, and weight loss. Headache is the dominant symptom and often appears in association with malaise and muscle aches. Head pain may be unilateral or bilateral and is located temporally in 50% of patients but may involve any and all aspects of the cranium. Pain usually appears gradually over a few hours before peak intensity is reached; occasionally, it is explosive in onset. The quality of pain is only seldom throbbing; it is almost invariably described as dull and boring, with superimposed episodic stabbing pains similar to the sharp pains that appear in migraine. Most patients can recognize that the origin of their head pain is superficial, external to the skull, rather than originating deep within the cranium (the pain site for migraineurs). Scalp tenderness is present, often to a marked degree; brushing the hair or resting the head on a pillow maybe impossible because of pain. Headache is usually worse at night and often aggravated by exposure to cold. Additional findings may include reddened, tender nodules or red streaking of the skin overlying the temporal arteries, and tenderness of the temporal or, less commonly, the occipital arteries.

The erythrocyte sedimentation rate (ESR) is often, although not always, elevated; a normal ESR does not exclude giant cell arteritis. A temporal artery biopsy followed by immediate treatment with prednisone 80 mg daily for the first 4–6 weeks should be initiated when clinical suspicion is high. The prevalence of migraine among the elderly is substantial, considerably higher than that of giant cell arteritis. Migraineurs often report amelioration of their headaches with prednisone; thus, caution must be used when interpreting the therapeutic response.

GLAUCOMA:

Glaucoma may present with a prostrating headache associated with nausea and vomiting. The headache often starts with severe eye pain. On physical examination; the eye is often red with a fixed, moderately dilated pupil.

MATERIALS AND METHODS

SOURCE OF DATA:

The data will be collected from patients attending outpatient and inpatient departments of a tertiary care center.

METHODS OF COLLECTION OF DATA:

STUDY DESIGN: This study is a Descriptive Cross-sectional study over a period of one year. A minimum of 100 patients having complaint of headache, admitted in the hospital or attending outpatient department will be recruited in the study. A detailed history will be taken, blood pressure measurement and vitals recording will be done. A detailed physical examination including that of central nervous system, ophthalmic examination, ent examination will be done.

INCLUSION CRITERIA:

1. Adult patients of age 18 years and above.
2. Both male and female adult patients.

EXCLUSION CRITERIA:

1. Patients younger than 18 years of age.

DATA ANALYSIS:

Data collected will be analyzed by frequency, percentage, mean, standard deviation, chi-square test.

Data entry will be done in MS-EXCEL Spreadsheet.

CLINICAL PROFILE OF HEADACHE IN PATIENTS

ATTENDING TERTIARY CARE CENTER

PROFORMA

NAME:

AGE/SEX:

RELIGION:

OCCUPATION:

EDUCATION:

ADDRESS:

INCOME:

MARITAL STATUS:

MRD NO:

CLINICAL PRESENTATION:

C/O HEADACHE

SINCE HOURS/DAYS

LOCATION: UNILATERAL/ BILATERAL

FRONTAL/ OCCIPITAL/ PARIETAL/ TEMPORAL/

HOLOCRANIAL/ HEMICRANIAL

CONTINUOUS/ INTERMITTENT

TIMING (DAY / NIGHT) FREQUENCY:

DURATION:

MILD/ MODERATE/ SEVERE

PAIN QUALITY: STEADY/ THROBBING/ STABBING RADIATION:
PRECIPITATING FACTORS: (ALCOHOL/ SLEEP DEPRIVATION/

OVERSLEEPING/ FOODS/ BRIGHT LIGHT/ MENSTRUAL

VARIATIONS/ OTHERS)

RELIEVING FACTORS: (REST/ QUIET SURROUNDINGS/ DARK

ROOM/ ACTIVITY/ ANALGESIC MEDICATIONS/ OTHERS)

C/O NAUSEA, VOMITING, ANOREXIA

C/O TINGLING, NUMBNESS, PARASTHESIAS

C/O WEAKNESS OF UL/LL/FACE

C/O SPEECH DISTURBANCES

C/O INVOLUNTARY MOVEMENTS

C/O BLURRING OF VISION/ PHOTOPHOBIA/ OTHER VISUAL SYMPTOMS.

C/O NASAL DISCHARGE/ OTHER

H/O TRAUMA

PAST HISTORY:

HTN: YEARS DM: YEARS

IHD: YEARS CVA: YEARS

P/H/O TB/JAUNDICE H/O PSYCHIATRIC ILLNESS P/H/O MOTION SICKNESS OTHERS:

DRUG HISTORY:

ON ANY DRUG: YES/NO

OC PILLS

OTHERS

TREATMENT HISTORY:

FAMILY HISTORY: HTN/ DM/ IHD/ CVA/ OTHERS

PERSONAL HISTORY:

DIET: VEG/ NON VEG/ MIXED

APETITE: ADEQUATE/ DECREASED/ INCREASED

SLEEP: ADEQUATE/ INCREASED/ DECREASED BLADDER HABBITS:

BOWEL HABBITS:

ADDICTION: SMOKING ALCOHOL TOBACCO OTHERS

MARRIED/ UNMARRIED

MENSTRUAL AND OBSTETRIC HISTORY:

GENERAL EXAMINATION VITALS ON ADMISSION:

TEMP: PULSE: BP:

CONSCIOUS: YES/ NO

ORIENTED TO TIME, PLACE, PERSON: YES/ NO BUILT AND NOURISHMENT:

PALLOR: PRESENT/ ABSENT

ICTERUS/ CYANOSIS: PRESENT/ ABSENT

EDEMA: PRESENT/ ABSENT

CLUBBING/KOILONYCHIA/LYMPHADENOPATHY: YES/NO

OTHER FINDINGS

CNS EXAMINATION

HIGHER FUNCTIONS CONSCIOUSNESS:

ORIENTATION TO TIME/PLACE/PERSON:
SPEECH:

BEHAVIOUR/ INTELLIGENCE:

MEMORY (PAST / PRESENT):

HALLUCINATIONS/ DELUSIONS: PRESENT/ ABSENT

CRANIAL NERVES

ANY CRANIAL NERVE INVOLVED: YES/NO IF YES THEN WHICH?

MOTOR SYSTEM

NUTRITION

TONE

	RIGHT	LEFT
UL		
LL		

POWER

	RIGHT	LEFT
UL		
LL		

COORDINATION

INVOLUNTARY MOVEMENTS: PRESENT/ABSENT

SENSORY SYSTEM

SUPERFICIAL SENSATIONS

DEEP SENSATIONS

CORTICAL SENSATIONS

BOWEL/BLADDER INVOLVEMENT: YES/NO

REFLEXES

	BICEPS	TRICEPS	SUPINATOR	KNEE	ANKLE	PLANTAR
RT						
LT						

SIGNS OF MENINGEAL IRRITATION: PRESENT/ABSENT

CEREBELLAR SIGNS: PRESENT/ABSENT

GAIT: NORMAL/ ABNORMAL

OPHTHAL EXAMINATION

PUPILS:

VISUAL ACQUITY: NORMAL/ABNORMAL D FUNDUS EXAMINATION:

C FUNDUS EXAMINATION:

OTHER FINDINGS

ENT EXAMINATION

ANY ABNORMAL FINDINGS:

RESPIRATORY SYSTEM:

CVS EXAMINATION:

PER ABDOMEN:

INVESTIGATIONS

HB(gm/dl)	
TC(/cumm)	
DC	
PLATELET COUNT(/cumm)	
PSMP	
ESR(mm/hr)	
RBS(mg/dl)	
S.CREATININE(mg/dl)	
B.UREA(mg/dl)	
URINE ALBUMIN	
SGPT/LFTS	
S.NA/S.K/OTHER ELECTROLYTES	
OTHERS	

ECG:

IMAGING

CT / MRI BRAIN: FINDINGS:

OTHERS:

TREATMENT:

COMPLICATIONS IF ANY:

OUTCOME:

SPECIFIC REMARKS:

OBSERVATIONS

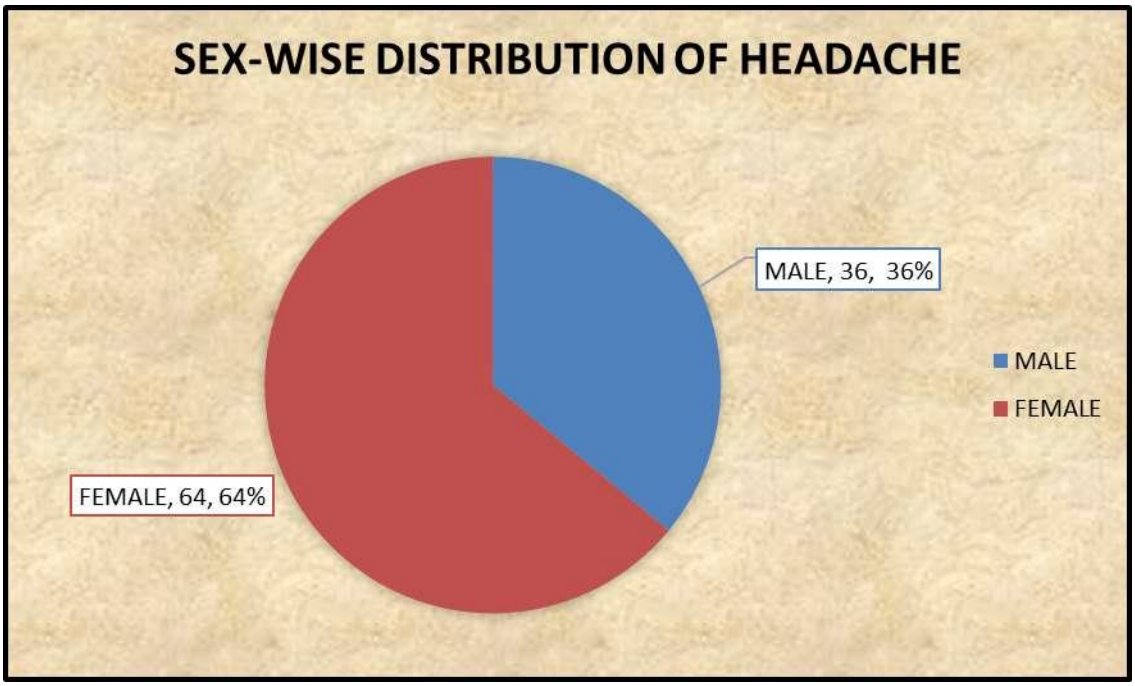
OBSERVATIONS

In the present study of clinical profile of headache, 100 patients who presented with the complaint of headache at the outpatient department or were admitted in the inpatient department of our tertiary care center were selected and studied over a period of one year.

The various observations made are described as follows.

1. DISTRIBUTION OF HEADACHE IN PATIENTS ACCORDING TO SEX

Sex	Number of patients(n=100)	Percentage
MALE	36	36%
FEMALE	64	64%
TOTAL	100	
MALE:FEMALE RATIO	1:1.78	

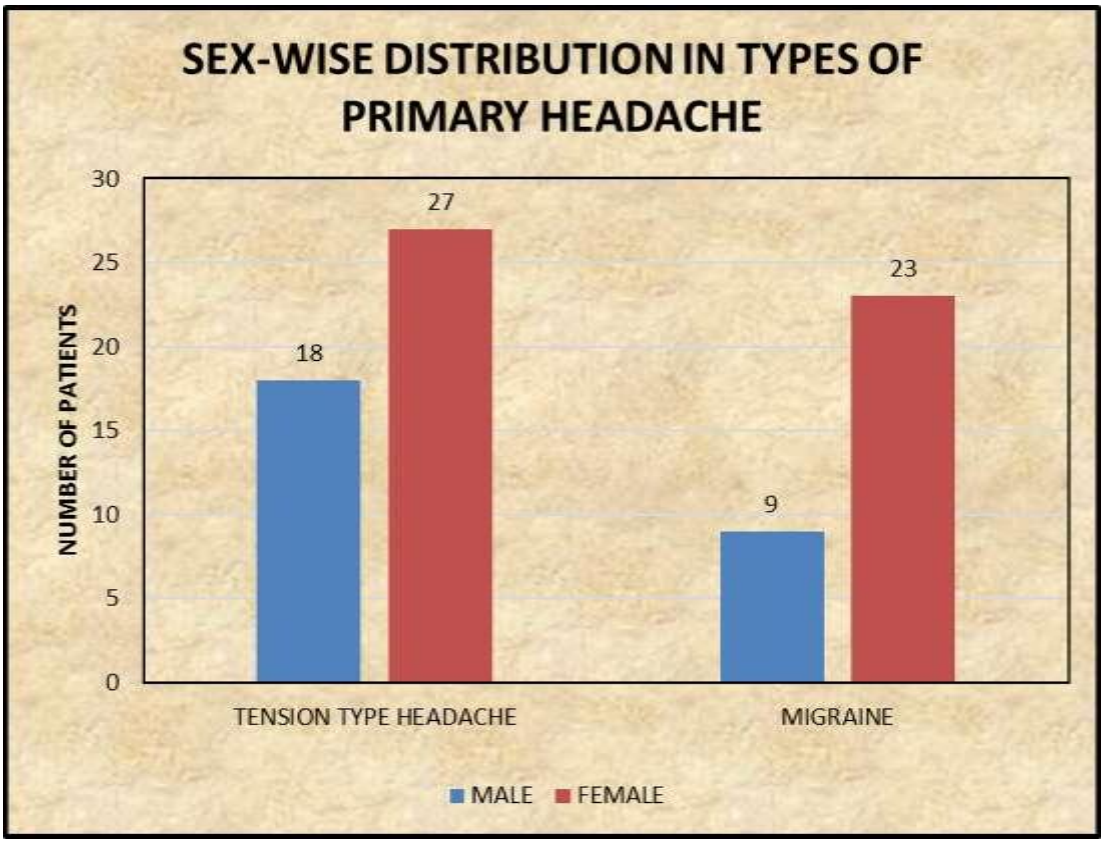


In the present study, amongst the 100 patients, 36 patients were male and 64 patients were female.

Male to female ratio in current study was 1:1.78.

2. SEX-WISE DISTRIBUTION IN TYPES OF PRIMARY HEADACHE

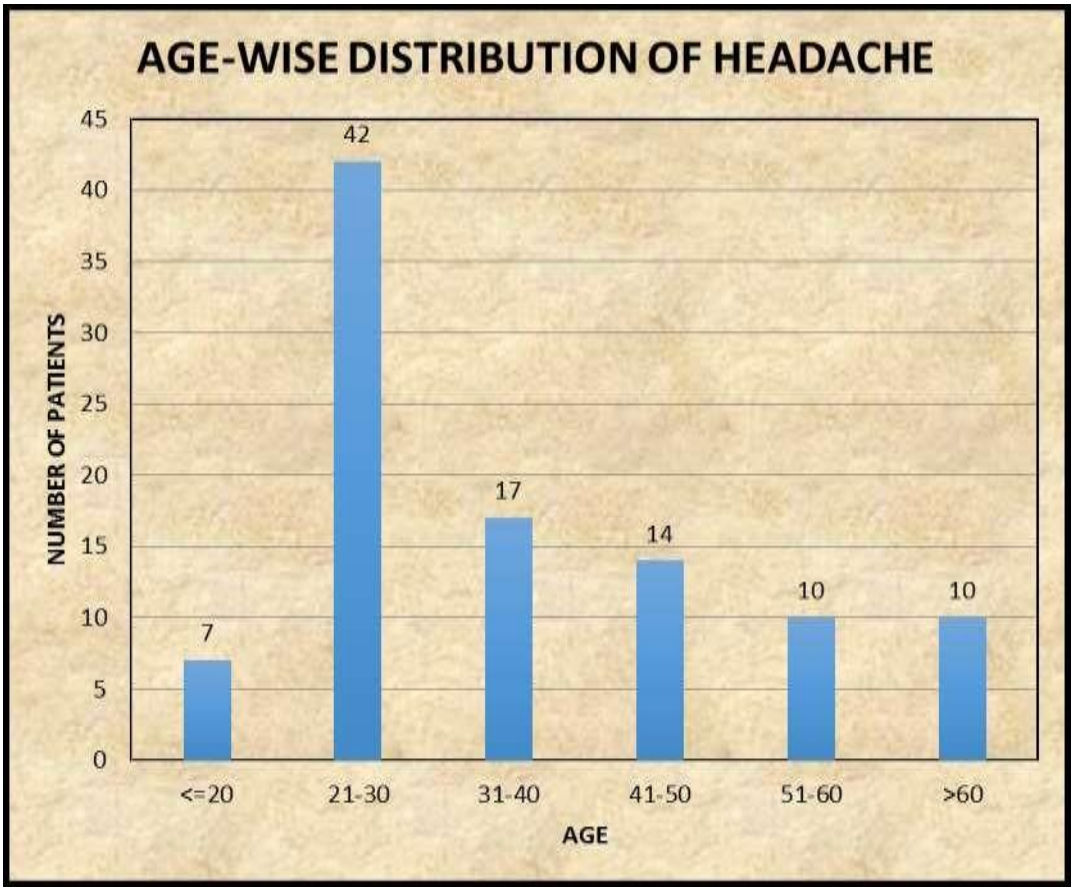
	Tension type headache	Migraine
MALE	18	9
FEMALE	27	23
TOTAL	45	32
MALE TO FEMALE RATIO	1:1.5	1:2.55



In the present study among 45 patients having tension type headache, 18 were male and 27 were female, with male to female ratio of 1:1.5. Among 32 patients having migraine headache, 9 were male and 23 were female with male to female ratio of 1:2.55.

3. DISTRIBUTION OF HEADACHE AMONG VARIOUS AGEGROUPS

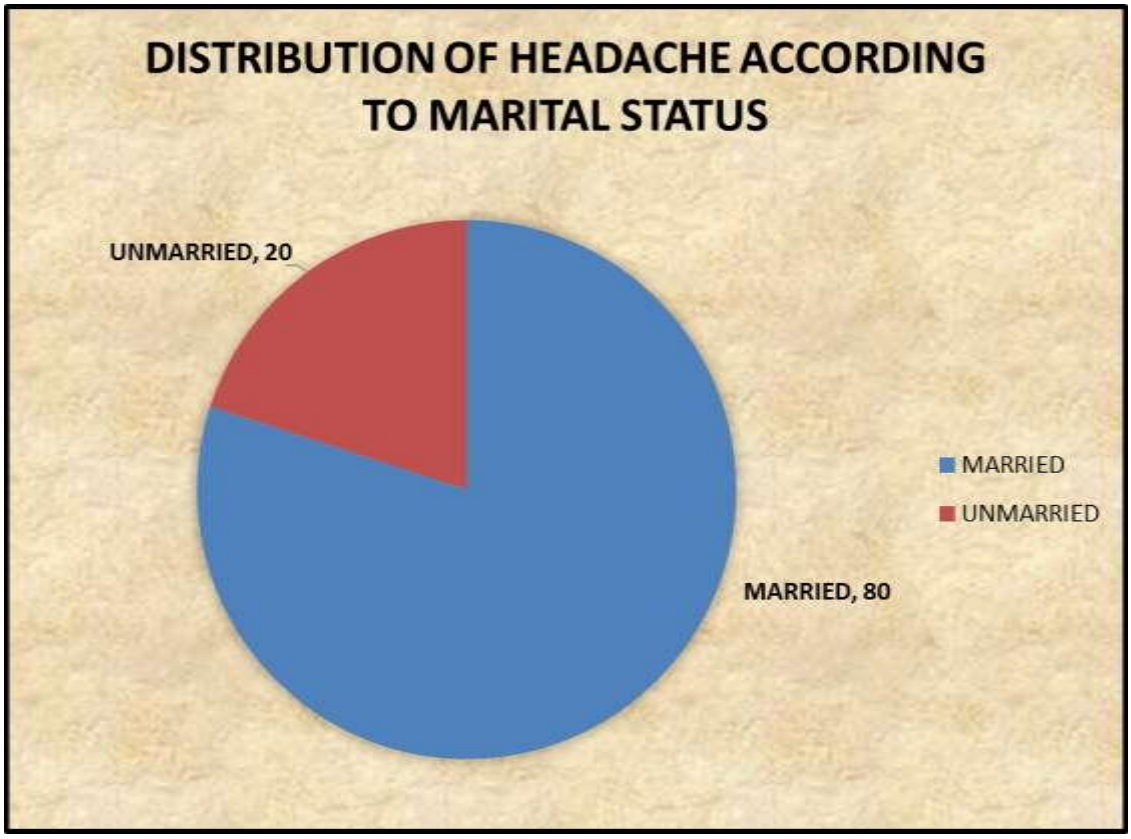
Age (years)	Number of patients	Percentage
<=20	7	7%
21-30	42	42%
31-40	17	17%
41-50	14	14%
51-60	10	10%
>60	10	10%
TOTAL	100	



The peak prevalence of headache in the present study of 100 cases is seen in 21 to 30 years age group, accounting for 42% of the total cases. The prevalence in the age group of 20 years and less is 7%, in 31 to 40 years is 17%, 41-50 years is 14%, 51-60 years is 10% and 60 years or more is 10%.

4. DISTRIBUTION OF HEADACHE ACCORDING TO MARITAL STATUS

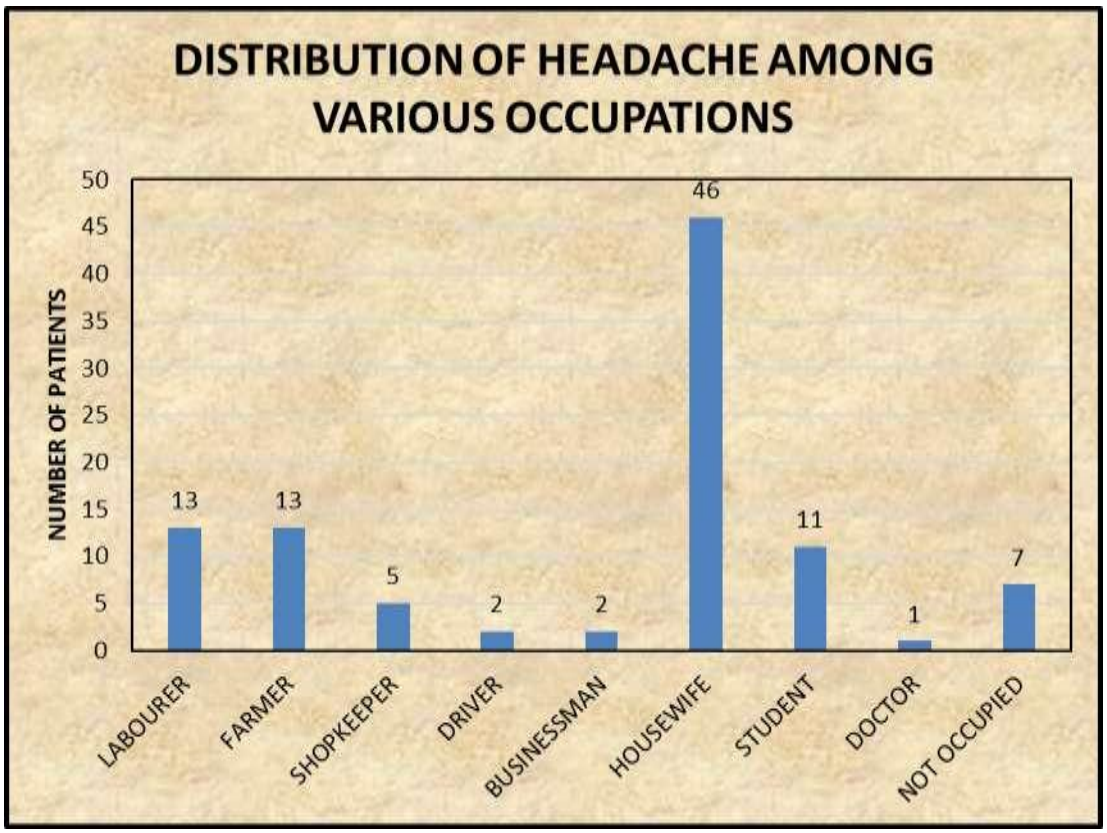
MARITAL STATUS	NUMBER OF PATIENTS
MARRIED	80
UNMARRIED	20
TOTAL	100



In the present study, among 100 patients with headache, 80 patients were married and 20 were unmarried.

5. DISTRIBUTION OF HEADACHE AMONG VARIOUS OCCUPATIONS

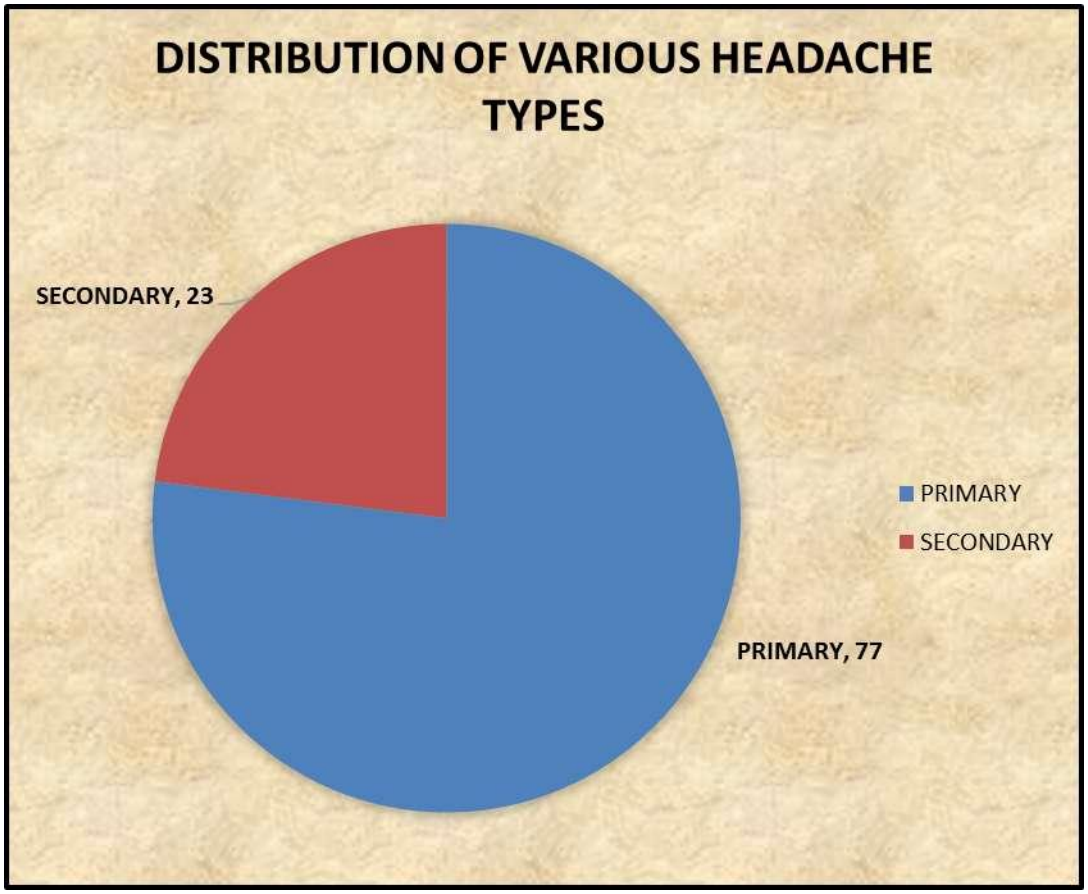
Occupation	Number of patients
Labourer	13
Farmer	13
Shopkeeper	5
Driver	2
Businessman	2
Housewife	46
Student	11
Doctor	1
Not occupied	7
Total	100



In the present study the prevalence of headache was maximum among housewives accounting for 46% of total patients, followed by laborers (13%), farmers (13%) and students (11%).

6. DISTRIBUTION OF VARIOUS HEADACHE TYPES

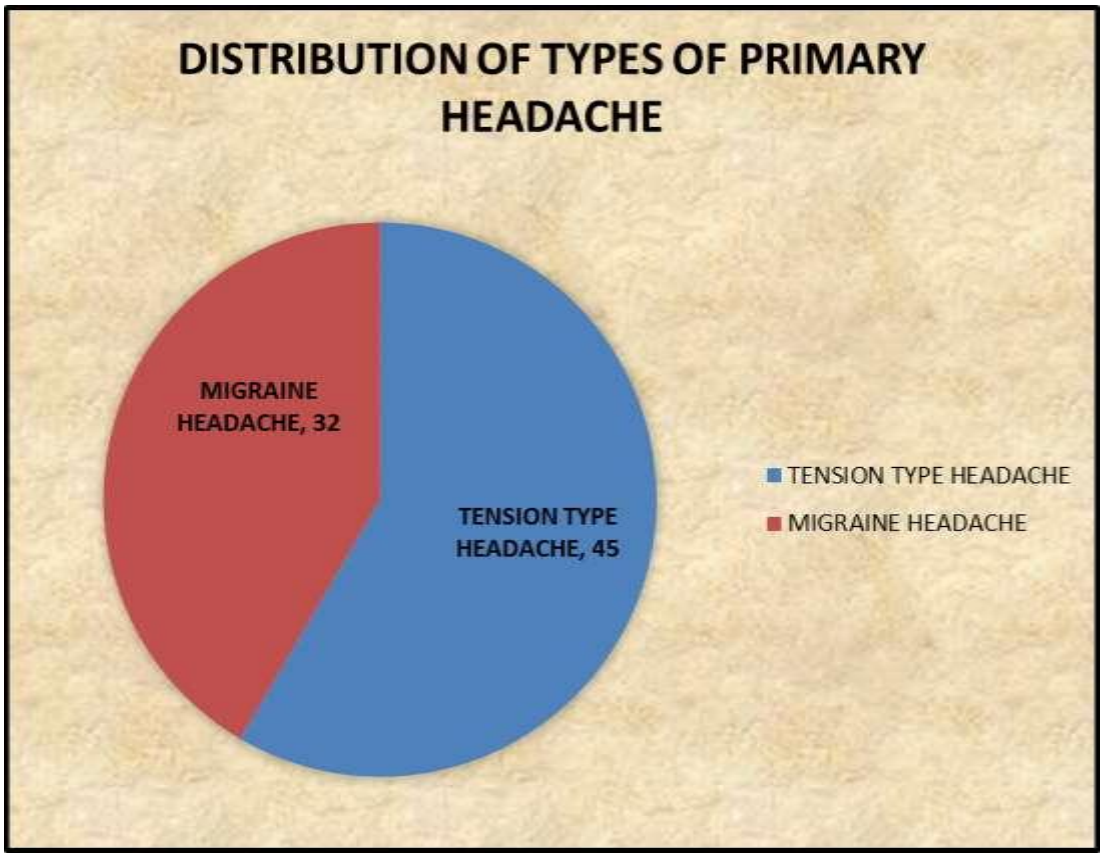
Headache type	Number of patients
Primary	77
Secondary	23
Total	100



In the present study among 100 patients, prevalence of primary headache was 77%, while that of secondary headache was 23%.

7. DISTRIBUTION OF THE TYPES OF PRIMARY HEADACHE

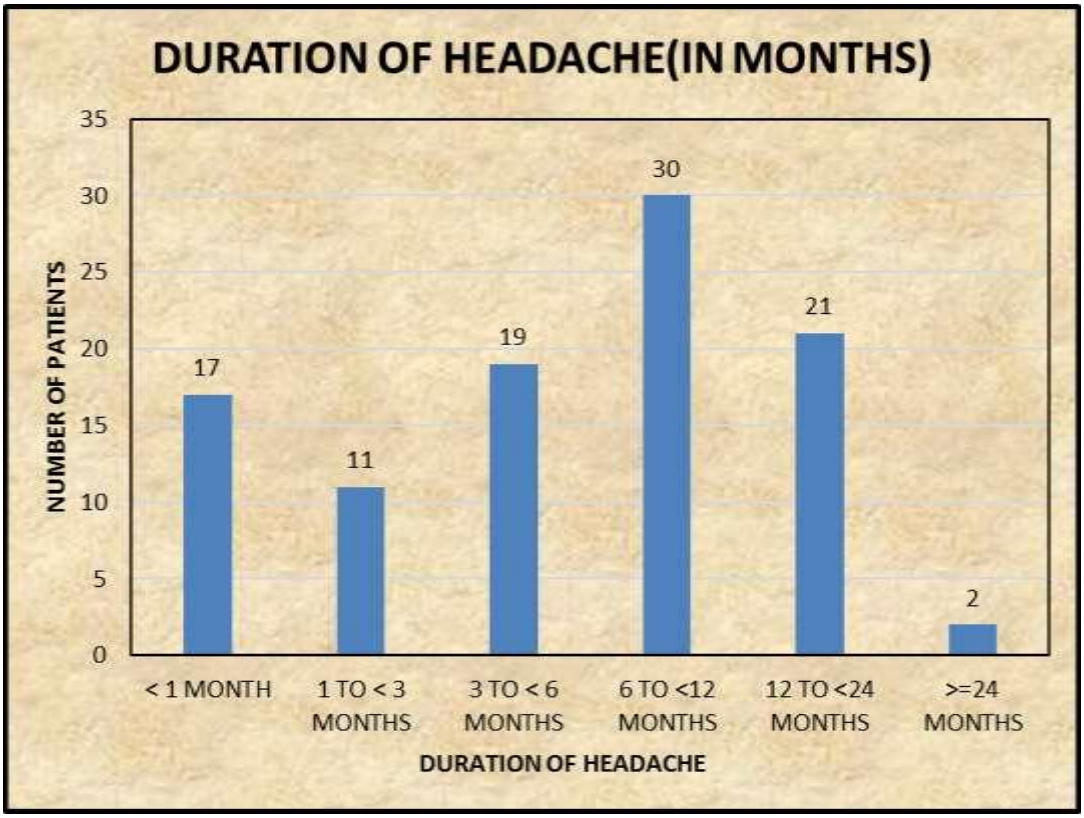
Type of primary headache	Number of patients	Percentage
TENSION TYPE HEADACHE	45	58.4%
MIGRAINE HEADACHE	32	41.6%
TOTAL	77	



In the present study, among the primary headache types, the prevalence of migraine headache was 41.6% and that of tension type headache was 58.4%. Thus, the most common headache type among primary headache was tension type headache.

8. DURATION OF HEADACHE (IN MONTHS)

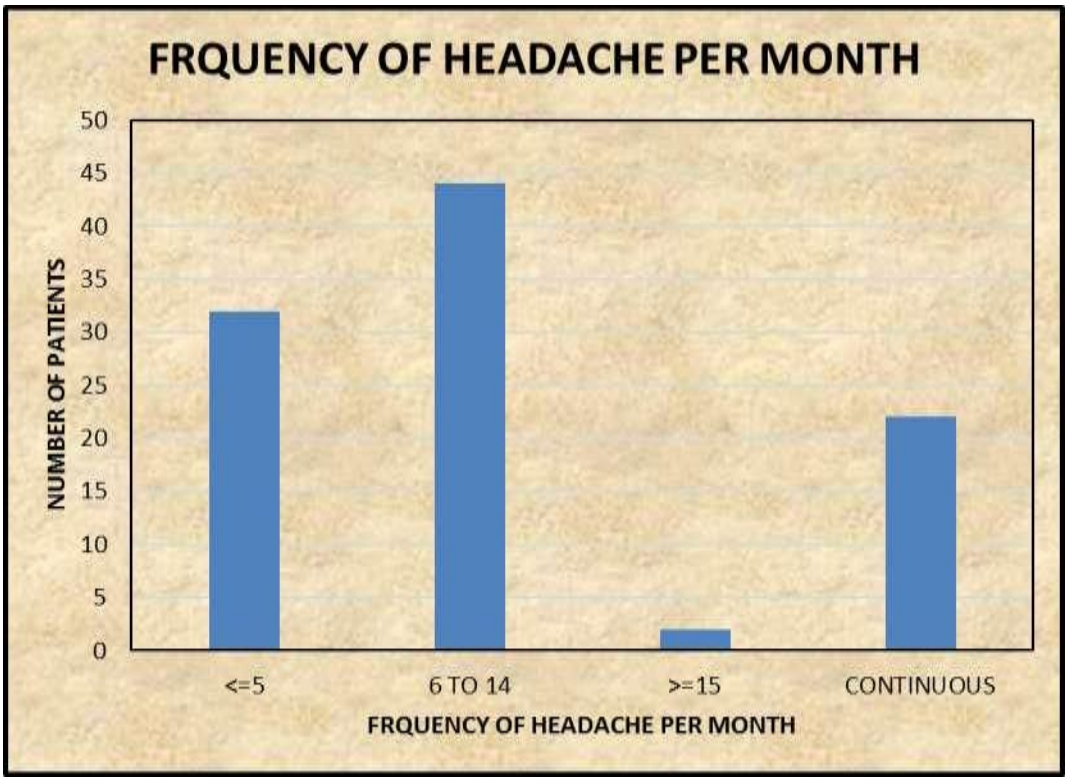
Duration of symptoms(months)	No of patients
< 1 MONTH	17
1 TO < 3 MONTHS	11
3 TO < 6 MONTHS	19
6 TO <12 MONTHS	30
12 TO <24 MONTHS	21
>=24 MONTHS	2
TOTAL	100



In the present study maximum patients (30%) had their symptom duration between 6 to 12 months.

9. FREQUENCY OF HEADACHE AMONG PATIENTS (EPISODES PER MONTH)

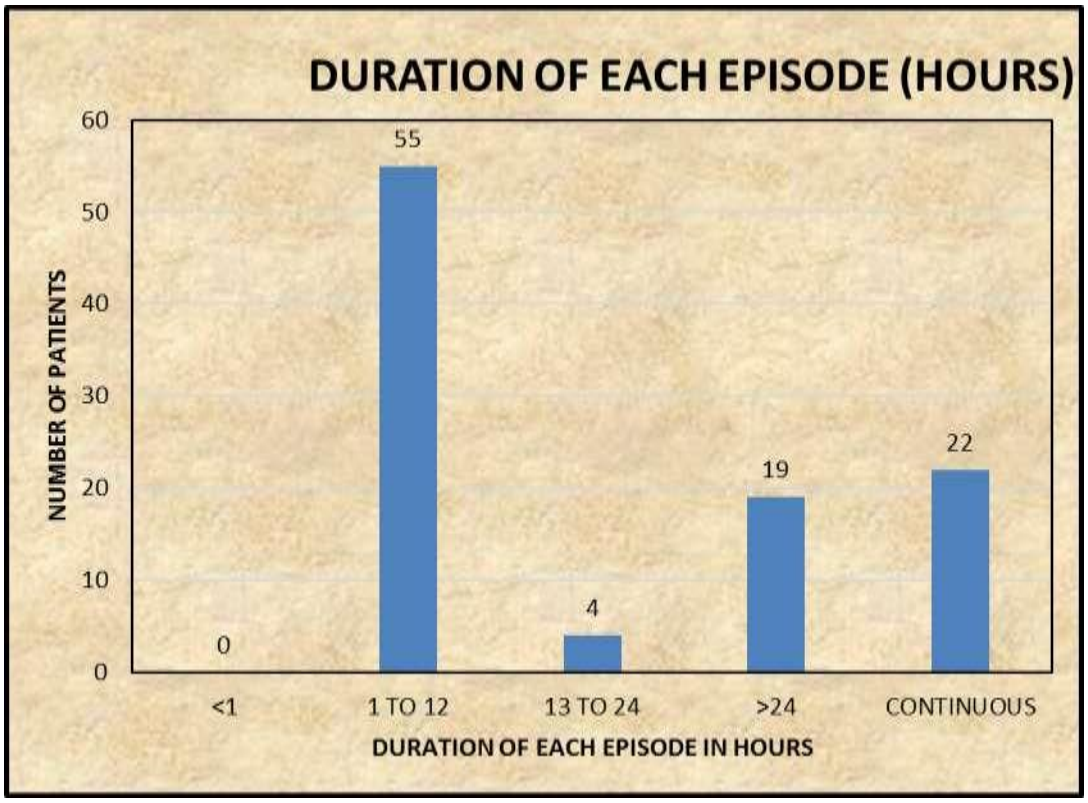
Frequency of headache (per month)	Number of patients	Percentage
<=5	32	32%
6 TO 14	44	44%
>=15	2	2%
CONTINUOUS	22	22%
TOTAL	100	



In the present study maximum patients had 6 to 14 episodes of headache per month which accounted for 44% of the total cases, while 32% patients had 5 or less episodes per month, 2% had 15 or more episodes per month, and 22% had continuous headache.

10. DURATION OF EACH EPISODE OF HEADACHE (IN HOURS) AMONG THE PATIENTS.

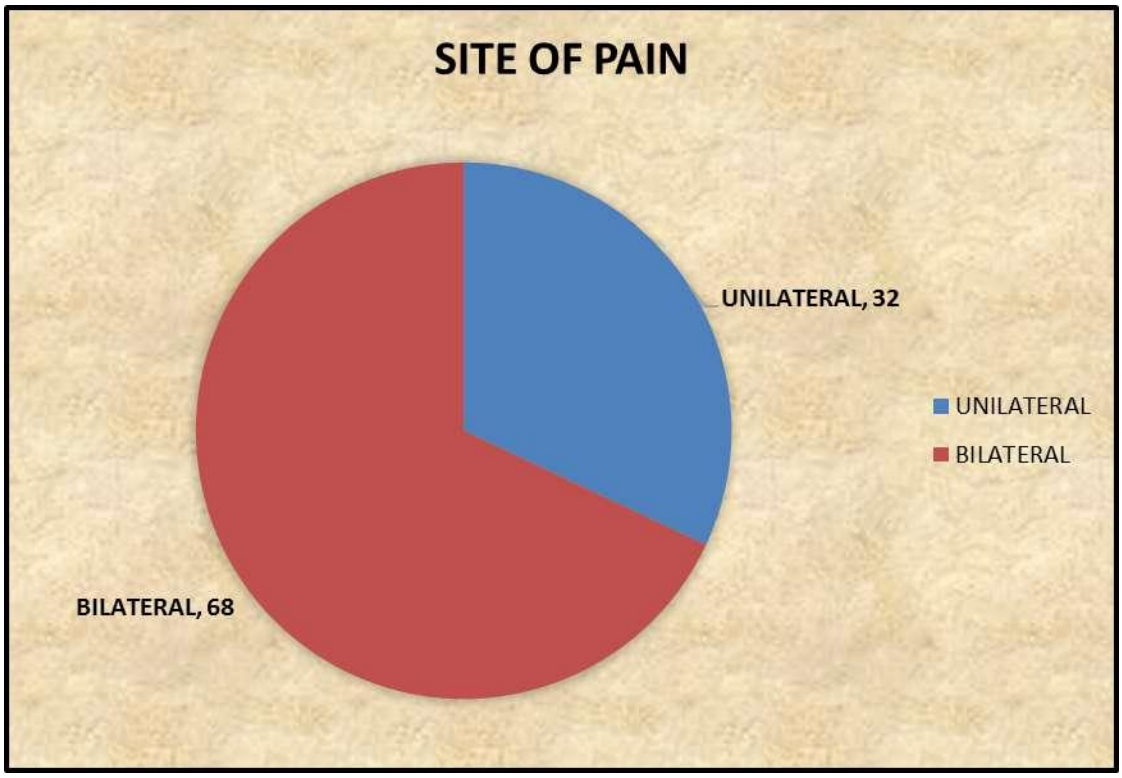
Duration of each episode(hours)	Number of patients
<1	0
1 TO 12	55
13 TO 24	4
>24	19
CONTINUOUS	22
TOTAL	100



In the present study, 55% patients had each episode of headache lasting for around 1 to 12 hours, while 19% had each episode lasting for more than 24 hours, 4% had each episode for 13 to 24 hours and 22% had continuous headache.

11. SITE OF PAIN AMONG THE PATIENTS HAVING HEADACHE.

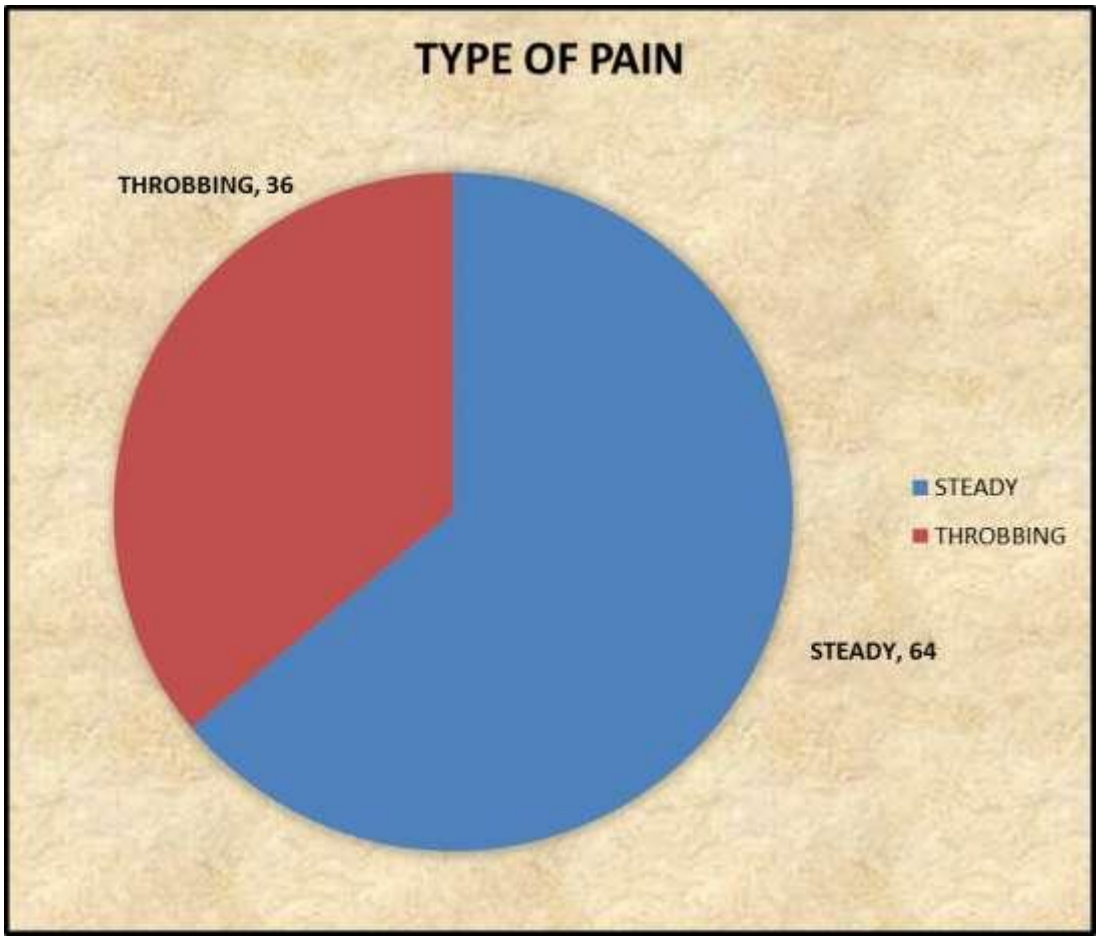
Site of pain	Number of patients
UNILATERAL	32
BILATERAL	68
TOTAL	100



In the present study of 100 cases of headache, 68% patients had bilateral headache, while 32% patients had unilateral headache.

12. Type Of Pain Among The Headache Patients

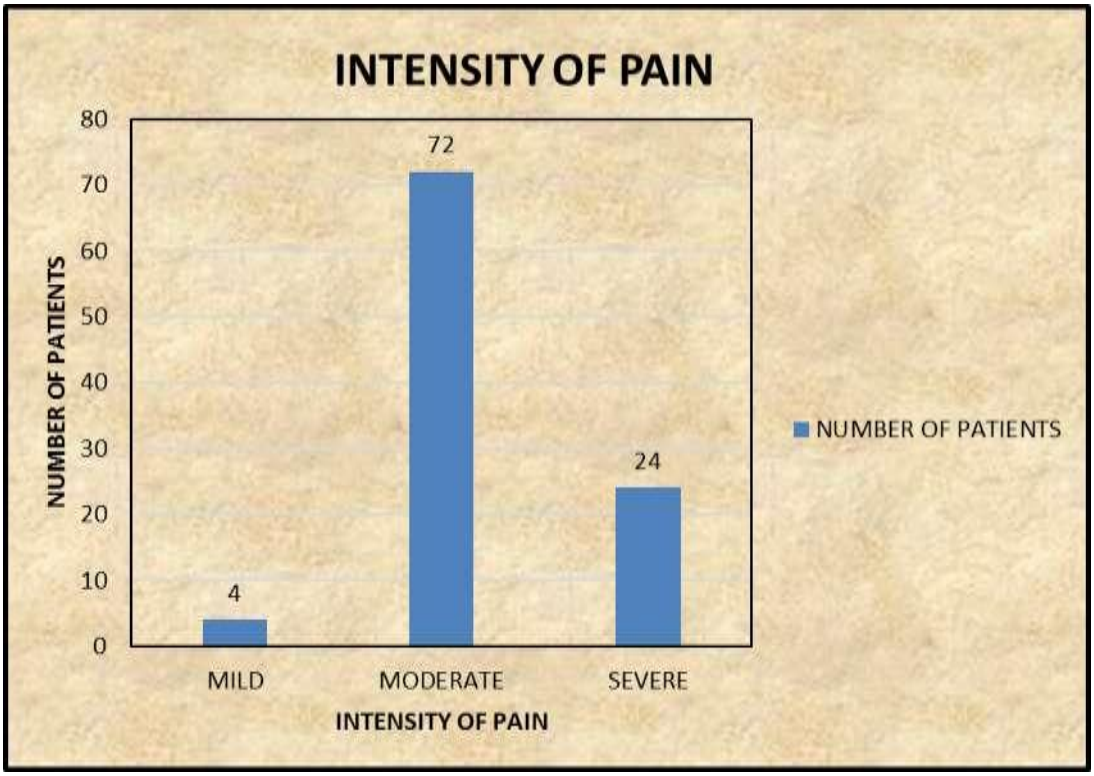
Type of pain	Number of patients
STEADY	64
THROBBING	36
TOTAL	100



In the present study, the type of pain was throbbing in 36% of patients, while it was steady in 64% of the patients.

13. INTENSITY OF THE PAIN AMONG HEADACHE PATIENTS

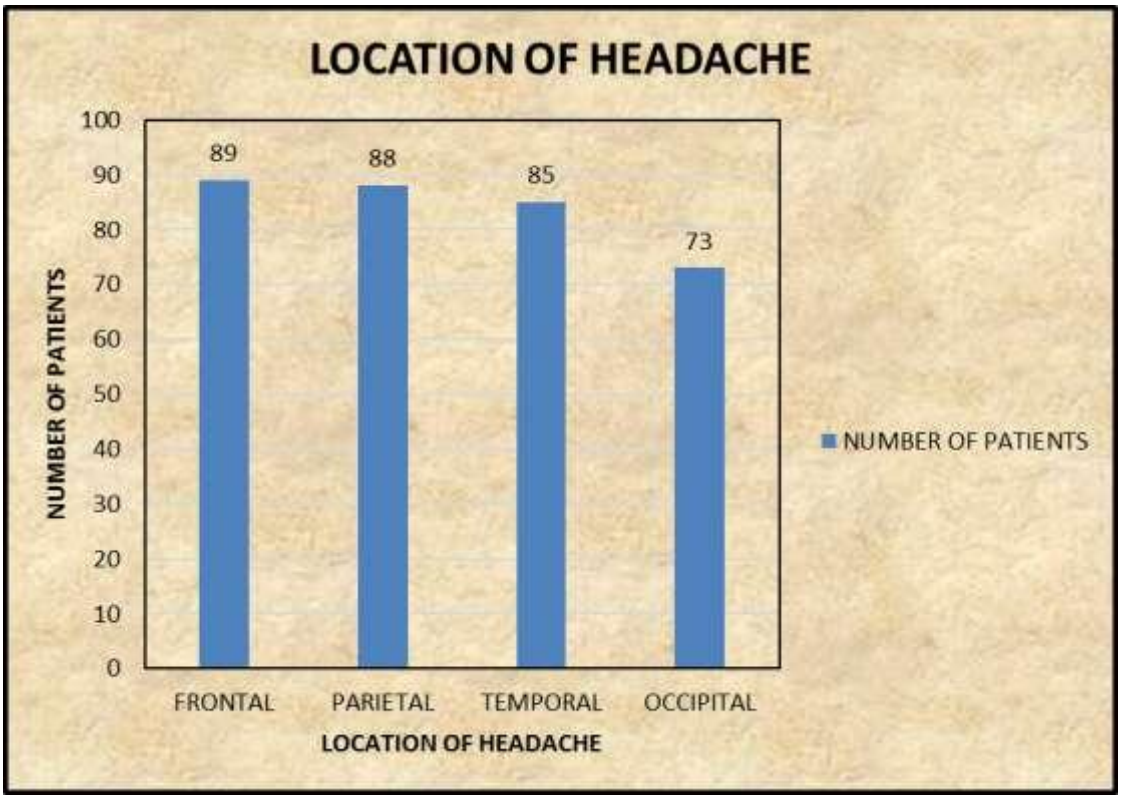
Intensity of pain	Number of patients
MILD	4
MODERATE	72
SEVERE	24
TOTAL	100



In the present study, the intensity of pain of headache was moderate in72% of patients, while it was severe in 24% of the patients and mild in 4% of the patients.

14. LOCATION OF THE HEADACHE AMONG THE PATIENTS

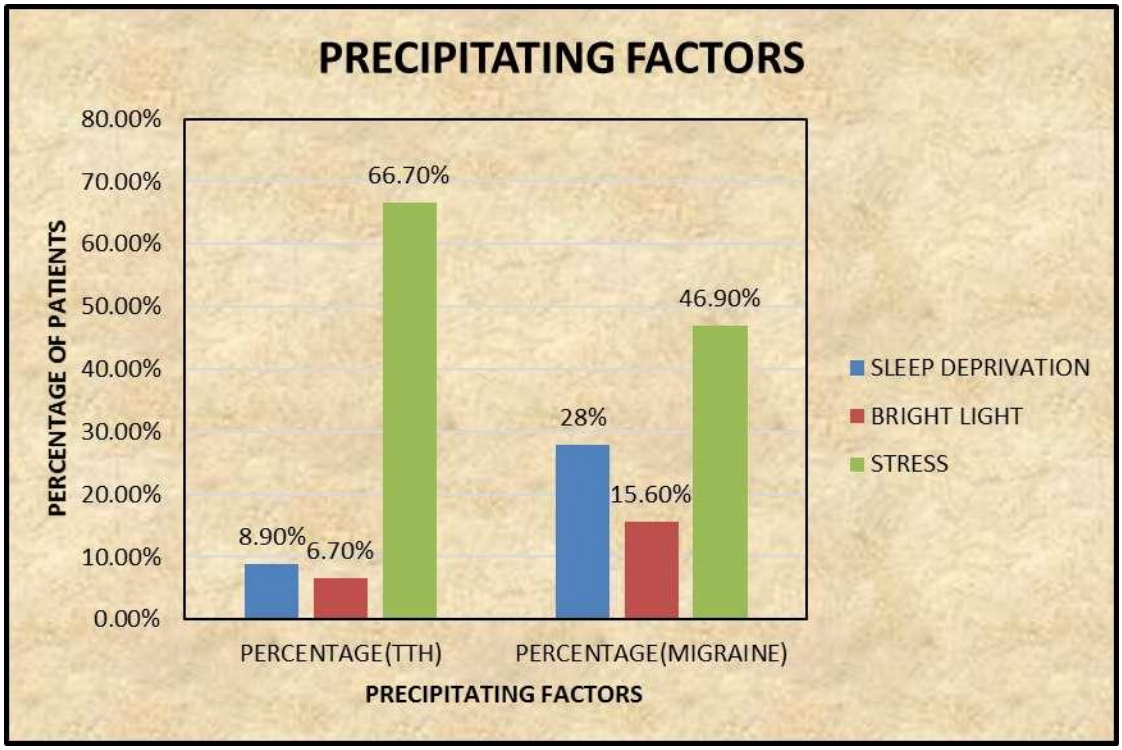
Location of pain	Number of patients
FRONTAL	89
PARIETAL	88
TEMPORAL	85
OCCIPITAL	73



In the present study, 89% patients had headache involving frontal region, 88% involved parietal region, 89% involved temporal region and 73% involved occipital region.

15. PRECIPITATING FACTORS AMONG MIGRAINE AND TENSION TYPE HEADACHES

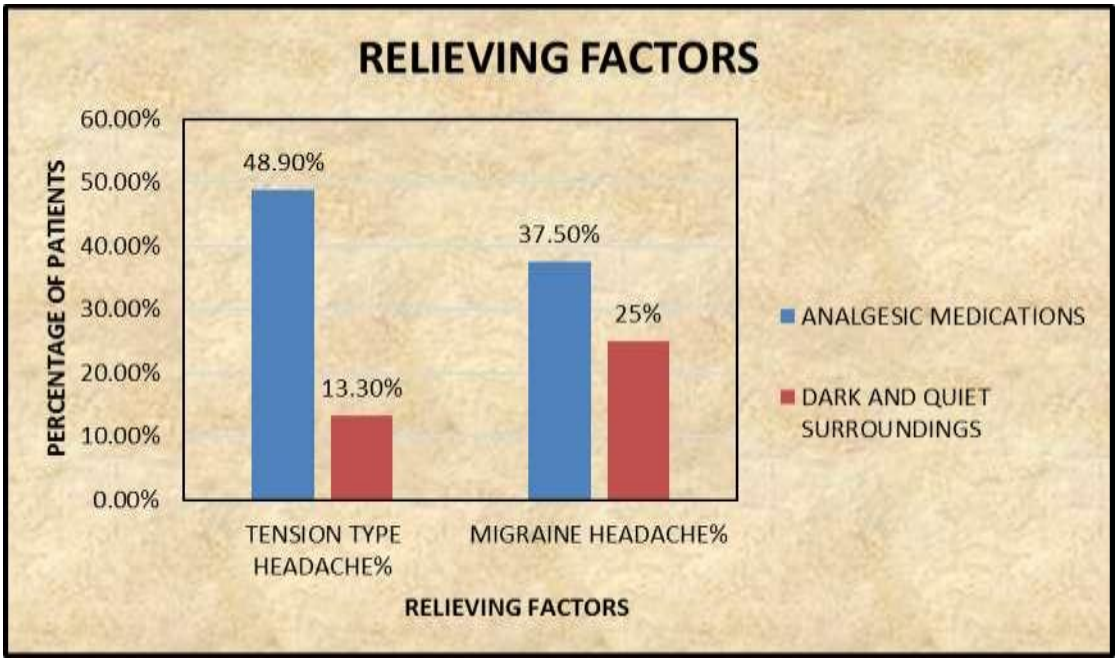
Precipitating factors	Tension Type headache (n=45)	Percentage (TTH)	Migraine headache (n=32)	Percentage (migraine)
SLEEP DEPRIVATION	4	8.90%	9	28%
BRIGHT LIGHT	3	6.70%	5	15.60%
STRESS	30	66.70%	15	46.90%



In the present study, sleep deprivation was found to be precipitating factor in 8.9% patients of tension type headache and 28% patients of migraine headache, while bright light was precipitating factor in 6.7% cases of tension headache and 15.6% cases of migraine headache and stress was precipitating factor in 66.7% cases of tension headache and 46.9% cases of migraine headache.

16. RELIEVING FACTORS IN TENSION TYPE AND MIGRAINE HEADACHES

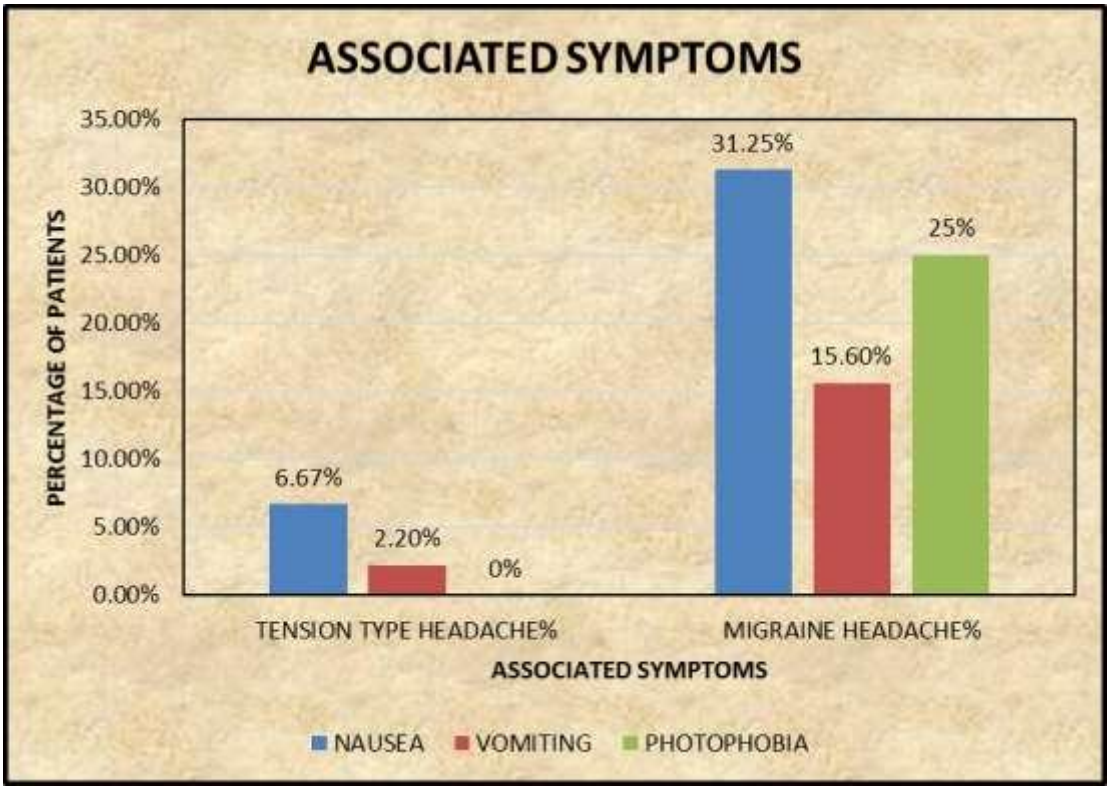
Relieving factor	Tension type headache	Tension type headache%	Migraine headache	Migraine headache%
ANALGESIC MEDICATIONS	22	48.90%	12	37.50%
DARK AND QUIET SURROUNDINGS	6	13.30%	8	25%



In the present study, analgesic medications were found to be relieving factors in 48.9% patients of tension type headache and 37.5% patients of migraine headaches, while dark and quiet environment was a relieving factor in 13.3% cases of tension headache and 25% cases of migraine headaches.

17. ASSOCIATED SYMPTOMS IN TENSION TYPE HEADACHE AND MIGRAINE HEADACHE

Associated symptoms	Tension type headache	Tension type headache%	Migraine headache	Migraine headache%
NAUSEA	3	6.67%	10	31.25%
VOMITING	1	2.20%	5	15.60%
PHOTOPHOBIA	0	0%	8	25%

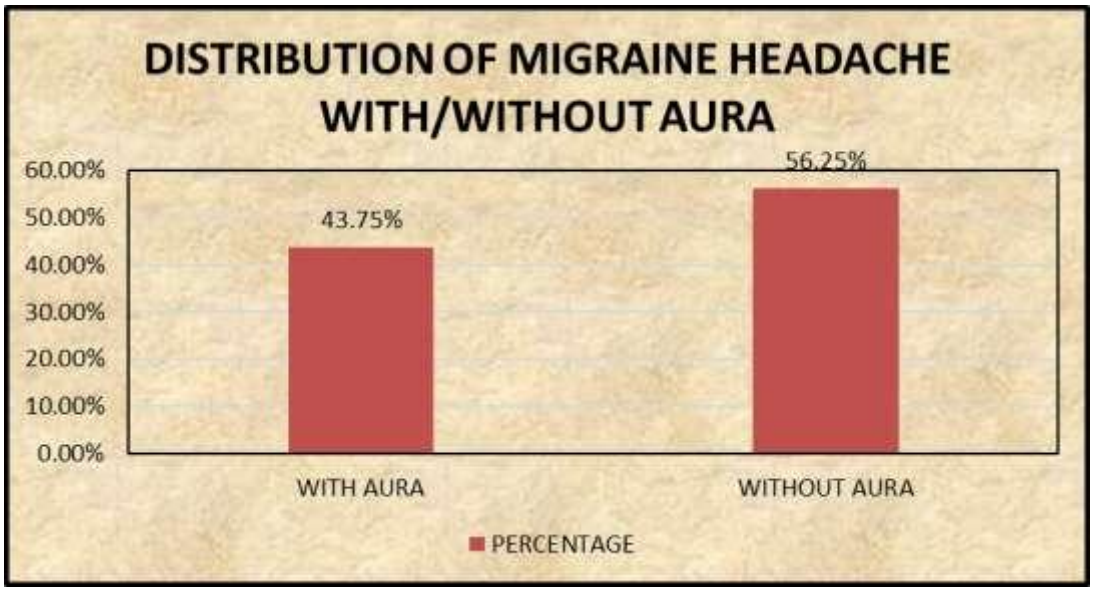


In the present study nausea was an associated symptom in 6.67% of patients with tension type headache and 31.25% patients with migraine headache, vomiting was associated with 2.2% of patients with tension headache and 15.6% of patients with migraine headache while photophobia was associated with 25% of patients with migraine headache.

18. DISTRIBUTION OF MIGRAINE HEADACHE WITH/ WITH OUT AURA

Migraine	Number of patients	Percentage

WITH AURA	14	43.75%
WITHOUT AURA	18	56.25%

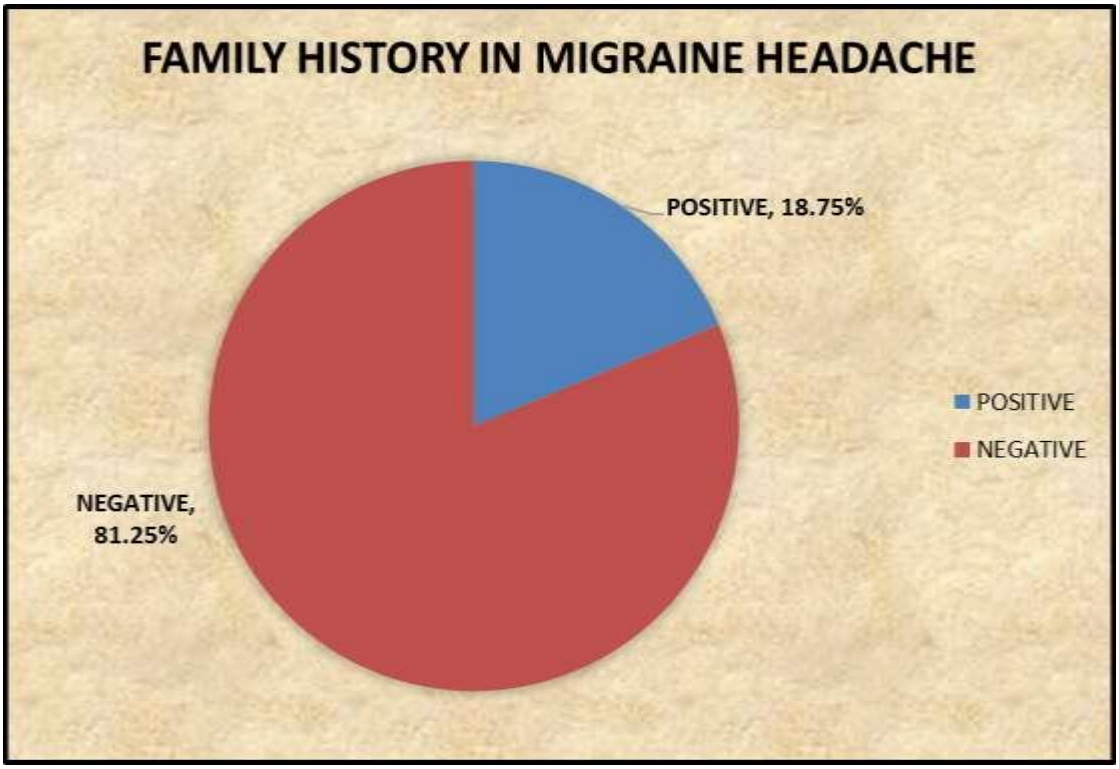


In the present study, 43.75% patients with migraine headache had accompanying aura while 56.25% of migraine patients did not experience aura.

19. FAMILY HISTORY IN PATIENTS WITH MIGRAINE HEADACHE

Family history	Number of patients	Percentage
POSITIVE	6	18.75%

NEGATIVE	26	81.25%
TOTAL	32	

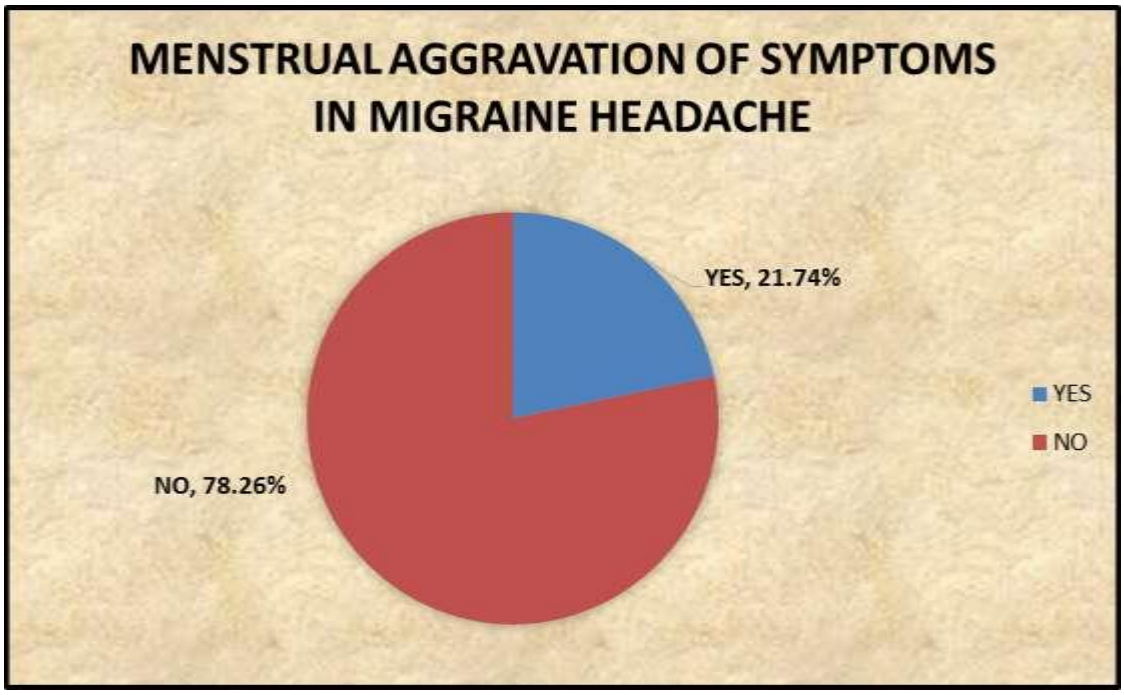


In the present study, positive family history was found in 18.75% patients with migraine headache while it was negative in 81.25% patients with migraine.

20. MENSTRUAL AGGRAVATION OF SYMPTOMS IN FEMALE PATIENTS WITH MIGRAINE HEADACHE

Menstrual aggravation of symptoms	Number of patients	Percentage

YES	5	21.74%
NO	18	78.26%
TOTAL FEMALE PATIENTS	23	

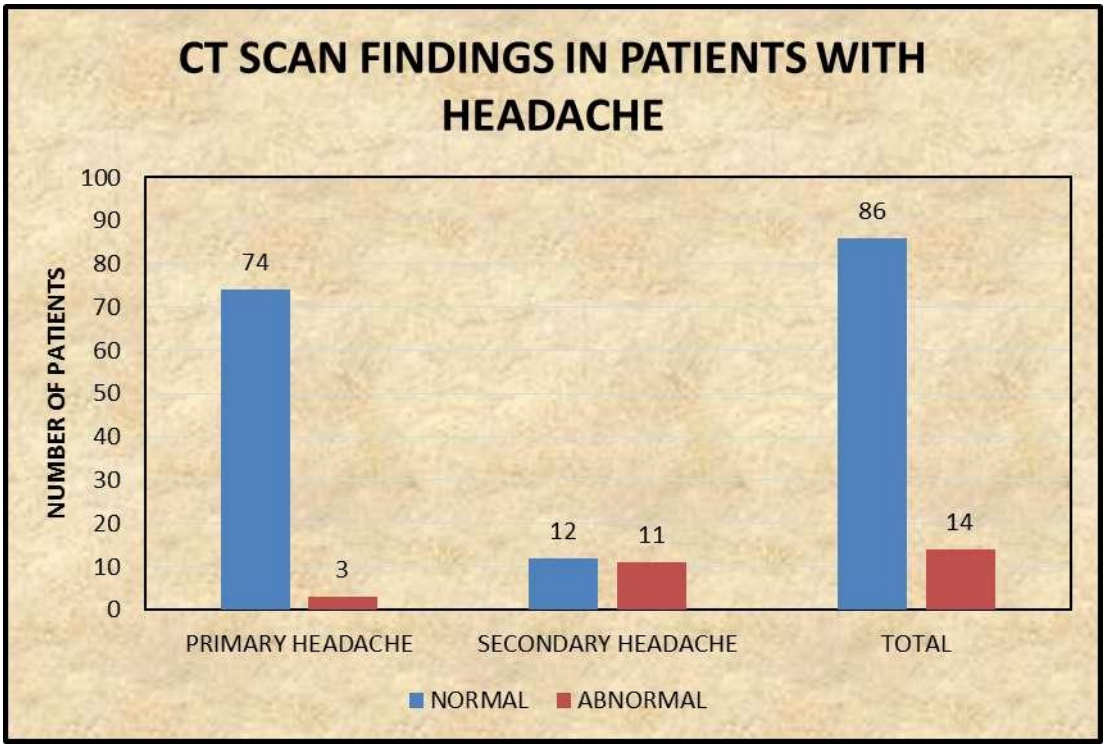


In the present study, menstrual aggravation of the headache symptoms was found in 21.74% of female patients with migraine headache.

21. CT SCAN FINDINGS IN PATIENTS WITH HEADACHE

Ct scan findings	Primary headache	Secondary headache	Total

NORMAL	74 (96%)	12 (52%)	86
ABNORMAL	3 (4%)	11 (48%)	14
	77	23	100



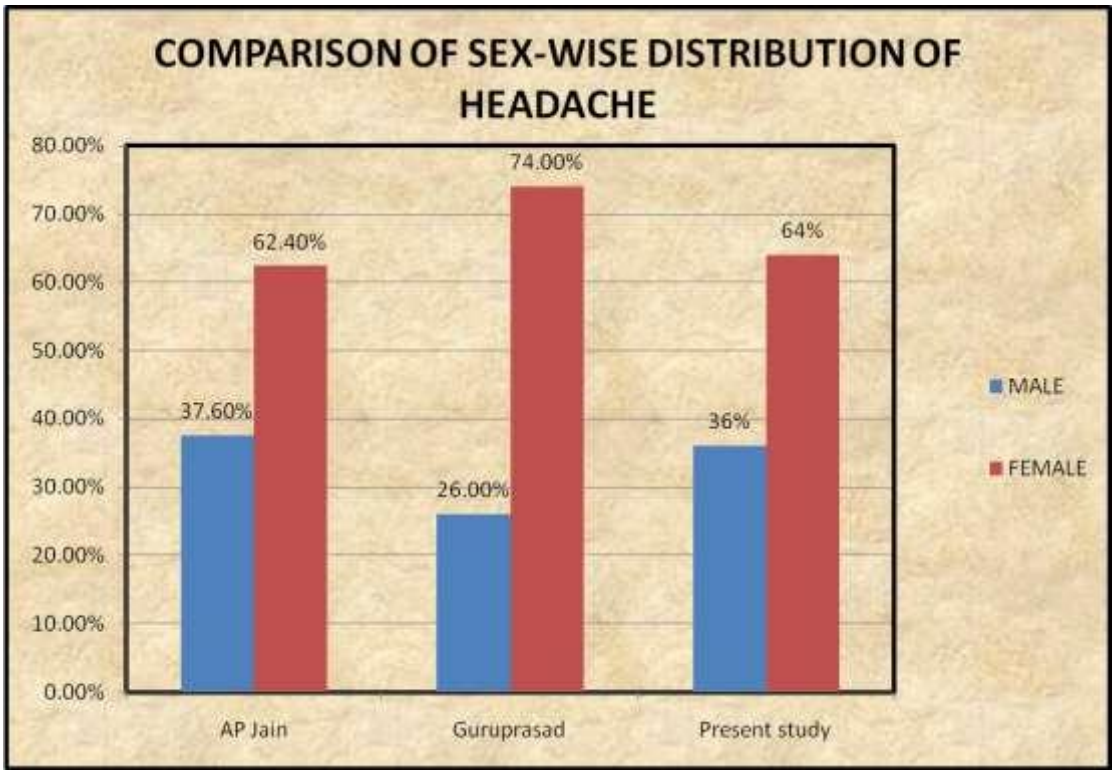
In the present study 86% of patients with headache had normal CT Scan findings, while among 77 patients with primary headache, 74 patients had normal CT Scan and among 23 patients with secondary headache, 12 patients had normal CT Scan.

DISCUSSION

Headache is a very common complaint of the patients attending the tertiary care center. In the present study, the clinical profile of 100 patients presenting with the complaint of headache at the tertiary care center has been studied. During the study, following observation were made with respect to age group, sex group, patient characteristics and clinical presentation.

1. Comparison of the distribution of headache in patients according to Sex

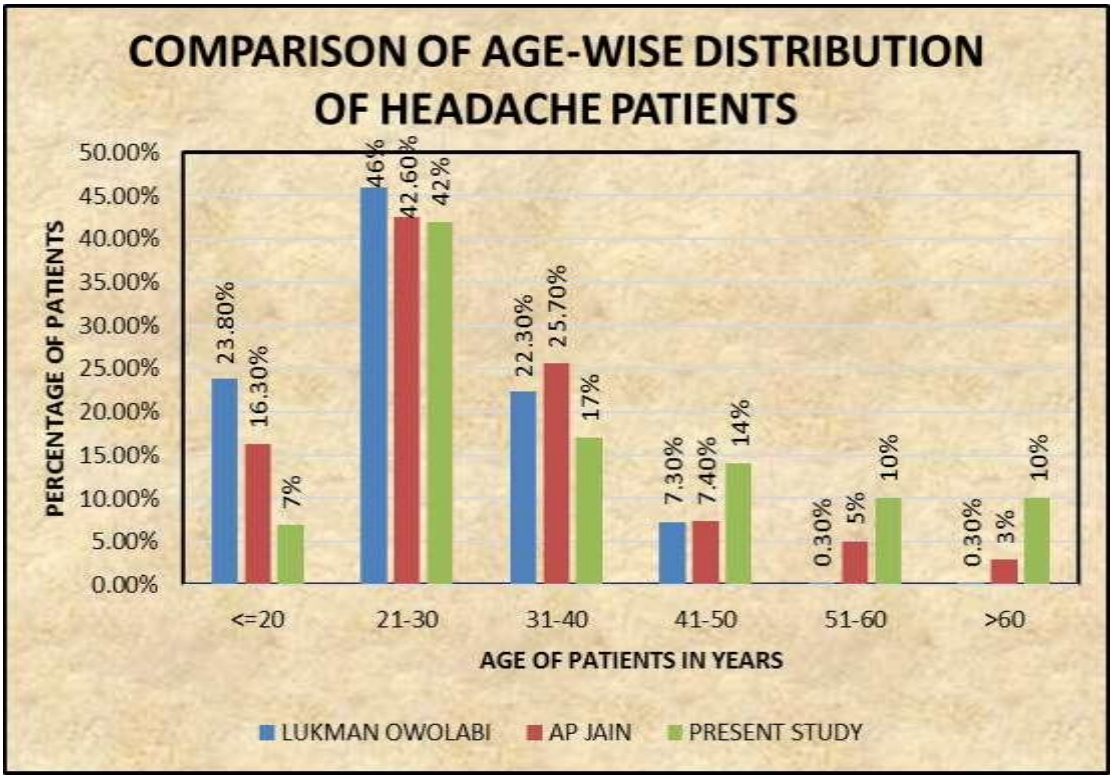
Sex	AP Jain[112]	Guruprasad[110]	Present study
MALE	37.60%	26.00%	36%
FEMALE	62.40%	74.00%	64%



In the present study, amongst the 100 patients, 36% patients were male and 64% patients were female, these observations were similar to many other studies carried out in different decades in our country.

2. Comparison of the distribution of headache among various agegroups

Age (years)	Lukman owolabi[111]	AP Jain[112]	Present study
<=20	23.80%	16.30%	7%
21-30	46%	42.60%	42%
31-40	22.30%	25.70%	17%
41-50	7.30%	7.40%	14%
51-60	0.30%	5%	10%
>60	0.30%	3%	10%

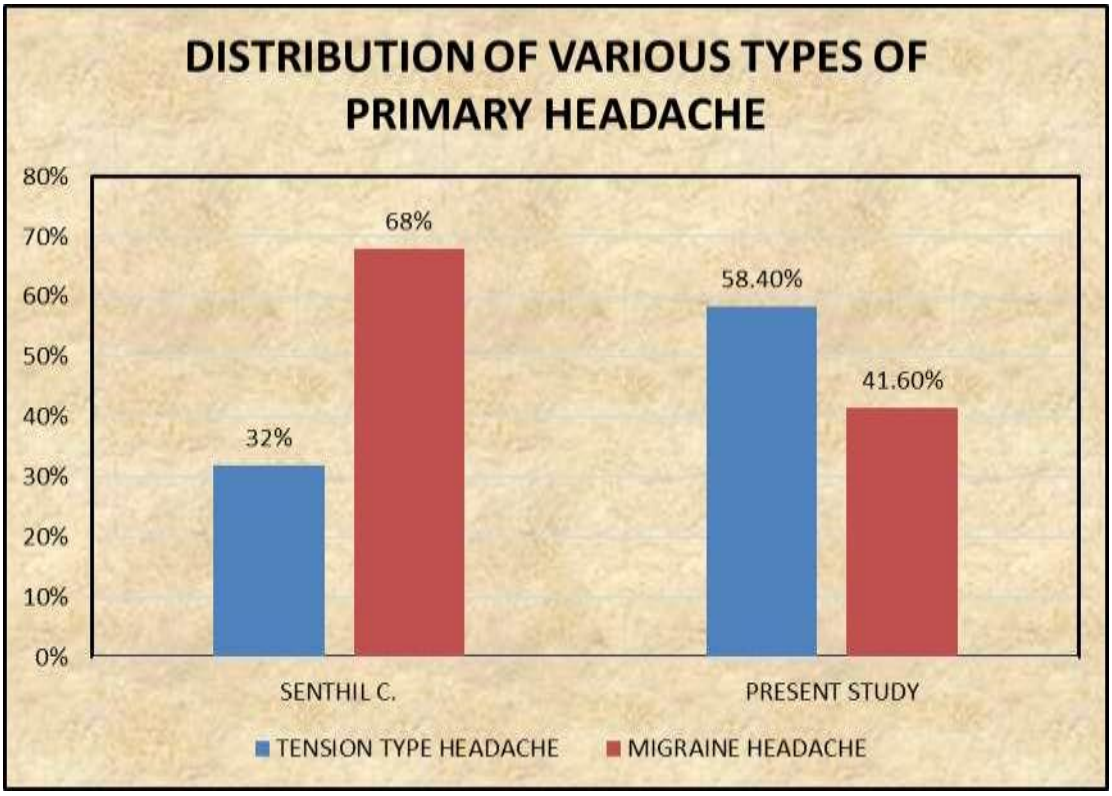


In the present study, the peak prevalence of headache is seen in 21 to 30 years age group, accounting for 42% of the total cases. The prevalence in the age group of 20 years and less is 7%, in 31 to 40 years is 17%, 41-50 years is 14%, 51-60 years is 10% and 60 years or more is 10%. These observations were similar to many other studies carried out at different times.

3. The comparison of the distribution of the types of primary headache

Type of primary headache	Senthil c[114].	Present study

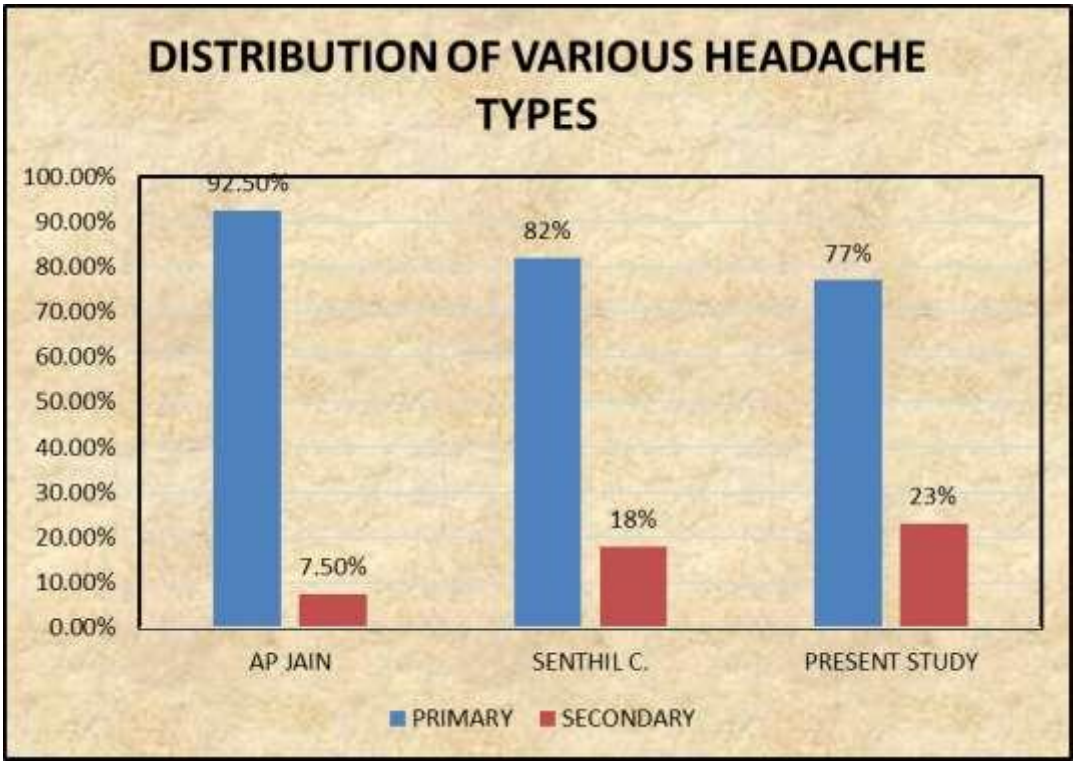
TENSION TYPE HEADACHE	32%	58.40%
MIGRAINE HEADACHE	68%	41.60%



In the present study, among the primary headache types, the prevalence of migraine headache was 41.6% and that of tension type headache was 58.4%. Thus, the most common headache type among primary headache was tension type headache, while in the study done by Senthil C, the most common primary headache type was migraine headache which constituted 68% of the patients.

4. Comparison of the Distribution of various headache types

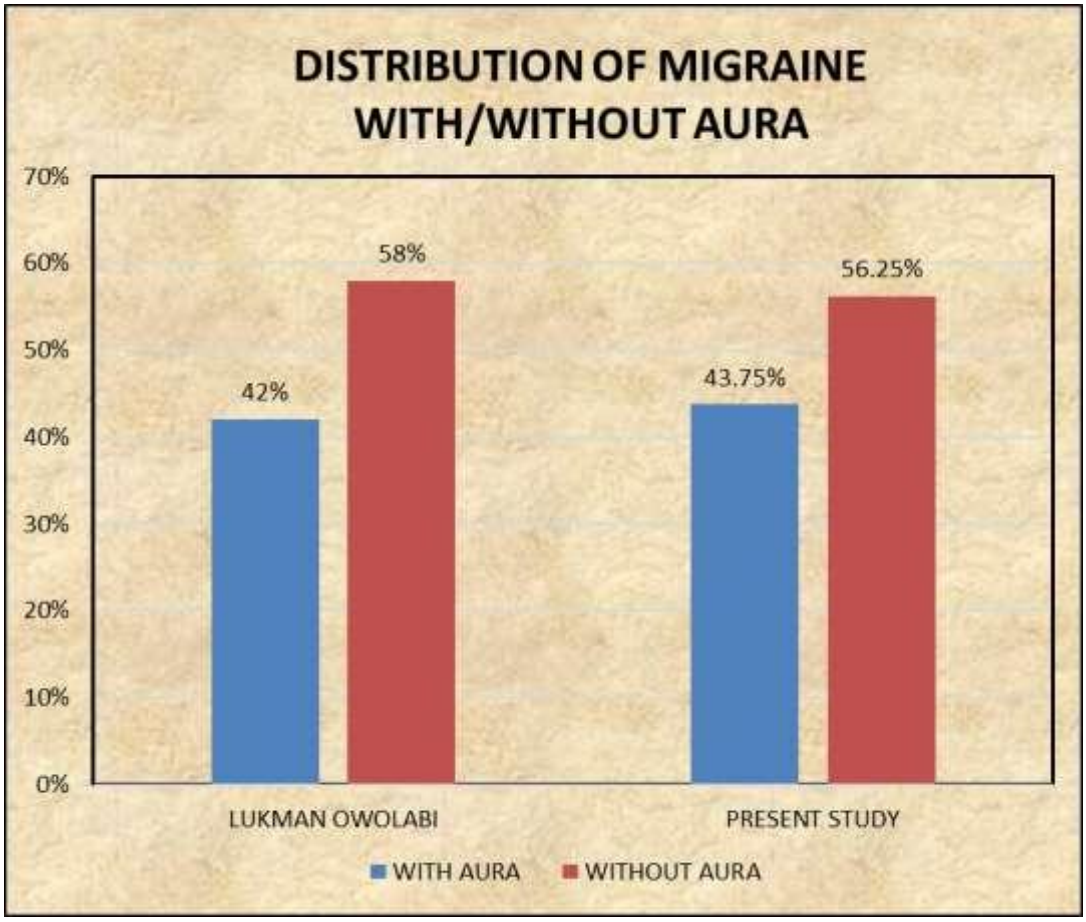
Headache type	AP Jain[112]	Senthil c[114]	Present study
PRIMARY	92.50%	82%	77%
SECONDARY	7.50%	18%	23%



In the present the prevalence of primary headache was 77%, while that of secondary headache was 23% while in the study done by AP Jain, the prevalence of primary headache was 92.5% and of secondary was 7.5% and in the study done by Senthil C, the prevalence of primary headache was 82% and secondary was 18%.

5. Comparison of the distribution of migraine headache with/without aura

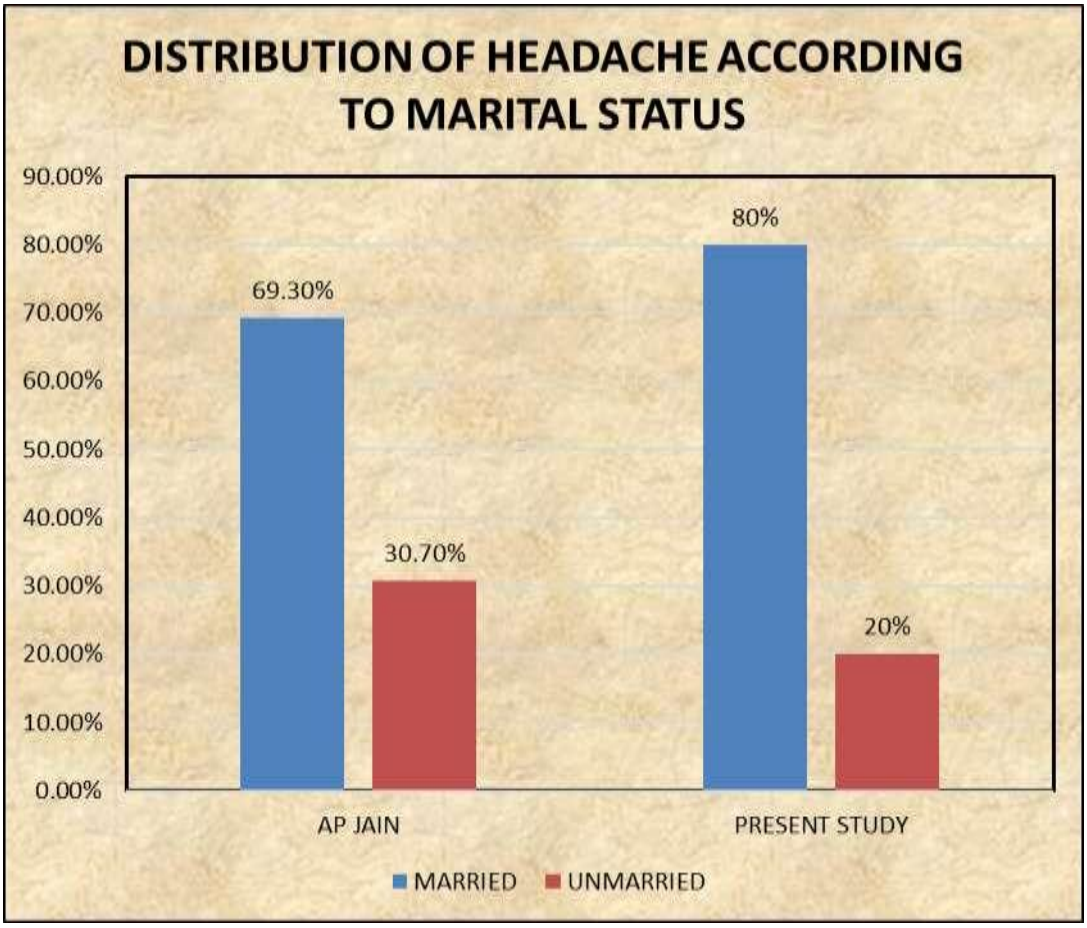
Migraine	Lukman owolabi[111]	Present study
WITH AURA	42%	43.75%
WITHOUT AURA	58%	56.25%



In the present study, 43.75% patients had migraine with aura while 56.25% did not experience aura, thus more common was migraine headache without aura. This observation is similar to various other studies.

6. Comparison of the distribution of headache according to marital status

Marital status	Ap jain[112]	Present study
MARRIED	69.30%	80%
UNMARRIED	30.70%	20%

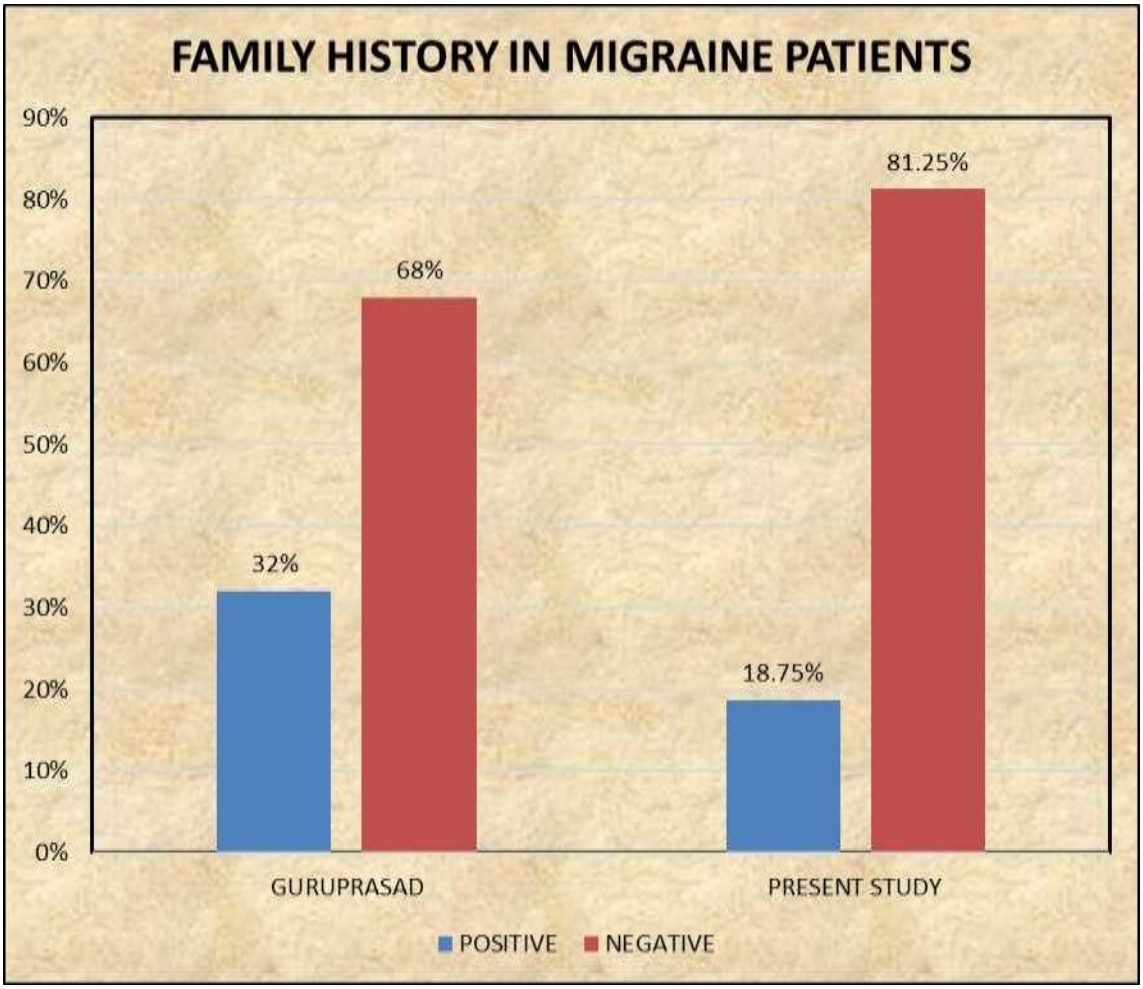


In the present study, 80% of the patients were married and 20% were unmarried while in the study done by AP Jain 69.3% were married

30.70% were unmarried.

7. Family history in patients with migraine headache

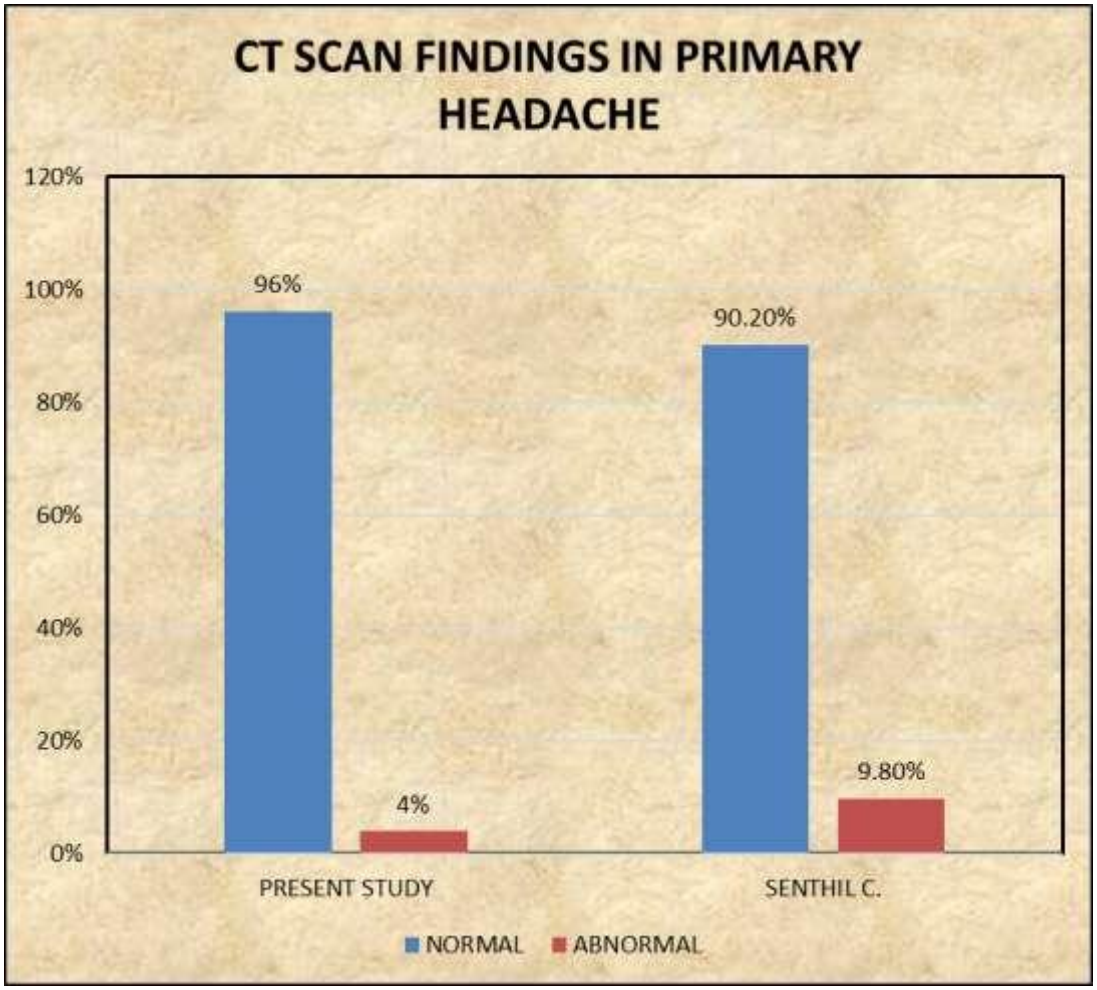
Family history	Guruprasad[110]	Present study
POSITIVE	32%	18.75%
NEGATIVE	68%	81.25%



In the present study, positive family history was found in 18.75% patients while it was negative in 81.25% patients with migraine, while in the study done by Guruprasad, family history was found positive in 32% patients and negative in 68% patients.

8. CT Scan findings in patients with primary headache

Ct scan findings	Present study	Senthil c[114].
NORMAL	96%	90.20%
ABNORMAL	4%	9.80%



In the present study 96% of patients with primary headache had normal CT Scan findings while in the study done by Senthil C, 90.20% patients had normal CT Scan findings.

SUMMARY AND CONCLUSION

- The present study is a study of the clinical profile of headache in the patients attending the tertiary care center, in which 100 such cases were studied.
- In the present study the prevalence of headache was more common in female (64%) as compared to male (36%).
- In the present study the tension type headache was more common in female as compared to male (male to female ratio of 1:1.5). Also the migraine headache was found to be more common among female than male (male to female ratio of 1:2.55).
- The peak prevalence of headache in the present study was seen in 21 to 30 years age group, accounting for 42% of the total cases. The prevalence in the age group of 20 years and less is 7%, in 31 to 40 years is 17%, 41-50 years is 14%, 51-60 years is 10% and 60 years or more is 10%.
- Headache was found to be more common in married patients (80%) as compared to unmarried patients (20%).
- In the present study the prevalence of headache was maximum among housewives accounting for 46% of total patients, followed by laborers (13%), farmers (13%) and students (11%).
- Among the headache types, the most common was the primary headache (77%) as compared to secondary headache (23%).
- In the present study, the most common headache type among primary headache was tension type headache (58.4%) and second most common type was migraine headache (41.6%).
- In the present study maximum patients (30%) had their symptom duration between 6 to 12 months.
- In the present study maximum patients had 6 to 14 episodes of headache per month which accounted for 44% of the total cases, while 32% patients had 5 or less episodes per month, 2% had 15 0r more episodes per month and 22% had continuous headache.
- In the present study, 55% patients had each episode of headache lasting for around 1 to 12 hours, while 19% had each episode lasting for more than 24 hours, 4% had each episode for 13 to 24 hours and 22% had continuous headache.

- In the present study the most common site of pain was bilateral in 68% of patients, while 32% patients had unilateral headache.
- In the present study, the most common type of pain was steady in 64% of the patients while 36% patients had throbbing pain.
- In the present study, the intensity of pain of headache was moderate in72% of patients, while it was severe in 24% of the patients and mild in 4% of the patients.
- In the present study, 89% patients had headache involving frontal region, 88% involved parietal region, 89% involved temporal region and 73% involved occipital region.
- In the present study, sleep deprivation was found to be precipitating factor in 8.9% patients of tension type headache and 28% patients of migraine headache, while bright light was precipitating factor in 6.7% cases of tension headache and 15.6% cases of migraine headache.
- Stress was the most common precipitating factor in 66.7% cases of tension headache and 46.9% cases of migraine headache.
- In the present study, analgesic medications were found to be relieving factors in 48.9% patients of tension type headache and 37.5% patients

  of migraine headaches, while dark and quiet environment was a relieving factor in 13.3% cases of tension headache and 25% cases of migraine headaches.
- In the present study nausea was an associated symptom in 6.67% of patients with tension type headache and 31.25% patients with migraine headache, vomiting was associated with 2.2% of patients with tension headache and 15.6% of patients with migraine headache while photophobia was associated with 25% of patients with migraine headache.
- In the present study migraine headache without aura (56.25%) was more common than migraine with aura (43.75%).
- In the present study, positive family history was found only in 18.75% patients with migraine headache.
- In the present study, menstrual aggravation of the headache symptoms was found in 21.74% of female patients with migraine headache.

In the present study 86% of patients with headache had normal CT Scan findings, while among 77 patients with primary headache, 74 patients had normal CT Scan (96%) and among 23 patients with secondary headache, 12 patients had normal CT Scan (52%).

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KEY TO MASTER CHART

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Clinical profile of patients with chronic headache in a tertiary care hospital Senthil C., GunasekaranN.

KEY TO MASTER CHART

- Y: Yes / Present
- N: No/ Absent
- Sex:
    - M: Male ▪F: Female Occupation:
    - H: House-wife
    - L: Labourer
    - SK: Shopkeeper
    - FR: Farmer
    - B: Businessman
    - S: Student
    - D: Driver
    - DR: Doctor ▪- : Not occupied Marital status:
    - M: Married
    - U: Unmarried
- DOH: Duration Of Headache
- FOH: Frequency Of Headache DOEE: Duration Of Each Episode Site of pain:
    - UL: Unilateral

- BL: Bilateral Type of pain:
- TH: Throbbing
- ST: Steady Severity of pain:
- SEV: Severe ▪MOD: Moderate

- Past history:
  - DM: Diabetes Mellitus
  - HTN: Hypertension Menstrual history:
  - N: Normal ▪A: Abnormal Cranial nerves:
  - N: Normal
  - 7: 7th cranial nerve involved Motor system:
  - N: Normal ▪HP: Hemiparesis

- Reflexes:
  - N: Normal ▪A: Abnormal Visual acuity:
  - N: Normal ▪A: Abnormal CT brain:
  - N: Normal
  - ICH: Intra Cerebral Hemorrhage
  - VST: Venous Sinus Thrombosis
  - ACM: Arnold Chiari Malformation
  - SAH: Sub Arachnoid Hemorrhage
  - BA: Brain Abscess MRI Brain:
  - N: Normal
  - VST: Venous Sinus Thrombosis
  - BA: Brain Abscess
  - TBM: Tuberculous Meningitis

- NA: Not Done Diagnosis:
- TTH: Tension Type Headache
- ICH: Intra Cerebral Hemorrhage
- SAH: Sub Arachnoid Hemorrhage
- TBM: Tuberculous Meningitis
- RE: Refractive Error
- ACM: Arnold Chiari Malformation
- HTN: Hypertension
- VST: Venous Sinus Thrombosis

- BA: Brain Abscess