CLINICAL AND RADIOLOGICAL PROFILE OF CHRONIC HEADACHE

submitted to
The Tamil Nadu Dr.M.G.R.Medical University

M.D. DEGREE EXAMINATION
BRANCH – I (GENERAL MEDICINE)

KILPAUK MEDICAL COLLEGE

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI

APRIL 2011

ACKNOWLEDGEMENT

I humbly submit this work to the **Almighty** who has given the health and ability to pass through all the difficulties in the compilation and proclamation of this blue print.

I wish to express my sincere thanks to our Dean, **Dr.V.KANAGASABAI , M.D.,** for permitting me to use the resources of this institution for my study.

I owe special thanks to the superintendent Prof. **Dr.K.THIYAGARAJAN, M.S., MCH, DNB (URO) GOVT ROYAPETTAH HOSPITAL**, for permitting me to use the resources .

I feel indebted to **Prof. Dr. G.RAJENDRAN, M.D.,** Head of the Department, Department of General medicine , for his constant encouragement, innovative ideas , and timely suggestions during my work.

I owe special thanks to the Deputy Superintendent and my Unit Chief, Prof. **Dr. N. GUNASEKARAN M.D.,** Department of General medicine , for his constant support , invaluable suggestions , erudite guidance in my study and for being a source of inspiration in my endeavours.

My sincere thanks to Prof. **Dr. S.BALASUBRAMANIAN, M.D., DM(NEURO)** former chief, Neurology department, Govt. Royapettah Hospital, Chennai, for permitting to carry out my study.

I also express my sincere thanks to our Assistant Professors, **Dr.S.MAYILVAHANAN, M.D., Dr.T.RAVINDRAN, M.D Dip. DIAB., Dr. T. RAMESHKUMAR, M.D DCH Dr.K.MANICKAM M.D., D.C.H** for their support in my study.

I would like to thank our former Assistant Professor **Dr. NASREEN BEGUM M.D.,** for their valuable assistance in my study.

I would like to thank all my colleagues and all staffs of Department of General medicine , Govt. Royapettah hospital, Chennai for their help and encouragement.

I would like to thank the Institutional Ethical committee for approving the study.

Finally I am indebted to my parents and my wife who have been solid pillars of everlasting support and encouragement and for their heartfelt blessings.

CONTENTS

INTRODUCTION

Headache disorders are ranked amongst the ten most disabling conditions in the world by World Health Organisation (WHO) .[1] Headache is one of the most common complaint encountered in general medical practice, accounting for 4% of outpatient physician visits. It has the dubious distinction of being the earliest recognized symptom of a wide spectrum of diseases.[2]

Headache has been referred to in many ancient literatures. The earliest reference dates back to a papyrus (ancient medical text book) as old as 3500 BC in the tomb of Thebes that mention the king in the tomb had suffered all his life of a sickness of half head.

Hippocrates (460-377BC) wrote a description of a disease of the hemicrania that relieved on vomiting.

According to different population based studies , over 70% of the general population experience atleast one headache per year and about 15% consult a physician.[3]

The global prevalence of active headache diseases in the adult population is 46%. About 3% of the world's population is affected by chronic headache that lasts for more than 15 days per month.[1, 4]

While headache has been an unaddressed cause of morbidity around the world, it has remained largely unrecognised in the developing world.[1, 5]

Most clinical and epidemiological studies have originated in developed countries and there is scarce literature to support treatment guidelines or public health interventions to deal with headache in low and middle income countries where 85% of the world's population lives. [1]

Inspite of the fact that headache is one of the most common of medical complaints , most headaches in practice continue to be under diagnosed and under treated. Even today many treating physicians feel that nothing much can be done for headache patients! And since more than 90% of headaches seen in practice are primary headaches , it is to be realised that matters have come a long way in the last decade and there is now enough evidence to prove that primary headaches are a genuine potentially treatable biological problem.

It is therefore important that clinicians do not err in making the right diagnosis and choosing the correct drug options. [6]

This work of dissertation has been done with an aim at documenting the patients presenting with different types of chronic headache, their clinical profile, and diagnostic modalities, at the Neurology department, Govt. Royapettah Hospital, Chennai during a one year period.

AIM OF THE STUDY

1. To classify and to study the various types of Chronic Headache.
2. To study the clinical profile of Chronic Headache.
3. To study the radiological profile of Chronic Headache.

REVIEW OF LITERATURE

Headache is one of the most common types of recurrent pain as well as one of the most frequent symptoms in neurology. Although almost everyone get occasional headaches, there are well defined headache disorders that vary in incidence , prevalence and duration. These disorders are usually divided into two broad categories : primary headache and secondary headache. In secondary disorders, headaches are attributed to another condition , such as brain tumour or head injury ; for the primary disorders the headache is not due to another condition. [7]

CHRONIC HEADACHE

Chronic headache / Chronic daily headache is defined as frequent headache occurring more than 15 days/month for over 3 months including those headaches associated with medication overuse. Such headaches may be primary / secondary. [8,9]

Chronic headache affects up to 4% of adults and between 1% and 2% of middle school children. [10,11] The headache characteristics in patients with chronic headache can vary between patients as well as within individual patient. Their headache may present as severe intermittent migraine attacks , intermittent low severity headaches , continuous headache pains , or as a combination of these headache types. This variation continues to make this condition remarkably challenging to understand and to treat.[10]

In 1994 and 1996, Siberstein and Lipton subdivided Primary CDH of long duration in to

- Transformed migraine
- Chronic tension type headache,
- Hemicrania continua and
- New daily persistent headache. [9,12]

In 2004, the International headache society published criteria for Chronic migraine, hemicrania continua, new daily persistent headache , and for chronic tension type headache. The IHS also published criteria for medication- overuse headache as a type of secondary headache disorder. [13] There are no IHS criteria for transformed migraine. Hence chronic migraine is considered as the subset for transformed migraine. [14]

Primary Chronic Daily Headache (CDH), defined as ≥ 15 headache days / month for > 3 months in the absence of organic pathology . [15] Primary CDH is divided in to shorter duration headaches(< 4 hrs) such as chronic cluster headache , chronic paroxysmal hemicrania and hypnic headache and longer duration headache (> 4 hours) includes Chronic migraine(CM) , Chronic tension type headache (CTTH), New daily persistent headache (NDPH), and Hemicrania continua. Most CDH sufferers in the general population have chronic tension type headache or migrainous headache. [16]

Secondary CDHs occur 15 or more times a month and have some identifiable underlying cause. Secondary cause of frequent headaches include post- traumatic headache, cervical spine disorders , headache associated with vascular disorders , headache associated with non vascular intracranial disorders, and other disorders (i.e., temporo mandibular joint disorder or sinus infection. [8]

Epidemiology

The prevalence rate of CDH in the general population averages 3% - 4% .The prevalence in childhood or adolescence is lower, averaging about 1% - 2%. Prevalence in the elderly is similar to that seen in the general population. [16] The prevalence of CDH with medication overuse was found to increase with age, reaching its maximum during the sixth decade of life. The prevalence of transformed migraine in many studies ranged from 1.0-2.4%. CDH affects more women than men by a factor of roughly 2:1. [14,17]

Currently 40-50% of primary headaches seen in hospital OPD and 60-70% seen in doctors' clinic fall within this category. [18]

CHRONIC DAILY HEADACHE

APPROACH TO DIAGNOSIS

Headache > 15 days/ month for > 3 months

↓

History, Examination, Chest X-ray, Hematology, CT/MRI, CSF study

↓

Rule out secondary CDH → yes → diagnose

↓

No

↓

Primary CDH -Classify

< 4 hours duration	> 4 hours duration
Chronic cluster headache	Chronic migraine
Paroxysmal hemicrania	CTTH
Hypnic headache	NDPH
Idiopathic stabbing headache	Hemicrania continua[8]

There are some modifiable and non-modifiable factors that appears to influence risk of headache progression.

Non modifiable risk factors for CDH:-

- Age and gender

- Socioeconomic status
- Marital status

Modifiable risk factors for CDH:-

- ❖ Obesity
- ❖ Snoring
- ❖ Comorbid pain
- ❖ Head or neck injury
- ❖ Caffeine intake
- ❖ Medication use[16]
- ❖ Stressful Life event[19]

Pathophysiology

Although the source of pain in primary CDH is unknown and may be dependent on the subtype, research findings suggest that the following mechanisms, alone or in combination, contribute to the process.

1. Abnormal excitation of peripheral nociceptive afferent fibers in the meninges;
2. Enhanced responsiveness of trigeminal nucleus caudalis neurons;
3. Decreased pain modulation from higher centers such as the pariaqueductal gray matter;
4. Spontaneous central pain generated by activation of the “on cells” in the medulla;

5. Decreased serotonin levels; and
6. Central sensitization. [20]

Sarchielli and colleagues (2001) reported that the CSF of patients with CDH contained a high level of nerve growth factor and substance P compared with controls. Gallai and associated (2003) demonstrated a significant increase in glutamate and nitrite levels in the CSF of patients with CDH.

The increase in nitrites was accompanied by a rise in cyclic guanosine monophosphate (cGMP). This suggests a central disturbance of the receptors for glutamate, *N*-methyl-D-aspartate (NMDA) receptors. Substance P and calcitonin - gene-related peptide (CGRP) were also elevated in the CSF compared with that of control subjects. Whether these changes indicate a causative role or are a response to chronic pain remains open to conjecture. [21]

For the past 15 years, the first edition of the diagnostic criteria of the International Headache Society (ICHD-1) has been the accepted standard. The second edition of the International classification of Headache Disorders(ICHD-2) reflects our improved understanding of some disorders and the identification of new disorders (Headache Classification Subcommittee of the International Headache Society 2004). Like its predecessor, the ICHD-2 separates headache into primary and secondary disorders. [7]

INTERNATIONAL CLASSIFICATION OF HEADACHE DISORDERS-2 (ICHD-2)

PRIMARY HEADACHES

1 Migraine

1.1 Migraine without aura

1.2 Migraine with aura

1.3 Childhood periodic symptoms that are commonly precursors of migraine

1.4 Retinal migraine

1.5 Complications of migraine

1.5.1 Chronic migraine

1.6 Probable migraine

1.6.1 Probable migraine without aura

1.6.2 Probable migraine with aura

2 Tension Type Headache

2.1 Infrequent episodic Tension Type

2.2 Frequent episodic Tension Type

2.3 Chronic Tension Type

2.3.1 Chronic tension-type associated with pericranial tenderness

2.3.2 Chronic tension-type not associated with pericranial tenderness

2.4 Probable Tension Type

3 Cluster Headache and other trigeminal autonomic cephalalgias

3.1 Cluster Headache

3.1.1 Episodic Cluster Headache

3.1.2 Chronic Cluster Headache

3.2 Paroxysmal hemicrania

3.3 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)

3.4 Probable trigeminal autonomic cephalagia

4 Other Primary Headaches

4.1 Primary stabbing headache

4.2 Primary cough headache

4.3 Primary exertional headache

4.4 Primary headache associated with sexual activity

4.5 Hypnic headache

4.6 Primary thunderclap headache

4.7 Hemicrania continua

4.8 New daily persistent headache

SECONDARY HEADACHES

5 Headache attributed to head and/ or neck trauma

6 Headache attributed to cranial or cervical vascular disorders

7 Headache attributed to non- vascular intracranial disorder

8 Headache attributed to its substance or its withdrawal

9 Headache attributed to infection

10 Headache attributed to disorder of homeostasis

11 Headache or facial attributed to disorder of cranium , neck ,eyes , ears , nose , mouth , sinuses , teeth, mouth , or other facial or cranial structures

12 Headache attributed to psychiatric disorder

13 Cranial neuralgias and central causes of facial pain.[13]

Migraine(1.0)

Migraine is a neurological disorder characterised by episodic attacks of headache and associated symptoms.[7] Migraine is a common and disabling disorder that may be divided into 2 broad groups based on the number of headache days. If attacks occur less than 15 days per month the term episodic migraine is applied; when headaches occur on 15 or more days per month the terms chronic migraine or transformed migraine is used. [22]

Epidemiology:-

Epidemiological studies have documented its prevalence and high socioeconomic and personal impacts. It is now ranked by the World Health Organisation as number 19 among all diseases world wide causing disability. It exerts the greatest impact on quality of life as defined by increased

absenteeism from school or work, and more frequent consultation with physicians. [13]

Migraine with or without aura is remarkably common condition. Migraine incidence is more common in females than males. It usually begins in adolescence. More than 80% the onset is before 30 years of age. Although migraine usually diminishes in severity and frequency with age it may actually worsens in some postmenopausal women and estrogen therapy may increase or paradoxically diminish the incidence of headaches.

Pathophysiology :-

Burstein and colleagues showed that approximately 75% of migraine sufferers develop central sensitization (sensitization of the second order trigeminal neuron, which is clinically manifested by the development of cutaneous allodynia) during the course of a migraine attack. Central sensitization appears to be associated with triptan refractoriness. Central sensitization explains the progression of attacks but also may play a role in the progression of the disease itself. It is suggested that repeated central sensitization episodes are associated with permanent neuronal damage, preventive treatment refractoriness, and disease progression. [23,24,25]

1. Central sensitization of pain-procession neurons in the central nervous system by repeated activation of trigeminal nociceptors

2. Depletion of serotonin and noradrenaline in the endogenous pain-control system, permitting unrestrained inflow of afferent impulses.[20,26]

In the study by Yarnitsky and colleagues (2003), blockade of parasympathetic outflow to cerebral and meningeal blood vessels by intranasal lidocaine eased the pain of migraine headache in some patients, whereas allodynia continue to worsen. This was interpreted as indicating that the inflow of nociceptive impulses from dilated vessels in the brain and dura mater during migraine headache had sensitized second-order neurons and then third-order neurons to cause allodynia.[27]

There is considerable overlap in mechanisms mediating migraine headache pain and sustained opioid-induced paradoxical pain. One change that occurs following chronic morphine is increased expression of calcitonin gene-related peptide (CGRP) in primary afferent neurons. Central changes induced by chronic morphine use include increased descending facilitation from the rostral ventromedial medulla and increased excitatory neurotransmission at the level of the dorsal horn. During migraine, CGRP release from primary afferent neurons innervation the intracranial vasculature contributes to neurogenic vasodilatation, and increased excitability of dorsal horn neurons is evident. Descending facilitation from

the rostral ventromedial medulla may contribute to this increased excitability and hypersensitivity.[28]

Using high resolution magnetic resonance imaging (MRI) techniques, abnormally high levels of non-heme iron were detected in the midbrain periaqueductal gray (PAG) of patients with episodic migraine between attacks, as well as in CDH patients who were over using acute medications. Iron levels increased with the duration of illness.[29]

Migraine is divided into six major categories, namely

(i) Migraine without aura(1.1)

(ii) Migraine with aura(1.2)

(iii) Childhood peridodic symptoms that are commonly precursors of migraine(1.3)

(iv) Retinal migraine(1.4)

(v) Complications of migraine(1.5)

Chronic migraine (1.5.1)

(vi) Probable migraine.[13]

1.1 Migraine without aura

Previously used terms:

Common migraine, hemicrania simplex

Description

Recurrent headache disorder manifesting in attacks lasting 4-72 hours. Typical characteristics of the headache are unilateral location , pulsating quality , moderate or severe intensity , aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia.

ICHD-2 diagnostic criteria for migraine without aura(1.1)

A. Atleast 5 attacks fulfilling criteria B-D

B. Headache attacks lasting 4 -72 hours(untreated or unsuccessfully treated)

C. Headache has atleast two of the following characteristics

   a. Unilateral location

   b. Pulsating quality(Throbbing or varying with the heartbeat at rest or with movement)

   c. Moderate or severe pain intensity

   d. Aggravation by or causing avoidance of routine physical activity (i.e. walking or climbing stairs)

D. During headache atleast one of the following

   a. Nausea and / or vomiting

   b. Photophobia and Phonophobia

E. Not attributed to another disorder

Migraine without aura is the commonest subtype of migraine. It has a higher average attack frequency and is usually more disabling than 1.2 Migraine with aura.[13, 30, 31, 32]

1.2 Migraine with aura

Previously used terms

Classical migraine , ophthalmic , hemiparesthetic , hemiplegic or aphasic migraine , complicated migraine

Description

Recurrent disorder manifesting in attacks of reversible focal neurological symptoms that usually develop gradually over 5 – 20 minutes and last for less than 60 minutes. Headache with the features " migraine without aura" usually follows the aura symptoms. Less commonly , headache lacks migrainous feature or its completely absent (i.e., the aura may occur without any subsequent sequelae)

Diagnostic criteria

A. Atleast 2 attacks fulfilling criteria B-E

B. Fully reversible visual and/or sensory and / or speech symptoms but no motor weakness

C. Atleast two of the following

1. Homonymous visual symptoms including positive features(i.e flickering lights, spots, lines) and/or negative features (i.e. loss of vision) and/or unilateral sensory symptoms including positive features(i.e. pins and needles) and / or negative features(i.e. numbness)

2. Atleast one symptom develop gradually over ≥ 5 min and / or different symptoms occur in succession

3. Each symptom lasts ≥ 5 min and ≤ 60 min

D. Headache meets criteria B-D for migraine without aura (1.1) begins during the aura or follows aura within 60 min

E. Not attributed to another disorder.[13, 33]

1.5.1 Chronic migraine:-

Chronic migraine is defined as the presence of headache meets criteria for migraine without aura for at least 15 days/ month for greater than 3 months.

Chronic migraine is now recognised as one of the 4 types of primary chronic daily headache of long duration , a syndrome defined by primary headaches 15 or more days per month for at least 3 months with attacks that last 4 hours or more per day on average. [34, 35]

Diagnostic Criteria:-

A. Headache fulfilling criteria C & D for 1.1 migraine without aura on >15 days > 3 months

B. Not attributable to another disorder.[9, 13, 19]

1.6.5 Probable chronic migraine:-

Criteria:-

A. Headache fulfilling criteria C and D for migraine without aura on > 15 days/month for > 3 months.

B. Not attributed to another disorder but with in the last 2 months medication overuse should be present but should not fulfil the criteria for medication overuse headache. [13]

TREATMENT STRATEGIES FOR CHRONIC MIGRAINE

One of the most important benefits to a workable definition for CM is the ability for these patients to have a defined set of inclusion criteria for their diagnosis, making possible the standardization of clinical trials aimed at the treatment of CM. [36]

Nonpharmacological measures

Patients should be encouraged to eat, sleep, and exercise in a regular pattern. Caffeine consumption should be limited. Co morbid depression and anxiety must be addressed. Behavioural therapy (eg, biofeedback, relaxation, cognitive behavioural therapy) is effective for managing primary headaches such as migraine and also may help in the management of CM. Patients who had received biofeedback-assisted relaxation in addition to pharmacological therapy had greater sustained improvement on two out of three outcome measures (ie, fewer headache days and reduced intake of analgesic medications) and fewer relapses. [36]

Pharmacologic management

1 Amitriptyline

2 Fluoxetine

3 Tizanidine

4 Gabapentin

5 Topiramate

6 Botulinum Toxin Type A

7 Divalproex Sodium

Procedural and surgical options

1 Greater occipital nerve blockade

2 Occipital nerve stimulation

3 Vagal nerve stimulation [36]

TENSION-TYPE HEADACHE (TTH) (2.0)

Tension-type headache is the most prevalent form of primary headache . For the individual sufferer, the tension-type headache is less disabling than migraine or cluster headache, but from a social perspective it is important because of its high prevalence.

Epidemiology

TTH varies considerably in frequency and duration, from rare , short lasting episodes of discomfort to frequent, long-lasting , or even continuous disabling headaches. Its lifetime prevalence in the general population ranges in different studies from 30 to 78%.

Chronic tension headache is a serious disease causing decreased quality of life and high disability, high personal and socioeconomic costs[37, 38]

Pathophysiology:-

Srikiatkhachorn and Anthony (1996) demonstrated that the blood-platelet serotonin level was low in patients with chronic tension-type

headache treated with analgesics. This may reflect depletion in central serotonergic pathways known to be important in pain control. [26]

It recently was demonstrated that depression increases vulnerability to TTH in patients who have frequent headaches during and after a laboratory stress test and that the induced headache was associated with elevated pericranial muscle tenderness. [39]

Pericranial myofascial tissues are considerably more tender in patients who have TTH than in healthy subjects, and that the tenderness is positively associated with the intensity and the frequency of TTH [40]

The increased myofascial pain sensitivity in TTH could be the result of release of inflammatory mediators resulting in excitation and sensitization of peripheral sensory afferents.

The increased myofascial pain sensitivity in TTH also could be caused by central factors, such as sensitization of second-order neurons at the level of the spinal dorsal horn/trigeminal nucleus, sensitization of supraspinal neurons, and increased antinoceceptive activity from supraspinal structures. [41]

ICHD-2 distinguishes three forms of tension-type headache:

1 Infrequent episodic (2.1, < 1 attack per month)

2 Frequent episodic (2.2 , 1- 14 attacks per month

3 Chronic (2.3 , > 15 attacks per month

Each of these forms can be further classified into forms associated or not associated with pericranial tenderness

ICHD -2 DIAGNOSTIC CRITERIA FOR TENSION TYPE HEADACHE

A. Atleast 10 episodes fulfilling criteria B-E; headache < 1 day / month (episodic infrequent) , 1-14 days / month (episodic frequent) , or ≥ 15 days / month for **> 3 months (chronic).**

B. Headache lasting from 30 min to 7 days

C. Atleast two of the following pain characteristics

1. Pressing or tightening (non- pulsating) quality
2. Mild or moderate intensity
3. Bilateral location.
4. No aggravation by walking stairs or similar routine physical activity

D. Both of the following

- Neither moderate or severe nausea nor vomiting.
- Photophobia or phonophobia are absent, or one but not the other may be present

E. Not attributed to another disorder

2.X.1. Associated with pericranial tenderness [42]

A. Fulfills criteria for 2.X

B. Increased tenderness on pericranial manual palpation

2.X.2. Not associated with pericranial tenderness

A. Fulfills criteria for 2.X

B. Not associated with pericranial tenderness

X→ replaced by corresponding digit of infrequent(1), frequent episodic (2), or chronic.[13]

Management:-

A correct diagnosis should be assured by means of a headache diary recorded over at least 4 weeks.

The diary also may reveal triggers and medication overuse, and it establishes a baseline against which to measure the efficacy of treatments.

Significant comorbitities (eg. anxiety or depression) should be identified and treated concomitantly.

Nonpharmacologic management should be considered for all patients who have TTH and is used widely. [43]

Non – pharmocological measures

Improvement of posture, relaxation, exercise programs, hot and cold packs, ultrasound, and electrical stimulation. Various techniques, such as massage, relaxation, and home-based exercises, and found a modest effect. [43, 44]

Psychologic treatment strategies have reasonable scientific support for effectiveness. Relaxation training is a self-regulation strategy that provides patients with the ability to consciously reduce muscle tension.[45]

Pharmacological therapy:-

Acute pharmacologic therapy refers to the treatment of individual attacks of headache in patients who have episodic and chronic TTH drugs such as aspirin, acetaminophen, ibuprofen, naproxen sodium, Ketoprofen, diclofenac potassium. [43]

The combination of analgesics with caffeine, codeine, sedatives, or tranquilizers frequently is used and increased efficacy when adding caffeine to aspirin or ibuprofen has been reported. [46]

Prophylactic pharmacotherapy should be considered in patients who have chronic TTH who do not respond to nonpharmacologic treatment. Drug such as amitriptyline, maprotiline and mianserin, are reported as more effective.[47]

MIXED TENSION MIGRAINE HEADACHE

A mixed tension migraine is a chronic daily tension headache that occurs in addition to periodic migraine headaches. Some people get tension headaches daily in addition to occasional migraines. This is known as a mixed tension headache. It is very common to have both tension and migraine headaches. [48]

The exact reason people get either of these headaches is not known. Stress, fatigue, anxiety, or depression generally cause tension headaches. Migraines are thought to be due to spasms of blood vessels inside the skull. Bright light, noise, tension, or certain things in the diet, such as alcohol or caffeine, often bring them on. There seems to be an inherited risk of migraines in many people. People who have both types of headaches usually have depression that contributes to the headaches. [48]

Treatment & Monitoring

Treatment begins with a limitation on pain medications. This may cure the daily headaches completely. If this doesn't work, a person is usually started on other treatments. These may include behavioral and drug therapy. Stress-reduction methods may be effective. These methods include relaxation techniques, such as meditation. Biofeedback may also be useful. This involves the use of special machines to help a person reduce muscle tension. The machine may make a sound every time the muscles get tense, making someone aware of when they are getting tense. The person can then learn to reduce muscle tension, which makes the sound disappear. Eventually, people no longer need the machine to help them relax their muscles. [48]

A headache diary or journal may help identify headache triggers. Counselling and therapy may also help a person deal with depression, tension, or anxiety.

Medicines may be needed to treat depression. Other medicines may be given for the migraine headaches. Nonsteroidal anti-inflammatory drugs (NSAIDs), such as naproxen or ibuprofen, may be given for daily tension headaches.

Long-term treatment involving medicine and behavior methods may be needed. The goal of treatment is to reduce the number and severity of headaches as much as possible. [48]

3.1 CLUSTER HEADACHE

Previously used terms :

Ciliary neuralgia , erythromelalgia of the head, hemicrania neuralgiformis chronica , histaminic cephalagia , Horton's headache , Harris – Horton's disease , migrainous neuralgia , petrosal neuralgia. [13]

Description

Cluster headache is a form of intermittent, short lived, excruciating, unilateral head pain associated with autonomic dysfunction.

Prevalence estimates range from 0.1% to 0.4%.[49] for unknown reasons prevalence is 3- 4 times higher in men than in women. Recent evidence however suggests a progressively decreasing male preponderance. [50] Age of

onset is usually 20 -40 years. Cluster may be inherited (autosomal dominant) in about 5% of cases. [13]

Cluster headache is a severe , recurring , chronic disease . Attacks usually occurs in series (cluster periods) lasting for weeks or months separated by remission periods usually lasting months or years . However about 10 -15% of patients have chronic symptoms without remissions. [13]

The pain of cluster headache is described as sharp , stabbing, boring , drilling , piercing but generally not throbbing like migraine. The duration of pain is usually ranges from 15 to 180 minutes. During the attack , patients find it difficult to lie down as it aggravates pain. [51]

Cluster headache is divided into two forms

3.1.1 Episodic Cluster headache

-The term episodic cluster headache indicates remission in between in between attacks lasting for one month or more

3.1.2 Chonic Cluster headache [13, 52]

3.1.2 CHRONIC CLUSTER HEADACHE

Cluster headache attacks occurring for more than year without remission or with remissions lasting less than 1 month. [13]

The Chronic form of the disease can evolve from the episodic form (secondary chronic form), or it may develop de novo as primary chronic cluster headache. Chronic Cluster headache occurs in approximately in 15%

of sufferers, is unremitting from onset (primary chronic cluster) in 10%, and evolves from the episodic form in 5%. [13, 52]

ICHD -2 DIAGNOSTIC CRITERIA FOR CHRONIC CLUSTER HEADACHE

A. Atleast 5 attacks fulfilling criteria B-D

B. Severe or very severe unilateral orbital, supraorbital and/or temporal pain lasting 15-180 minutes if untreated

C. Headache is accompanied by atleast one of the following

1. Ipsilateral conjuctival injection and or lacrimation
2. Ipsilateral nasal congestion and/or rhinnorhoea]
3. Ipsilateral eyelid oedema
4. Ipsilateral forehead and facial sweating
5. Ipsilateral miosis and/or ptosis
6. A sense of restlessness or agitation

D. Attacks have a frequency from one every other day to 8 per day

E. Not attributed to another disorder

F. Attacks recur over > than 1 year without remission or with remissions lasting less than 1 month. [13, 52]

TREATMENT

Cluster headache treatment can be divide into three classes.

Abortive Therapy

1 Sumatriptan injection or nasal spray (>90% effective)
2 Oxygen
3 Dihydroergotamine
4 Ergotamine
5 Zolmitriptan
6 Intranasal lidocaine
7 Greater occipital nerve blockade

Transitional therapy

1. Corticosteroids
2. Naratriptan
3. Dihydroergotamine
4. Occipital nerve blockade

Preventive therapy

1. Verapamil
2. Lithium carbonate
3. Divalproex sodium
4. Topiramate
5. Melatonin[53]

Surgical Treatment of Cluster Headache

Surgical treatment of cluster headache should only be considered after a patient has exhausted all medical options

1. Surgery on the Cranial Parasympathetic System
2. Surgery on the Sensory Trigeminal Nerve
3. Radiofrequency thermocoagulation

New Strategies

1. Hypothalamic stimulation
2. Greater occipital nerve stimulation. [53]

4.8 NEW DAILY PERSISTENT HEADACHE

NDPH , while not as common as chronic migraine or chronic tension type headache , is common enough to be part of the general neurologist's practice. First described in 1986, it gained acceptance when it was placed within the Silberstein –Lipton criteria for CDH in 1994. The critical feature of the original description and subsequent S-L description was the onset : daily headache starts abruptly without a background of frequent or worsening headache. [54]

Previously used terms

De novo chronic headache , chronic headache with acute onset.

Description

Daily or near-daily primary headache that begins without a history of evolution from episodic headache and then persists is traditionally classified as new daily persistent headache

Headache that is daily and unremitting from very soon after onset (within 3 days at most). The pain is typically bilateral, pressing or tightening in quality and of mild to moderate intensity. There may be photophobia, phonophobia or mild nausea.

NDPH has been recognised by the second edition of IHS classification as a separate entity from chronic tension type headache. [13]

The prevalence of NDPH is approximately 0.1% of the general population.In speciality headache clinics , approximately 10% of patients who have Chronic daily headache meet the criteria for NDPH. NDPH has a female preponderance (2.5 :1) . Diagnosed at all ages , NDPH is usually begins in the second and third decade in women and fifth decade in men. Typically , patients are able to pin point the exact date their headache started. In atleast half the cases , headache begins in relation to an infection or flu like illness or stressful life event. [55]

Pathophysiology

The Pathophysiology of NDPH is unknown. Because of the abrupt nature of onset and a reputation for being difficult to treat pharmacologically , secondary etiologies always need to be sought in a patient with suspected NDPH. Work up may include a gadolinium enhanced MRI and / magnetic resonance venography of the head and possibly spinal fluid analysis.

Proposed etiologies are heterogenous and have revolved around various associated infections (Epstein Barr Virus , Salmonella, adenovirus , toxoplasmosis, varicella –zoster) although a minority of patients suffer a stressful life event prior to onset. The immune response to these infectious agents may induce an autoimmune cycle that maintains neurogenic

inflammation , but this is far from proven and upto 40% have no recognised trigger. [56]

Li and Rozen 2002 reported that 30% of 56 patients with NDPH started with an infection or flu like illness. 12% started after surgery , and 12 % after a stressful life event. Whether lingering infection can be responsible for daily headaches lasting for months or years remains to be determined. In Li and Rozen's series , 40 were men and 16 were women , ranging in age from 12 to 78 years. The headache was bilateral in 64% , associated with nausea in 68% and associated with photophobia in 66%.

MRI and CT scanning in 49 patients showed no significant abnormality. [57]

ICHD -2 DIAGNOSTIC CRITERIA FOR NEW DAILY PERSISTENT HEADACHE

A. Headache for > 3 months fulfilling criteria B-D
B. Headache is daily and unremitting from onset or from < 3 days from onset
C. Atleast two of the following pain characteristics:
    1. Bilateral location
    2. Pressing/tightening (non-pulsating) quality
    3. Mild or moderate intensity
    4. Not aggravated by routine physical activity such as walking or climbing stairs

D. Both of the following

1. No more than one of photophobia, phonophobia or mild nausea
2. Neither moderate or severe nausea nor vomiting

E. Not attributed to another disorder. [13]

Management

NDPH is difficult to manage. These patients commonly receive preventive medications used to treat migraine such as beta blockers, topiramate, valproic acid , or gabapentin , with very low efficacy. Tricyclics , SSRI and muscle relaxants are usually inefficacious . [55]

SECONDARY HEADACHES

5 HEAD ATTRIBUTED TO HEAD AND / OR NECK TRAUMA

Post Traumatic Headache:-

Headache as a result of head trauma are one of the most common secondary headaches. Headaches are estimated as occurring variably in 30 to 90% of persons who are symptomatic after mild head injury. [58]

According to the IHS, the onset of the headache should be less than 7 days after the injury. [13,58]

Epidemiology

The annual incidence of head trauma is about 200/100000 people in the united states but more than double that number in other parts of the world. [59,60] 75% of the cases of brain trauma are classified as mild and the annual

incidence ranges from approximately 100 to 500 per 100000 people. In post-traumatic headache tension type headache is the most common types. Moderate and severe injuries often cause structural lesions , example- cerebral laceration or intracranial hemorrhage. Microscopic changes are axonal degeneration or alteration in cerebral metabolism. [59]

Pathophysiology and pathogenesis:-

Pain immediately following head trauma may be generated via pain nerve terminals in stretched or otherwise traumatized muscles, ligaments, and cervical nerve roots, and perhaps from sympathetic nerve fibres on arteries accompanying cervical nerve roots. The convergence of nerve pathways from upper cervical roots and the spinal N of trigeminal nerve in the upper brainstem explains pain radiation from the neck to the frontal areas of the head and vice-versa.The evolution of acute post-traumatic headache to the chronic form may be associated with central pain sensitization. [59, 61]

Regional cerebral blood flow studies (rCBF) have shown abnormal asymmetrical rCBF and focal zones of decreased perfusion in people with chronic traumatic headache. In PET scan metabolic rate of glucose utilisation was decreased. [59, 62]

Clinical features:-

Post concussion syndrome:-

1) Trigger an attack of migraine with aura.

2) Blurred vision, photophobia, tinnitus.

3) Depression , anxiety and personality changes are common. [63]

4) Apathy, insomnia and impaired libido.

5) Forgetfulness & learning difficulties. [59]

Diagnostic Criteria for Chronic post traumatic headache attributable to mild head injury

A. Headche , no typical characteristics known, fulfilling criteria C and D

B. Head trauma with all the following:

C. Headache develops within 7 days after head trauma.

1. Either no loss of consciousness, or loss of consciousness of < 30 min duration
2. Glasgow coma scale > 13
3. Symptoms and/or signs diagnostic of concussion.

D. Headache persists for >3 months after head trauma[13, 59]

Diagnostic Criteria for Chronic post traumatic headache attributable to moderate head injury

Criteria:-

A Headache, no typical characteristics known, fulfilling criteria C and D

B. Head trauma with at least one of the following:

1. loss of consciousness for > 30 min traumatic brain lesions
2. Glasgow scale <13

3. Post-traumatic amnesia >48 hours
4. Imaging demonstration of a traumatic brain lesion

C. Headache develops with in 7 days after head trauma or after regaining consciousness following head trauma.

D. Headache persists for > 3 months after head trauma[13, 59]

Treatment:-

Tricyclic antidepressants, Antiepileptic (divalproex, gabapentin).

Lifestyle modifications:-

Exercise and relaxation

Diet and avoidance of other headache triggers.

Other used therapies:-

Biofeedback, Physical therapy (massage, heat,cold) , acupuncture.

Transcutaneous electrical stimulation.

Trigger point injections and nerve blocks. [59]

6 HEADACHE ATTRIBUTED TO CRANIAL OR CERVICAL VASCULAR DISORDERS

Headache attributed to ischemic stroke:-

Headache accompanies ischemic stroke in 17 – 34% of cases; it is more frequent in basilar than in carotid –territory strokes.

The headache of ischemic stroke is accompanied by focal neurological signs and/ or alterations in consciousness usually allowing easy

differentiation from the primary headaches. It is usually of moderate intensity and no specific characteristics.[13]

With cerebral infarction , headache tends to be more common with stroke in areas supplied by the posterior circulation . when present the headache tends to be non pulsatile and ipsilateral to the involved hemisphere.

Pathophysiology:-

1. Dilatation of collaterals supplying the ischemic region
2. Distension of occluded parent vessel .
3. Ischemia of arterial muscle
4. Acute Hypertension
5. Stroke- associated edema
6. Platelet aggregation & release of serotonin, alteration in neurotransmitter. [64,65]

Diagnostic Criteria:-

A. Any new headache fulfilling criteria C
B. Neurological signs and/or neuroimaging evidence of a recent ischemic stroke
C. Headache develops simultaneously with or in very temporal relation to signs or other evidence of ischemic stroke. [13]

Treatment:-

Analgesics & Supportive management.

7 HEADACHE ATTRIBUTED TO NON VASCULAR INTRACRANIAL DISORDER

7.1.1 Idiopathic intracranial hypertension

Previously used terms

Benign intracranial hypertension , Pseudotumour cerebri , Meningeal hydrops , Serous meningitis. [13]

Description

The term *pseudotumor cerebri* was coined by Nonne in 1914 and has remained a useful means of designating a common and highly characteristic syndrome of headache, papilledema (unilateral or bilateral), minimal or absent focal neurologic signs, and normal CSF composition, all occurring in the absence of enlarged ventricles or an intracranial mass on CT scan or MRI.

Pathophysiology

The mechanism of increased CSF pressure in the above-described idiopathic form of the disorder has remained elusive, but recent experience is converging on the possibility that there is a functional obstruction to outflow in the venous sinuses. Karahalios and colleagues and others have found the cerebral venous pressure to be consistently elevated in pseudotumor cerebri; in half of their patients, there was a venous outflow obstruction demonstrated by venography, often with a pressure gradient across the site.

Symptomatic Causes of Pseudotumor Cerebri

The main considerations in cases of generalized elevation of ICP and papilledema in the absence of an intracerebral mass are covert occlusion of

the dural venous sinuses, gliomatosis cerebri, occult arteriovenous malformation, and carcinomatous, infectious, or granulomatous meningitis. In addition to mechanical factors, a number of toxic and metabolic disturbances may give rise to a pseudotumor syndrome. In children, as chronic corticosteroid therapy is withdrawn, there may be a period of headache, papilledema, and elevated ICP with little or no enlargement of the lateral ventricles. Lead toxicity in children may be marked by brain swelling and papilledema. Excessive doses of tetracycline and vitamin A (particularly in the form of isotretinoin, an oral vitamin A derivative used in the treatment of severe acne) have also been shown to cause intracranial hypertension in children and adolescents. [66]

Diagnostic criteria :

A.Progressive headache with atleast one of the following characteristiiteria and fulfilling criteria C and D

1. Daily occurrence
2. Diffuse and / or constant (non- pulsating) pain
3. Aggravated by coughing or straining

B.Intracranial hypertension fulfilling the following criteria

1. Alert patient with neurological examination that is either normal or demonstration of any of the following abnormalities:
   a) Papilledema

b) Enlarged blind spot

c) Visual field defect

d) Sixth nerve palsy.

2. Increased CSF pressure

3. Normal CSF chemistry and cellularity

4. Intracranial diseases ruled out by appropriate investigations

5. No metabolic, toxic or hormonal cause of intracranial hypertension.

C. Headache develops in close relation to increased intracranial pressure.

D. Headache improves after withdrawal of CSF to reduce pressure to 120-170mm H2O and resolves within 72 hours of persistent normalisation of intracranial pressure. [13]

Treatment

Medical Management

- Diet and weight loss
- Diuretics(Acetozolamide , frusemide) [67,68]
- Corticosteroids[69]
- Tricyclic antidepressants
- Sodium valproate

Surgical treatment

- Optic nerve sheath decompression[70]
- CSF shunting[67]

7.4 HEADACHE ATTRIBUTED TO INTRACRANIAL NEOPLASM

Headache is said to be a significant symptom in about two-thirds of all patients with brain tumor, but it is infrequent , particularly as the heralding symptom of a tumor in an adult.

The pain has no specific features; it tends to be deep-seated, usually non throbbing (occasionally throbbing), and is described as aching or bursting. Attacks of pain last a few minutes to an hour or more and occur once or many times during the day. Physical activity and changes in position of the head may provoke pain, whereas rest diminishes its frequency. Nocturnal awakening because of pain occurs in only a small proportion of patients and is by no means diagnostic. Unexpected forceful (projectile) vomiting may punctuate the illness in its later stages. If unilateral, the headache is nearly always on the same side as the tumor. [71]

Diagnostic Criteria:-

A. Headache with at least one of the following characteristics and fulfilling criteria C and D
    - ❖ Progressive
    - ❖ Localised
    - ❖ Worse in the morning
    - ❖ Aggravated by coughing or bending forwards.
B. Intracranial neoplasm shown by imaging
C. Headache develops in temporal relation to the neoplasm

D. Headache resolves within 7 days after surgical removal or volume reduction of neoplasm or treatment with corticosteroids. [13]

Treatment:-

Treatment depends on the type of tumor, patient functional status and stage of disease. Patients who have metastatic brain tumors have a limited life expectancy and treatment is palliative. Corticosteroids such as dexamethasone often provide dramatic relief of headache and other symptoms caused by cerebral edema,.[72]

Whole brain radiotherapy often effectively controls symptoms. [73] In selected patients surgical resection or stereotactic radiosurgery may provide more durable symptom control. [74] High-grade gliomas are the most common primary brain tumors and definitive treatment with surgery and radiotherapy usually results in headache remission. [72]

8 HEADACHE ATTRIBUTED TO ITS SUBSTANCE OR ITS WITHDRAWAL

8.2 MEDICATION OVERUSE HEADACHE

Headaches can be an effect of acute or chronic medication exposure or be caused by withdrawal from a substance.

A headache is attributed to a substance if a new kind of headache occurs for the first time in close temporal relation to exposure to the substance, regardless of the characteristics of the headache , even it

resembles a primary headache such as migraine , tension-type headache or cluster headache. [75]

Patients who have frequent headaches often overuse analgesics, opiods, ergotamine , or triptans. This medication overuse can produce chronic daily headache (CDH)(Drug- induced rebound headache) , which is accompanied by dependence on symptomatic medication and refractoriness to prophylactic medication. [75,76,77] Stopping the symptomatic medication may result in withdrawal symptoms , a period of increased headache and then improvement. [78]

The diagnosis of medication overuse headache is extremely important clinically because patients usually do not respond to preventive medications while overusing drugs. [75]

The new IHS classification has several types of medication overuse headache:

Ergotamine overuse headache, Opiod overuse headache, Combination drug medication overuse headache , other substance overuse headache , and headache as a side effect of drugs used for other indication. [13,75]

Criteria for medication overuse headache

At least one of the following for at least one month

A. Simple analgesic use (> 1000 mg ASA/ acetaminophen) > 5 d/week

B. Combination analgesics (caffeine, barbituarate) (>3 tablets/day) > 3 d/wk

C. Opiods(>1 tablet/day) > 2d/wk

D. Ergotamine use (1mg po or 0.5 mg pr) > 2 days/wk.

Diagnostic criteria for Analgesic overuse headache.

A. Headache present on >15 days/month with at least one of the following characteristics and fulfilling criteria C and D.

- Bilateral
- Pressing/ tightening(non-pulsating) quality
- Mild or moderate intensity.

B. Intake of simple analgesics on > 15 days/month for > 3 months

C. Headache has developed or markedly worsened during analgesic overuse

D. Headache resolves or reverts to its previous patterns within 2 months after discontinuation of analgesics[13]

Treatment:-

Pharmacological:-

Outpatient Management:-

1) Substitute a long acting NSAID and taper the overused medication

2) Abruptly discontinuing the overused headache and substitute transitional medicine such as NSAID, DHE, or corticosteroid.

Inpatient Management:-

1 Detoxification

2 Rehydration

3 Pain control with parenteral therapy

4 Establishing effective prophylaxis

5 Interrupting the cycle of pain

6 Patient education

7 Establishing outpatients methods of pain control. [75,79]

Diet and lifestyle changes.

Physical therapy and exercise. [75]

11 HEADACHE OR FACIAL ATTRIBUTED TO DISORDER OF CRANIUM , NECK ,EYES , EARS , NOSE , MOUTH , SINUSES , TEETH, MOUTH , OR OTHER FACIAL OR CRANIAL STRUCTURES

11.8 POST SINUSITIS HEADACHE:-

Sinusitis is over diagnosed as a cause of headache because of the belief that pain over the sinuses must be related to the sinuses. In fact frontal headache is usually caused by migraine or tension headache. If patients does not respond to treatment for migraine and tension headache one should reconsider the diagnosis of sinus disease.

Diagnostic Criteria:-

A. Pain in one or more regions of the head, face, ears or teeth.

B. Clinical, laboratory, or imaging evidence of an acute rhinosinusitis

C. Simultaneous onset of headache and rhinosinusitis

D. Headache lasts < 7 days after

E. Remission or successful treatment of acute rhinosinusitis. [80]

Factors associated:-

Major factors

Facial pain/ pressure

Facial congestion/ fullness

Nasal obstruction/blockage

Nasal discharge

Hyposmia/anosmia

Fever (acute rhinosinusitis)

Purulence in nasal cavity on examination.

Minor Factors

- Headache
- Fever
- Halitosis
- Fatigue
- Dental pain
- Cough
- Ear pain/ pressure/ fullness.

Diagnostic Criteria:- > 2 major factors, or 1 major factor and 2 minor factors, or nasal purulence on examination.

Probable:- 1 major factor or >2 minor factor. [81]

Investigations:-

Anterior Rhinoscopy

CT-PNS [82]

Diagnostic fiberoptic endoscopy.

Treatment:-

Treat the bacterial infection by broad spectrum antibiotic.

Reduce ostial swelling by Nasal decongestants.

Drain the sinuses.

Maintain sinus ostia patency – Corticosteroids. [81]

12 HEADACHE ATTRIBUTED TO PSYCHIATRIC DISORDER

The most common cause of generalized persistent headache, both in adolescents and adults, is depression or anxiety in one of its several forms. The authors have also noted that many seriously ill psychiatric patients complain of frequent headaches that are not typically of the tension type. These patients report unilateral or generalized throbbing cephalic pain lasting for hours every day or two. The nature of these headaches, which in some instances resemble common migraine, is unsettled. Others have delusional symptoms involving physical distortion of cranial structures. As the psychiatric symptoms subside, the headaches usually disappear.

Odd cephalic pains, e.g., a sensation of having a nail driven into the head (*clavus hystericus*), may occur in hysteria and raise perplexing problems in diagnosis. The bizarre character of these pains, their persistence in the face of every known therapy, the absence of other signs of disease, and the presence of other manifestations of hysteria provide the basis for correct diagnosis. Older children and adolescents sometimes have peculiar behavioral reactions to headache: screaming, looking dazed, and clutching the head with an agonized look. Usually, migraine is the underlying disorder,

the additional manifestations responding to therapeutic support and suggestion. [71]

Diagnostic Criteria:-

A Headache , no typical characteristics known , fulfilling criterion C

B Presence of somatisation disorder fulfilling DSM IV criteria

1 History of many physical complaints beginning before age 30 that occur over a period of several years and result in treatment being sought and/or significant impairment in social, occupational or other important areas of functioning

2 At least four pain symptoms , two non-pain gastrointestinal symptoms, one sexual or reproductive symptom and one pseudo neurological symptom

3 After investigations symptoms cannot be fully explained by a general medical condition

C Headache not attributable to another cause. [13]

Treatment:-

Behavioural therapy

Treating underlying psychiatric disorder. [71]

13 CRANIAL NEURALGIAS AND CENTRAL CAUSES OF FACIAL PAIN.

13.1 TRIGEMINAL NEURALGIA

Trigeminal neuralgia is a distinct , painful disorder of the face that easily is evoked by trivial stimuli and undergoes a relapsing , remitting course. [83]

This is a common disorder of middle age and later life, consisting of paroxysms of intense, stabbing pain in the distribution of the mandibular and maxillary divisions (rarely the ophthalmic division) of the fifth cranial nerve. The pain seldom lasts more than a few seconds or a minute or two, but it may be so intense that the patient winces involuntarily; hence the term *tic*. It is uncertain whether the tic is reflexive or quasi-voluntary. The paroxysms recur frequently, both day and night, for several weeks at a time.

Another characteristic feature is the initiation of a jab or a series of jabs of pain by stimulation of certain areas of the face, lips, or gums, as in shaving or brushing the teeth, or by movement of these parts in chewing, talking, or yawning, or even by a breeze—the so-called trigger zones. [71]

Diagnostic Criteria:

1) Paroxysmal attacks affecting one or more divisions of the trigeminal nerve,lasting for a few seconds to less than two minutes.
2) Pain has at least one of the following characteristics: intense , sharp, superficial, stabbing, and precipitated from trigger areas or by trigger factors
3) There is no clinically evident neurological deficit
4) Attacks are stereotyped in the individual patient.
5) Not attributed to another disorder
6) Headache lasts < 3 months after successful treatment of the causative disorder.[13]

Treatment

Anticonvulsant drugs such as phenytoin (300 to 400 mg/day), valproic acid (800 to 1200 mg/day), clonazepam (2 to 6 mg/day), gabapentin (300 to 900 mg/ day), and particularly carbamazepine (600 to 1200 mg/day), alone or in combination, suppress or shorten the duration and severity of the attacks. Carbamazepine is effective in 70 to 80 percent of patients, but half become tolerant over a period of several years.

Baclofen may be useful in patients who cannot tolerate carbamazepine or gabapentin, but it is most effective as an adjunct to one of the anticonvulsant drugs. Capsaicin applied locally to the trigger zones or the topical instillation in the eye of an anaesthetic (proparacaine 0.5%) has been helpful in some patients.

Most of the patients with intractable pain come to surgery or an equivalent form of root destruction. The commonly used procedures are

1) Stereotactically controlled thermocoagulation of the trigeminal roots using a radiofrequency generator or similarly applied focused gamma radiation and
2) The procedure of vascular decompression, popularized by Jannetta, which requires a posterior fossa craniotomy but leaves no sensory loss. Gamma knife radiation is emerging as a less intrusive alternative, but its full effect is not evident for many months. In practice, an

anticonvulsant is often required for some period of time even after any of these procedures, and it must be reinstituted when symptoms reoccur, as they often do in our experience. [71]

HEADACHE AND RADIOLOGY

Headaches are one of the most common symptoms that neurologists evaluate. Although most are caused by primary disorders, the list of differential diagnoses is one of the longest, with over 300 different types and causes. The cause or type of most headaches can be determined by a careful history supplemented by a general and neurologic examination. Reasons for obtaining neuroimaging include medical indications as well as anxiety of patients, families and medico-legal concerns.

COMPUTED TOMOGRAPHY AND MAGNETIC RESONANCE IMAGING

At present the two most widely used imaging modalities in neuro radiology are **CT scan & MRI.** CT is used as the primary imaging modality in any case of headache. If diagnostic dilemma remains or if any surgical treatment is anticipated MRI & other imaging modalities can be used.

CT is better than MRI in detecting calcification in certain lesions like certain tumors, granuloma & parasites. It is also very useful for those patients who cannot tolerate or have contraindications to MRI scanning as in

cases of claustrophobia patients, with metallic bone prosthesis, Intracranial aneurysm clips, pacemakers etc. CT is comparatively cheap and is available in most of tertiary care centre.

The preferred CT technique in Headache is without contrast medium although the latter may be given if initial images suggests an alternative diagnosis. Ideally all patients should be scanned in the first instance with out IV contrast medium so that the decision as to whether contrast medium should be used is based on the clinical & imaging finding in each case.

As routine outpatient neuroimaging became available more than 20 years ago, controversy arose over appropriate selection of patients for computed tomography studies and subsequently magnetic resonance imaging studies. Nowhere is this more apparent than in headache. Because headache is a truly ubiquitous condition with a lifetime prevalence rate reaching 90%, it is clear that it is not possible to scan all patients with headache.

However, patients with new-onset headaches, headaches with a progressive course, headaches with a significant change in pattern, headaches that never alternate sides, and headaches associated with any neurologic findings or seizures have a substantially higher likelihood of a secondary cause such as tumor, arteriovenous malformation, or other structural lesion. In these situations, imaging must be considered as part of the workup.

Combining the results of multiple studies performed reveal the overall percentages of various pathologies as: brain tumors, 0.8%; arteriovenous malformations, 0.2%; hydrocephalus, 0.3%; aneurysm, 0.1%; subdural hematoma, 0.2%; and strokes, including chronic ischemic processes, 1.2% **EEG** is not useful in the routine evaluation

When new-onset headaches begin in patients over the age of 50 years, the physician always should consider whether it may be a secondary headache disorder requiring specific diagnostic testing and treatment. Up to 15% of patients 65 years and over who present to neurologists with new-onset headaches may have serious pathology such as stroke, TIA, neoplasm, and subdural hematoma.

DIFFUSION-PERFUSION IMAGING

White matter abnormalities have been reported on MRI 354 SB Desai studies of patients with all types of migraine, ranging from 12% to 46%. The cause of white matter bnormalities in migraine is not certain. Cerebral atrophy has been reported as more frequent and no more frequent in patients with migraine compared to controls. It has been found that during acute episodes one might have focal areas of ischemia detected by combination of diffusion & perfusion imaging which reverse with time. They may also have small infarcts at the gray-white matter junction. [84]

MATERIALS AND METHODS

STUDY PERIOD

This is an observational study undertaken over period of MAY 2009-APRIL-2010

STUDY PLACE

This study was carried out at the Neurology outpatient department, Govt. Royapettah hospital,Chennai.

ETHICAL CONSIDERATIONS

The study was reviewed and approved by the Institutional Ethical Committee, Kipauk Medical College and General Hospital, Chennai.

STATISTICAL ANALYSIS

Statistical analysis were carried out using statistical package for Social sciences and Epi- software by statistician .

STUDY POPULATION

Observational study which included patients attending neurology outpatient department with complaints of headache for more than three months.

INCLUSION CRITERIA

1) Patients with complaints of Headache for > 3 months.
2) Patients above 12 years of age group.

EXCLUSION CRITERIA

Pregnant mothers were excluded from the study.

100 patients were included in the study after getting written informed consent.

Detailed history was obtained from all patient .The questionnaire recorded the occupation , frequency , intensity, duration , laterality , character of pain, associated symptoms , aggravating factors and family history.

Detailed examination with reference to general condition, refractory error , fundus examination , neck movements and CNS examination.

Specialist opinion in ENT, Ophthalmology and Psychiatry was obtained for all the patients.

All patients of chronic headache were followed up with either CT/MRI after detailed questionnaire to rule out the secondary headache.

International Classification of Headache Disorder II criteria applied to classify headache.

RESULTS

This study was carried out during the period May 2009 to April 2010 in the Department of Neurology, Govt. Royapettah hospital, Chennai . A total of 100 patients were registered in the neurology clinics.

TABLE - 1

DISTRIBUTION OF PATIENTS WITH CHRONIC PRIMARY AND CHRONIC SECONDARY HEADACHE (n=100)

Type of Chronic Headache	Number of patients	Percentage
Chronic Primary headache	82	82
Chronic Secondary headache	18	18
Total	100	100

Chronic Primary headache is the most common type accounting for 82%.

TABLE -2

DISTRIBUTION OF DIFFERENT TYPES OF CHRONIC HEADACHE AMONG THE TARGET POPULATION (n = 100)

Types of chronic headache	No. of Patients	Percentage
Chronic Migraine headache	42	42
Chronic Tension headache	23	23
Probable Chronic Migraine	04	04
Episodic Migraine With aura transition to chronic migraine	03	03
New daily persistent headache	05	05
Chronic Cluster headache	01	01
Mixed Headache (Tension + Migraine)	04	04
Drug overuse headache	05	05
Post sinusitis headache	05	05
Trigeminal neuralgia	03	03
Post traumatic headache	02	02
Psychogenic headache	01	01
SOL Right Cerebral Hemisphere	01	01
Benign intracranial hypertension	01	01
Total	100	100

Chronic Migraine headache is the most common type of chronic headache accounting for 42%

TABLE -3

DISTRIBUTION OF PATIENTS WITH DIFFERENT TYPES OF CHRONIC PRIMARY HEADACHES (n = 82)

Types of Primary headache	ICHD type	No of Patients	Percentage
Migraine and its variants	1	49	60
Chronic Tension headache	2.3	23	28
New daily persistent headache	4.8	5	06
Chronic Cluster headache	3.1.1	1	01
Mixed(Tension+migraine)	-	4	5
Total	-	82	100

Migraine headache is the most common primary headache disorder seen in 49 patients (60%) followed by Chronic tension type seen in 23 patients (28%).

TABLE – 4

TYPES OF MIGRAINE HEADACHE

Types of Migraine headache	No. of Patients	Percentage
Chronic migraine	42	85
Episodic migraine with aura transition to chronic migraine	03	07
Probable Chronic Migraine	04	08
Total	49	100

Among the 49 patients with migrainous headache , 42 patients (85%) presented with Chronic migraine followed by 4 patients (8%) with probable chronic migraine and the remaining 3 patients had transition from migraine with aura to chronic migraine.

TABLE -5

DISTRIBUTION OF CHRONIC TENSION TYPE HEADACHES

Type of CTTH	ICHD-2 type	No of patients	Percentage
CTTH with Pericranial tenderness	2.3.1	02	09
CTTH without Pericranial tenderness	2.3.2	21	91
Total	-	23	100

Of the 23 patients with CTTH, 2 patients presented with pericranial tenderness accounting for 9%.

TABLE -6

DISTRIBUTION OF PATIENTS WITH DIFFERENT TYPES OF SECONDARY HEADACHE (n = 18)

Types of Secondary headache	No of Patients	Percentage
Benign intracranial hypertension	01	06
Drug overuse headache	05	28
Post sinusitis headache	05	28
Post traumatic headache	02	11
Psychogenic headache	01	06
SOL Right Cerebral Hemisphere	01	06
Trigeminal neuralgia	03	17
Total	18	100

Post sinusitis headache and drug overuse headache were more common among the patients with secondary headache.

TABLE 7

SEX DISTRIBUTION OF PATIENTS WITH CHRONIC HEADACHE

Sex	No. of patients	Percentage
Female	66	66 %
Male	34	34%
Total	100	100

Females outnumbered males accounting for 66% of the target population

TABLE 7A

SEX DISTRIBUTION OF PATIENTS WITH PRIMARY AND SECONDARY HEADACHE

Sex	Primary Headache n = 82		Secondary Headache n = 18	
	No. of Patients	%	No. of Patients	%
Male	28	34	06	33
Female	54	66	12	67

There was a female predominance among the patients with primary as well as secondary headache. There was no significant difference in the gender distribution among primary and secondary headache patients.

TABLE 7B

SEX DISTRIBUTION OF PATIENTS WITH PATIENTS WITH PRIMARY HEADACHE

Sex	Migraine headache n = 49		Tension headache n = 23		NDPH n = 5		Mixed headache n = 4	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Male	14	29	08	35	03	60	03	75
Female	35	71	15	65	02	40	01	25

Majority of the patients with migraine and tension type headache were females accounting for 71% and 65% respectively **.**

TABLE 8

AGE DISTRIBUTION IN PATIENTS WITH CHRONIC HEADACHE

Age (in years)	Migraine n = 49		Tension headache n = 23		NDPH n =5		Mixed headache n = 4		Secondary n = 18	
	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
13-20	09	19	03	13	00	00	02	50	02	11
21-30	15	31	09	39	02	40	02	50	08	44
31-40	14	28	08	35	01	20	00	00	03	17
41-50	08	16	03	13	02	40	00	00	02	11
> 51	03	06	00	00	00	00	00	00	03	17

Majority of the chronic headache patients were in the age group of 21-40 years. There was no significant difference in the age group distribution among the patients with different types of chronic headache.

TABLE -9

DISTRIBUTION OF HEADACHE CHARACTER

Headache character	Migraine n = 49		Chronic Tension n = 23		NDPH n =5		Mixed headache n = 4	
	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
Pulsating / Throbbing	49	100	0	0	0	0	02	50
Pressing	0	0	22	96	05	100	01	25
Others	0	0	01	04	0	0	01	25

Majority of the Migraine headache retains its pulsatile /throbbing quality whereas majority of patients with tension and NDPH had headache of pressing quality.

TABLE -10

INTENSITY OF HEADACHE AMONG PATIENTS WITH CHRONIC HEADACHE

Headache Intensity	Migraine N = 49		Chronic Tension n = 23		NDPH n =5		Mixed headache n = 4		Secondary n = 18	
	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
Moderate	01	2%	16	69%	05	100%	01	25%	10	56%
Severe	48	98%	07	31%	00	0%	03	75%	08	44%

Intensity of headache was more severe in majority of patients with migraine (98%) and mixed type of headache (75%). Majority of tension type, NDPH and secondary headache presented with headache of moderate intensity.

TABLE -11

DISTRIBUTION OF SITE OF PAIN IN CHRONIC HEADACHE PATIENTS

Site of Pain	Migraine n = 49		Chronic Tension n = 23		NDPH n =5		Mixed headache n = 4		Secondary n = 18	
	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
Unilateral	24	49	02	9	0	0	0	0	4	22
Bilateral	25	51	21	91	05	100	4	100	14	78

Among the migraine patients, 51% presented with bilateral headache and the remaining 49% with unilateral headache. Majority of patients with tension headache, NDPH and mixed headache presented with bilateral headache accounting for 91%, 100% and 100% respectively. Of the 18 patients with secondary headache, 78% presented with bilateral pain and the remaining 22% with unilateral location.

TABLE 12

DISTRIBUTION OF LOCATION OF HEADACHE IN PATIENTS WITH CHRONIC HEADACHE

Headache location	Migraine n = 49		Chronic Tension n = 23		NDPH n =5		Mixed headache n = 4	
	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
Frontal	9	18	2	8	0	0	1	25
Frontotemporal	27	55	16	70	3	60	1	25
Frontooccipital	6	12	2	9	1	20	0	0
Occipital	7	14	1	4	1	20	1	25
Temporal	0	0	0	0	0	0	0	0
Temporoparietal	0	0	2	9	0	0	1	25

Fronto temporal area is the most common site . Other common locations are frontal and occipital. Least common is temporal side.

TABLE -13
DISTRIBUTION OF NUMBER OF HEADACHE DAYS/MONTH IN PATIENTS WITH DIFFERENT TYPES OF CHRONIC HEADACHE

No of days of Headache/month	Migraine n = 49		Chronic Tension n = 23		NDPH n =5		Mixed headache n = 4		Secondary n = 18	
	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
15-20	43	88	21	91	01	20	3	75	13	72
20-25	5	10	2	9	0	0	1	25	2	11
25-30	1	2	0	0	04	80	0	0	3	17

Majority of chronic headache patients suffers from headache around 15-25 days / month. However in NDPH patients had headache through out the month.

TABLE -14
AGGRAVATING FACTORS IN PATIENTS WITH CHRONIC HEADACHE

Aggravating factors	Migraine n = 49		Chronic Tension n = 23		Post sinusitis headache n= 5		Trigeminal neuralgia n = 3	
	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
Stress	35	71	18	78	0	0	0	0
Noise	8	16	0	0	0	0	0	0
Smell	1	2	0	0	0	0	0	0
Lighting	7	14	0	0	0	0	0	0
Cough& cold	1	2	0	0	5	100	0	0
Washing face	0	0	0	0	0	0	3	100
Chewing	0	0	0	0	0	0	3	100

Stress is the most common aggravating factors in patients with chronic migraine (71%) as well as chronic tension headache (78%). Other aggravating factors such as noise, smell and lighting are commonly associated with migraine headache. Cough and cold are the major aggravating factors in patients with post sinusitis headache (100%) whereas washing face, chewing acted as trigger factors for patients with Trigeminal neuralgia (100%) .

TABLE – 15

ASSOCIATED SYMPTOMS IN CHRONIC HEADACHE PATIENTS

ASSOCIATED SYMPTOMS	Migraine	Tension	Cluster	NDPH	Mixed
Blurring of vision	23	11			2
Photophobia	24	-		1	1
Phonophobia	2	-			
Nausea	29	9		1	
Vomiting	28	5			
Rhinorrhea	-	-	1		
Lacrimation	2	-	1		1
Dizziness	-	-			

Predominant symptoms in migraine are nausea ,vomiting, photophobia, blurring of vision whereas tension headache has blurring of vision, nausea and vomiting in order of frequenc .

TABLE:-16

DURATION OF HEADACHE IN DIFFERENT TYPES OF CHRONIC HEADACHE

Duration of Headache in hours	Migraine n = 49		Chronic Tension n = 23		NDPH n =5		Mixed headache n = 4		Secondary n = 18	
	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
4-8	31	63	19	83	1	20	1	25	11	61
9-16	2	4	1	4	0	0	0	0	1	12
17-24	11	22	3	13	3	60	3	75	5	15
>24	5	11	0	0	1	20	0	0	1	1

The most of the migraine and tension headache patients presented with headache for a duration of 4-8 hours . However majority of NDPH and mixed headache persisted for 18-24 hours .

TABLE 17

CT FINDINGS IN PATIENTS WITH PRIMARY HEADACHE

CT in Primary headache	Migraine n = 49		Chronic Tension n = 23		NDPH n =5		Mixed headache n = 4	
	No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
Normal	45	92	20	87	5	100	3	75
Calcified granuloma	2	4	0	0	0	0	0	0
Calcification	1	2	2	9	0	0	1	25
Infarct	1	2	0	0	0	0	0	0
Age related atrophy	0	0	1	4	0	0	0	0

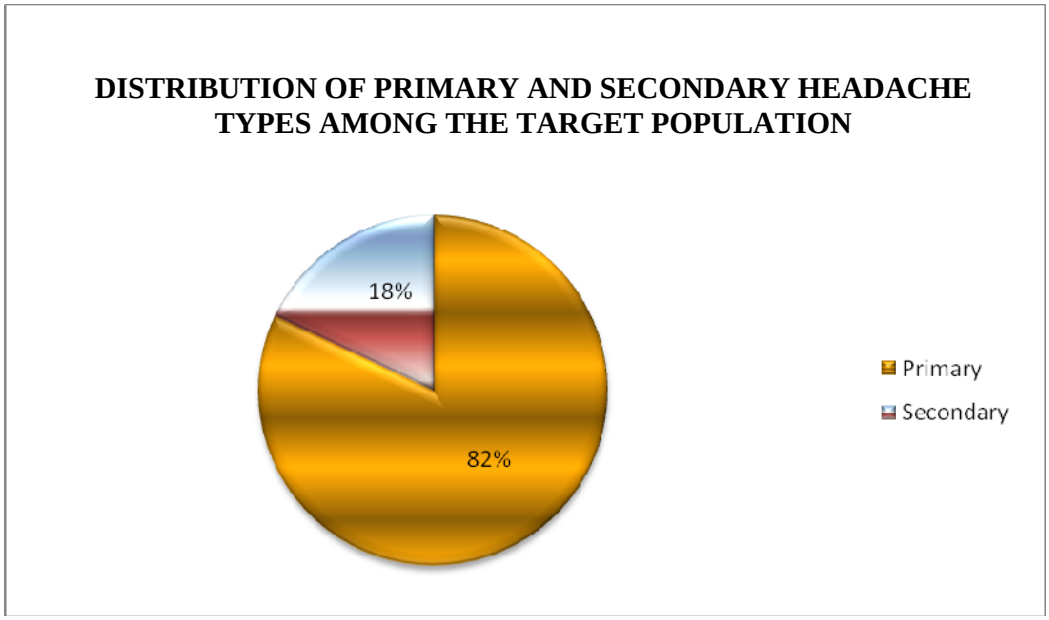
CT Brain of majority of primary headache patients showed normal study. In few patients CT is associated with calcified granuloma which is insignificant.

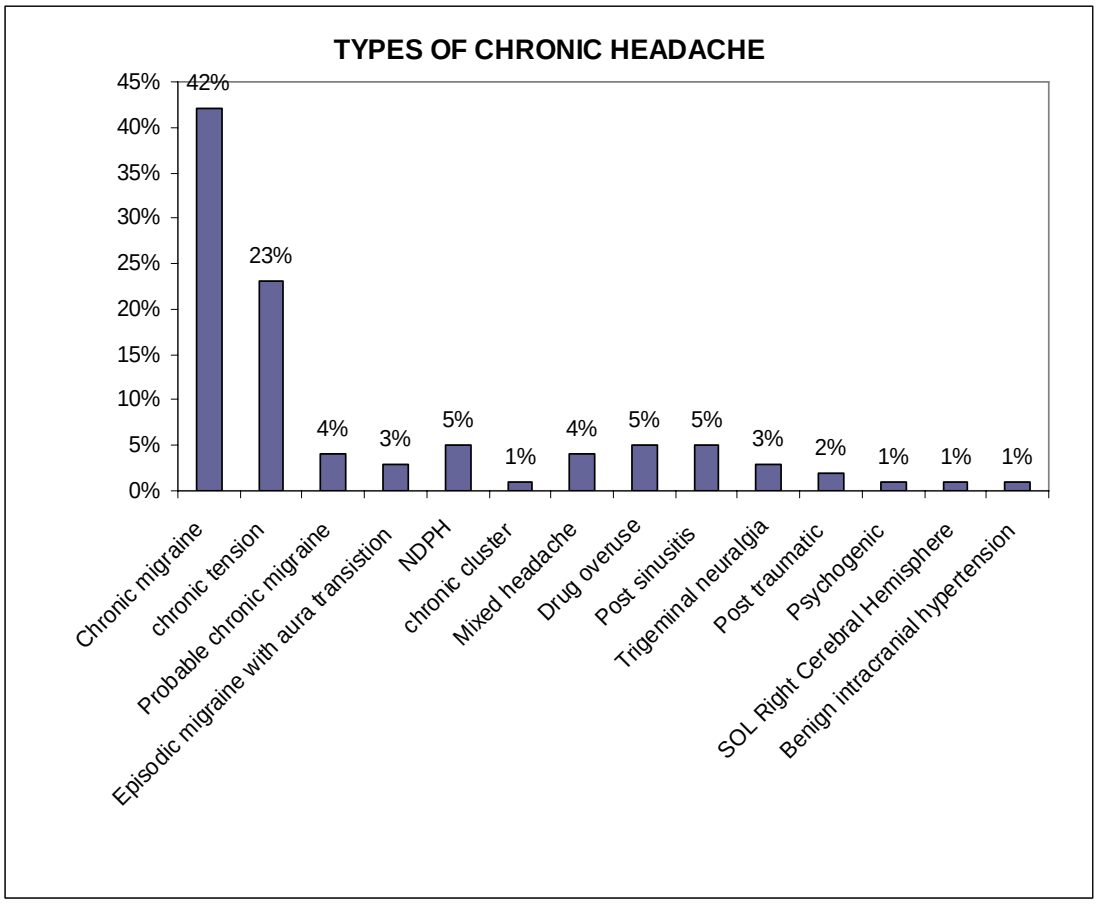
TABLE 18

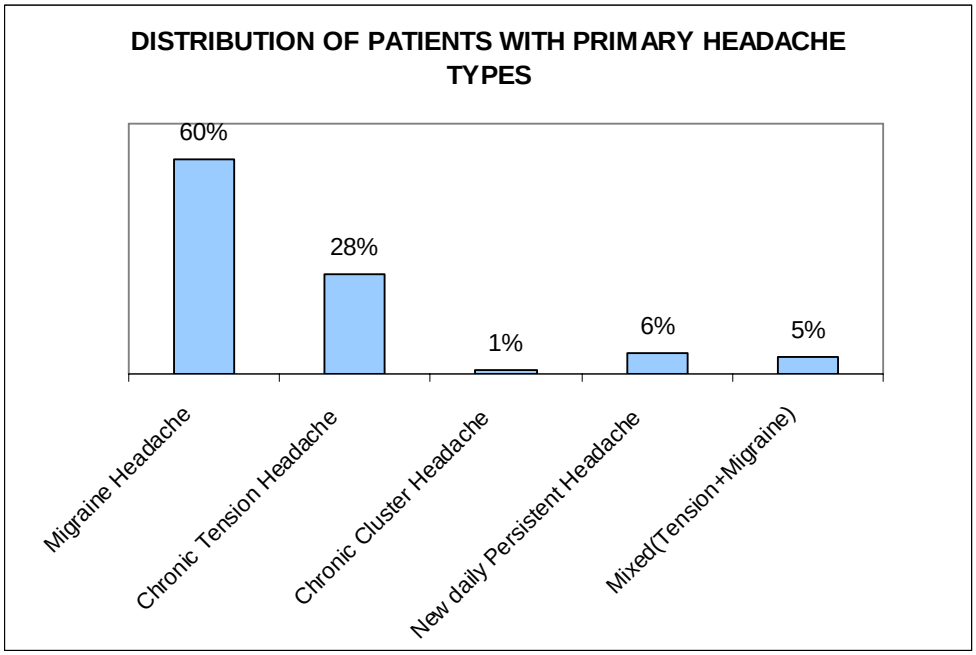
CT AND MRI IN PATIENTS WITH SECONDARY HEADACHE

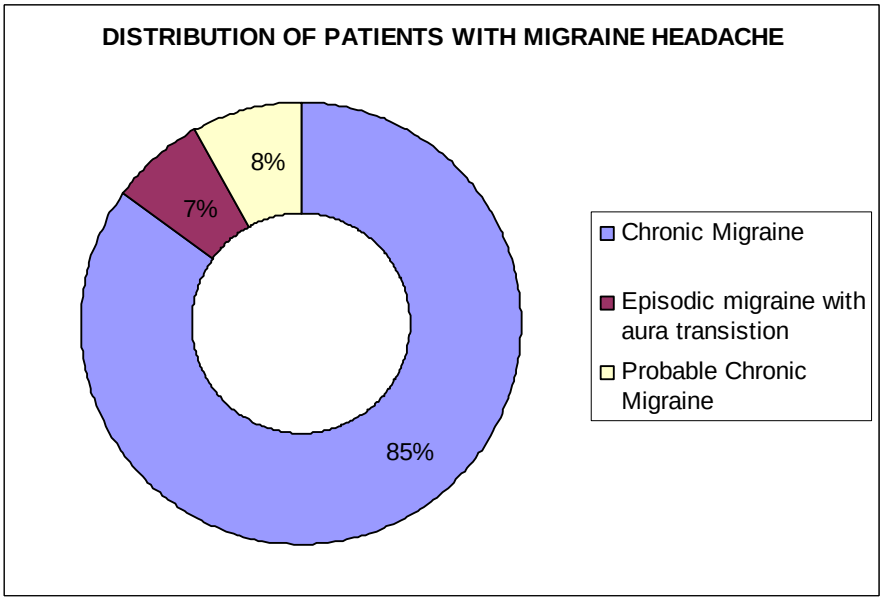
CT / MRI	Image results	Post sinusitis n= 5		Post traumatic n= 2		Benign Intracranial hypertension N=1		Space occupying lesion n=1	
		No. of patients	%	No. of patients	%	No. of patients	%	No. of patients	%
CT BRAIN	Normal	0	0	2	100	0	0	0	0
	SOL	0	0	0	0	0	0	1	100
CT PNS	Maxillary sinusitis	5	100	0	0	0	0	0	0
MRI BRAIN	Prominent Optic N	0	0	0	0	1	100	0	0

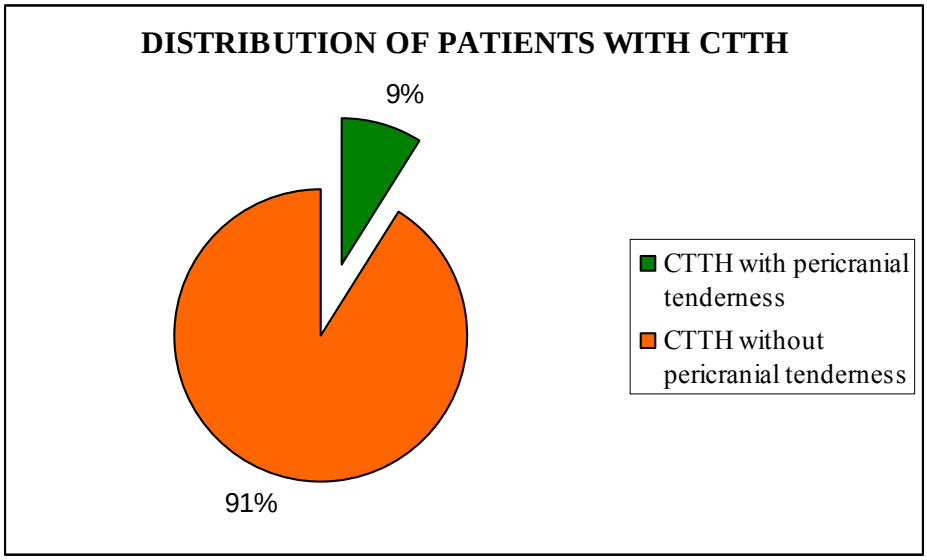
MRI Brain of the patient with benign intracranial hypertension revealed prominent optic nerve. CT Brain of the patients with chronic post traumatic headache was normal .

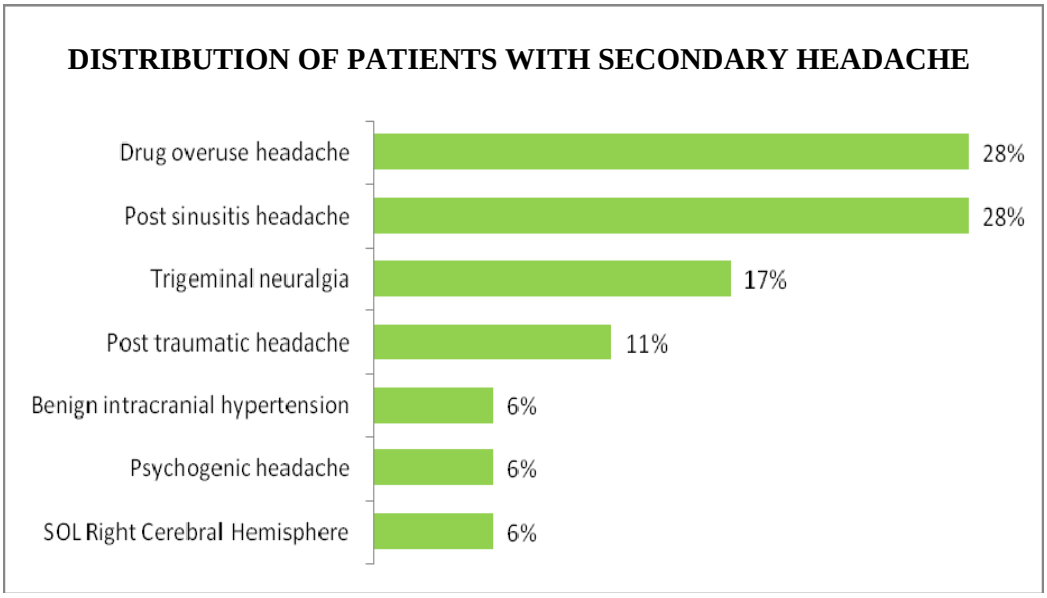

DISTRIBUTION OF PRIMARY AND SECONDARY HEADACHE TYPES AMONG THE TARGET POPULATION
18%
82%
Primary
Secondary

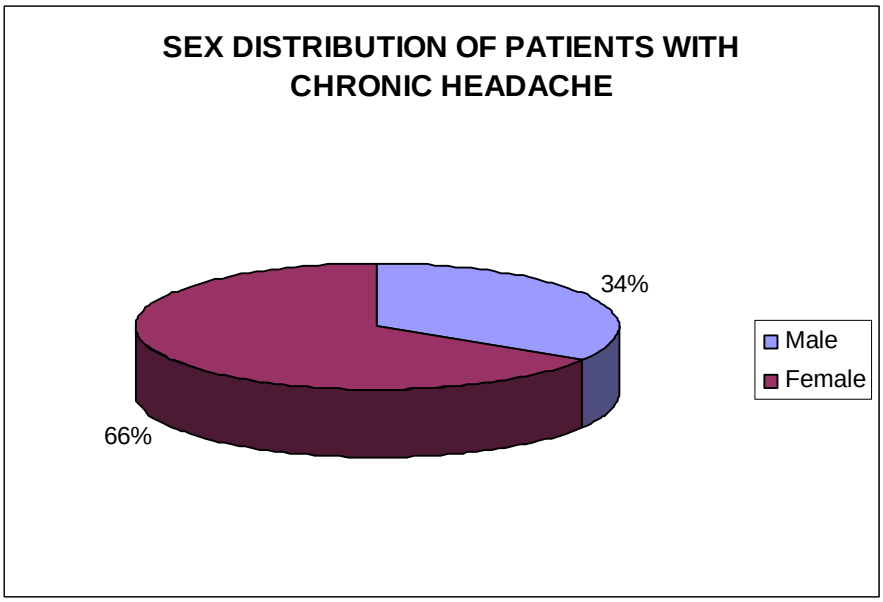

TYPES OF CHRONIC HEADACHE
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
42%
23%
4%
3%
5%
1%
4%
5%
5%
3%
2%
1%
1%
1%
Chronic migraine
chronic tension
Probable chronic migraine
Episodic migraine with aura transistion
NDPH
chronic cluster
Mixed headache
Drug overuse
Post sinusitis
Trigeminal neuralgia
Post traumatic
Psychogenic
SOL Right Cerebral Hemisphere
Benign intracranial hypertension

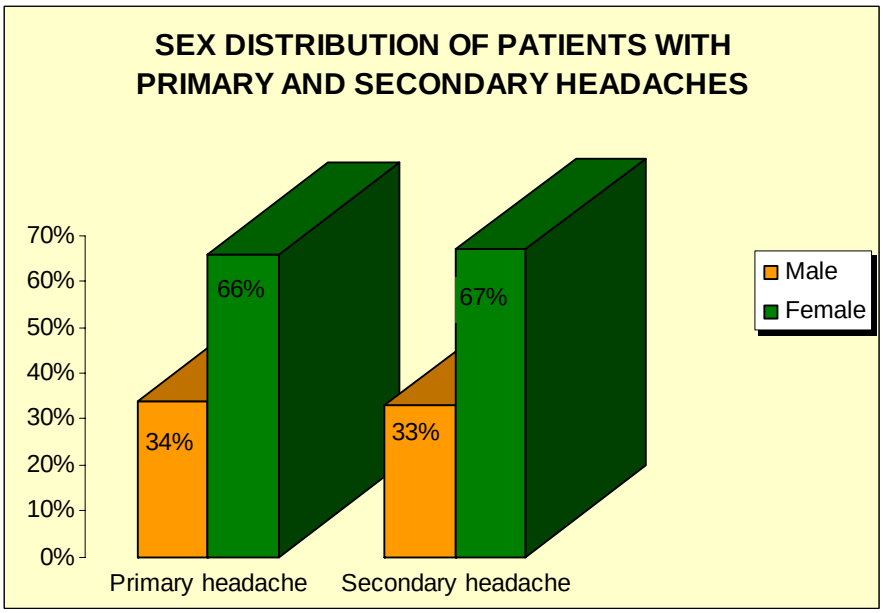

DISTRIBUTION OF PATIENTS WITH PRIMARY HEADACHE TYPES
60%
28%
1%
6%
5%
Migraine Headache
Chronic Tension Headache
Chronic Cluster Headache
New daily Persistent Headache
Mixed(Tension+Migraine)

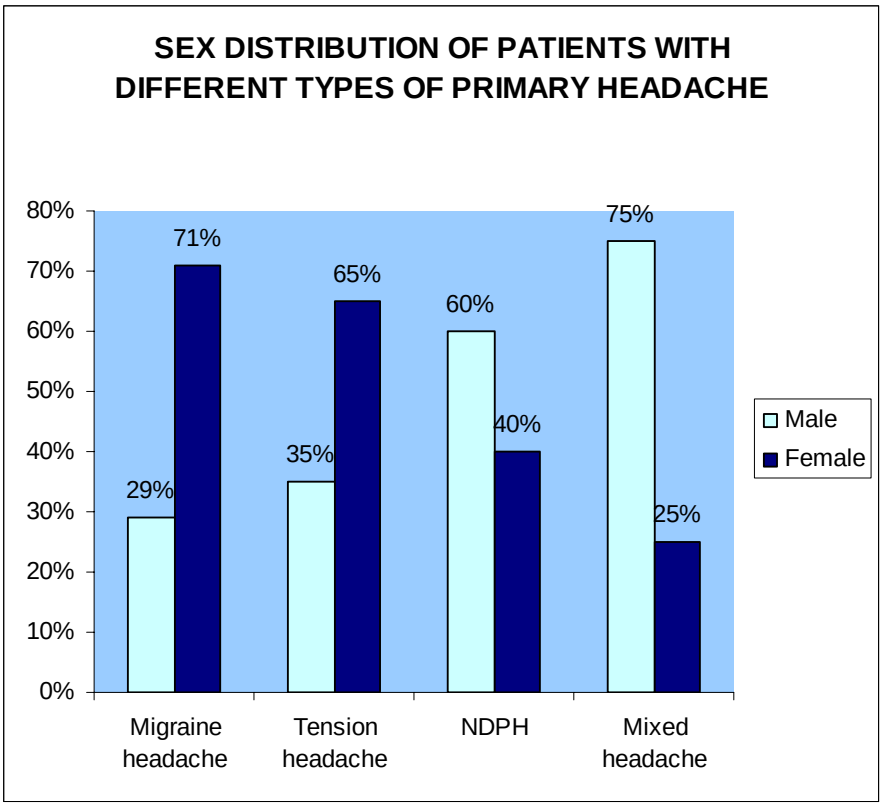

DISTRIBUTION OF PATIENTS WITH MIGRAINE HEADACHE
8%
7%
85%
Chronic Migraine
Episodic migraine with aura transistion
Probable Chronic Migraine

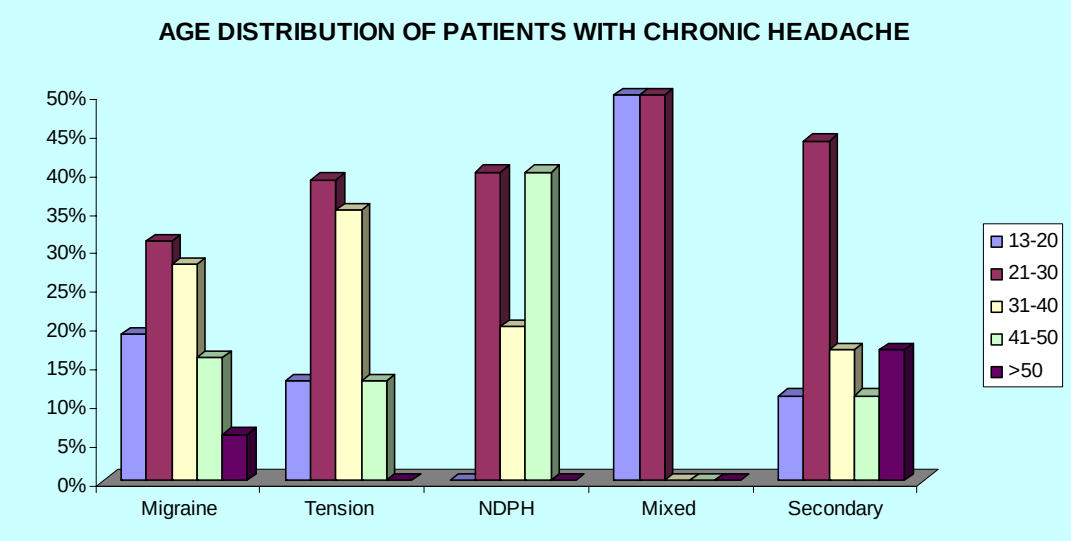

DISTRIBUTION OF PATIENTS WITH CTTH
9%
91%
CTTH with pericranial tenderness
CTTH without pericranial tenderness

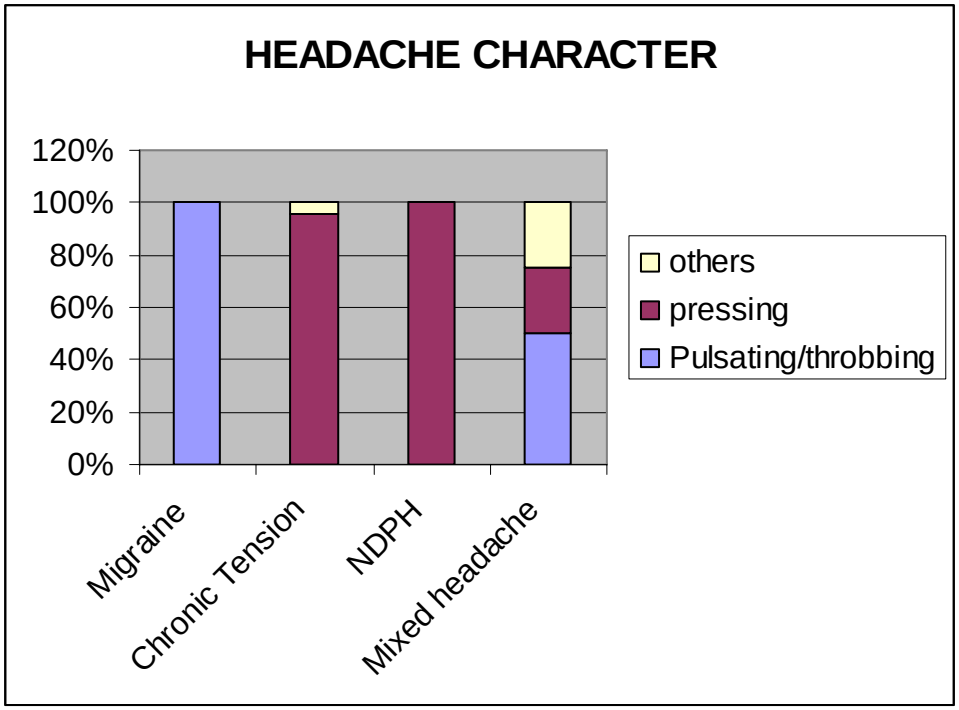

DISTRIBUTION OF PATIENTS WITH SECONDARY HEADACHE
Drug overuse headache
28%
Post sinusitis headache
28%
Trigeminal neuralgia
17%
Post traumatic headache
11%
Benign intracranial hypertension
6%
Psychogenic headache
6%
SOL Right Cerebral Hemisphere
6%

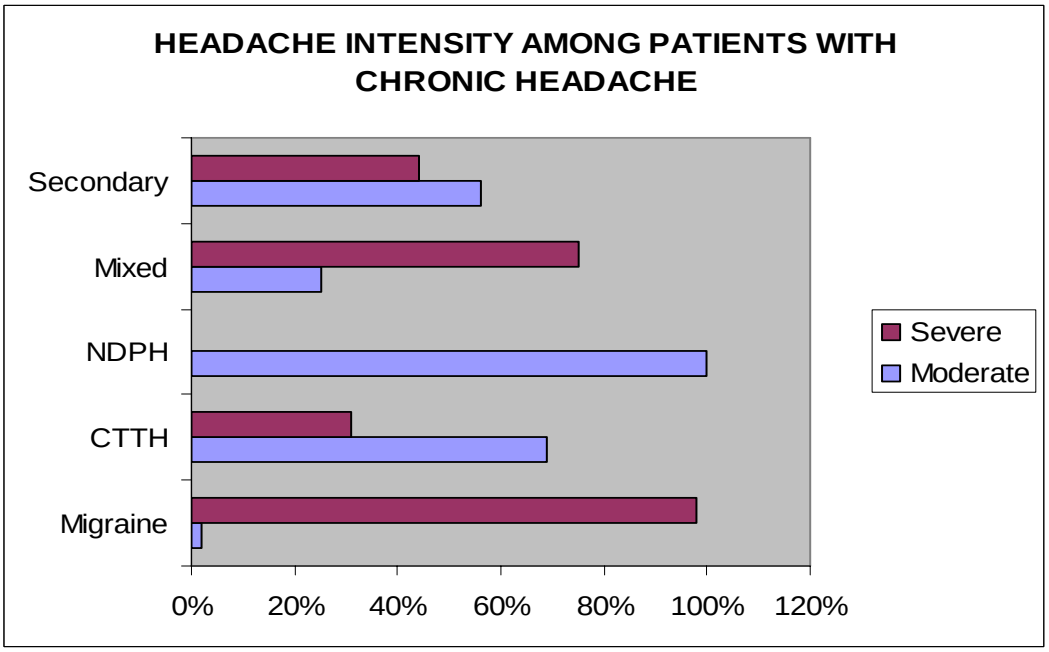

SEX DISTRIBUTION OF PATIENTS WITH CHRONIC HEADACHE
34%
66%
Male
Female


SEX DISTRIBUTION OF PATIENTS WITH PRIMARY AND SECONDARY HEADACHES
70%
60%
50%
40%
30%
20%
10%
0%
34%
66%
33%
67%
Primary headache
Secondary headache
Male
Female

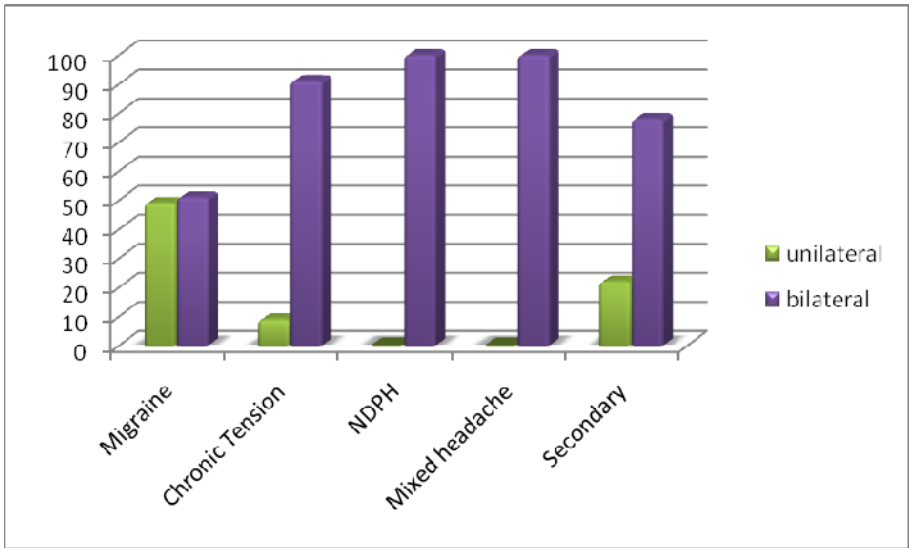

SEX DISTRIBUTION OF PATIENTS WITH DIFFERENT TYPES OF PRIMARY HEADACHE
80%
70%
60%
50%
40%
30%
20%
10%
0%
29%
71%
35%
65%
60%
40%
75%
25%
Migraine headache
Tension headache
NDPH
Mixed headache
Male
Female

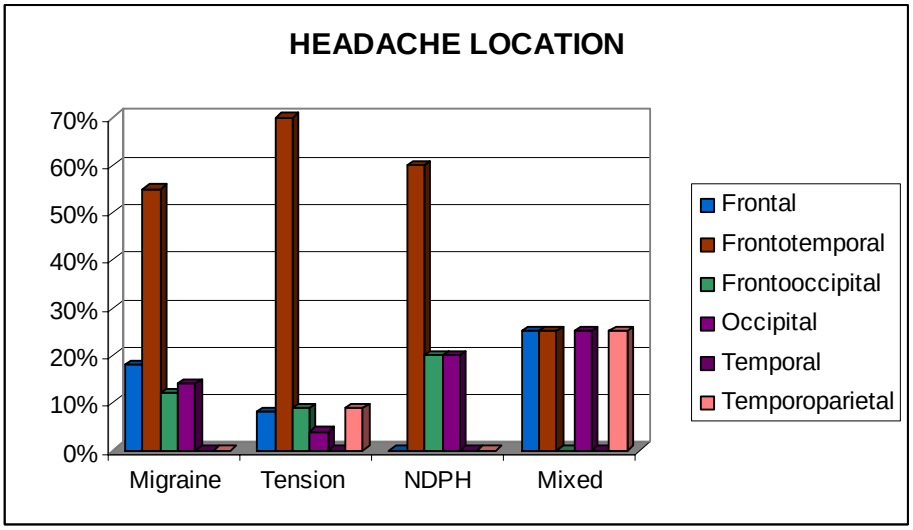

AGE DISTRIBUTION OF PATIENTS WITH CHRONIC HEADACHE
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
Migraine
Tension
NDPH
Mixed
Secondary
13-20
21-30
31-40
41-50
>50


HEADACHE CHARACTER
120%
100%
80%
60%
40%
20%
0%
Migraine
Chronic Tension
NDPH
Mixed headache
others
pressing
Pulsating/throbbing

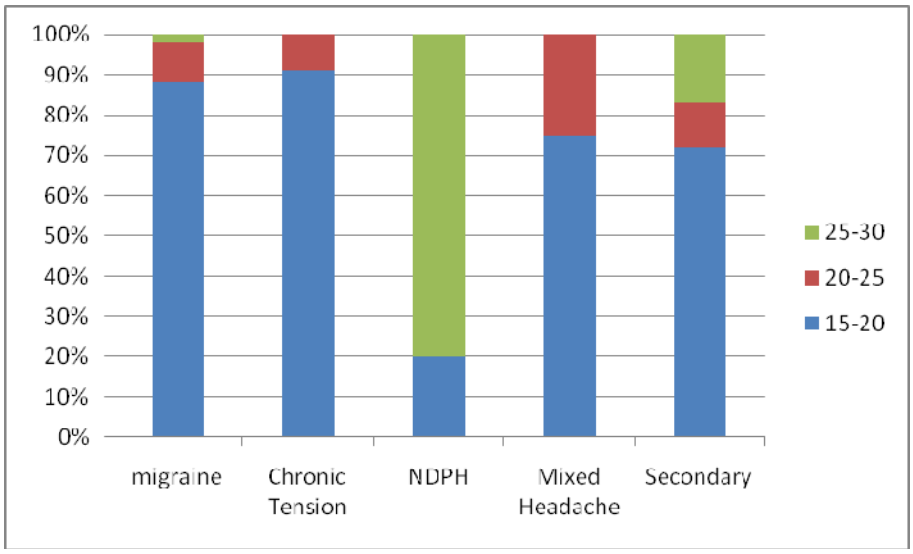

HEADACHE INTENSITY AMONG PATIENTS WITH CHRONIC HEADACHE
Secondary
Mixed
NDPH
CTTH
Migraine
0%
20%
40%
60%
80%
100%
120%
Severe
Moderate

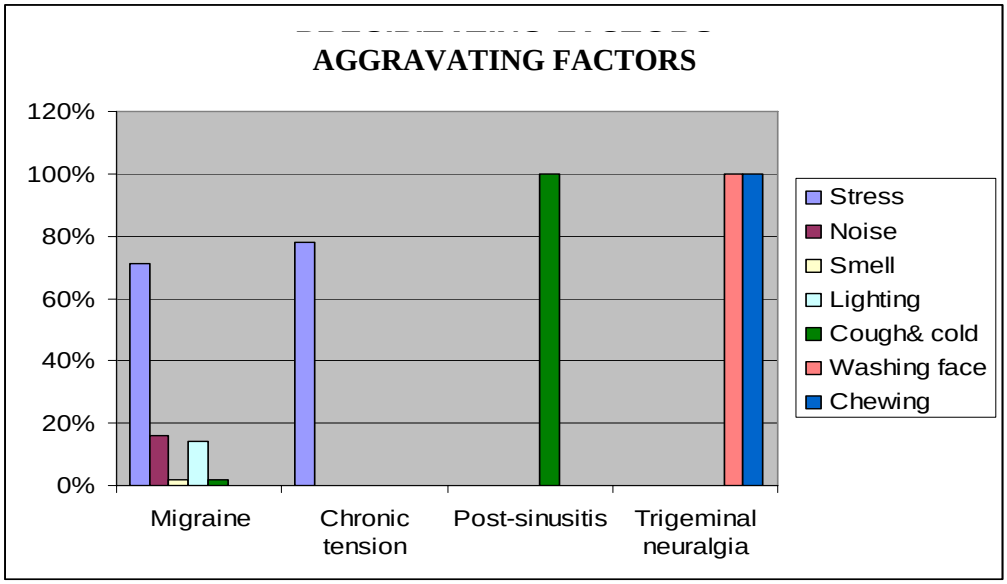
UNILATERALITY OF HEADACHE



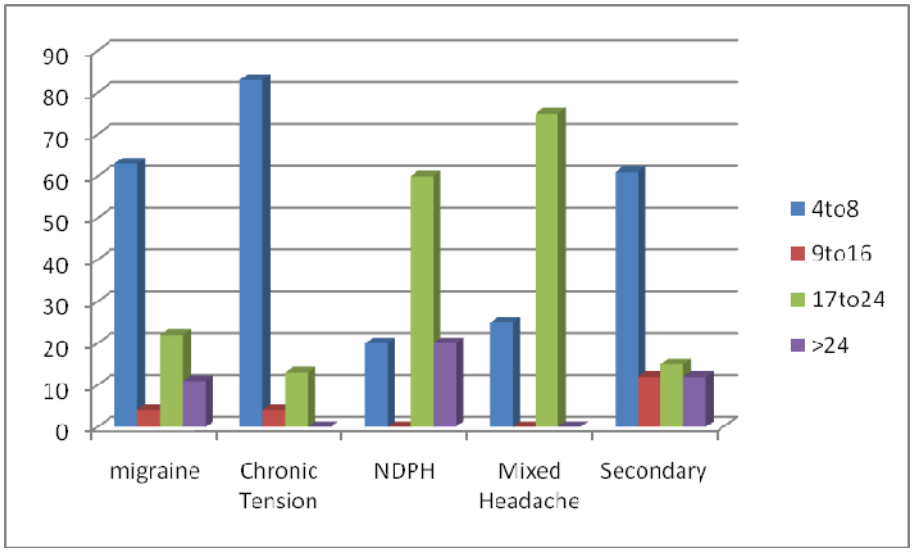


DISTRIBUTION OF FREQUENCY OF HEADACHE

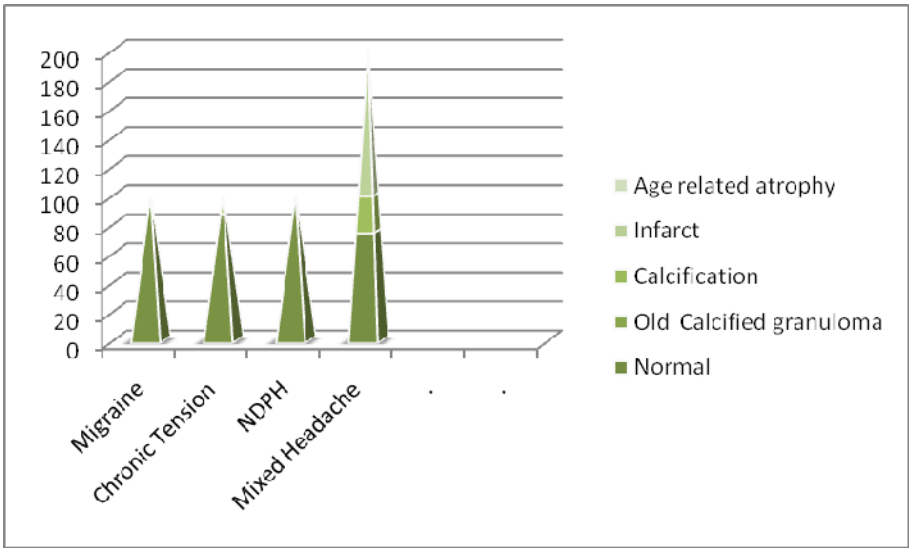


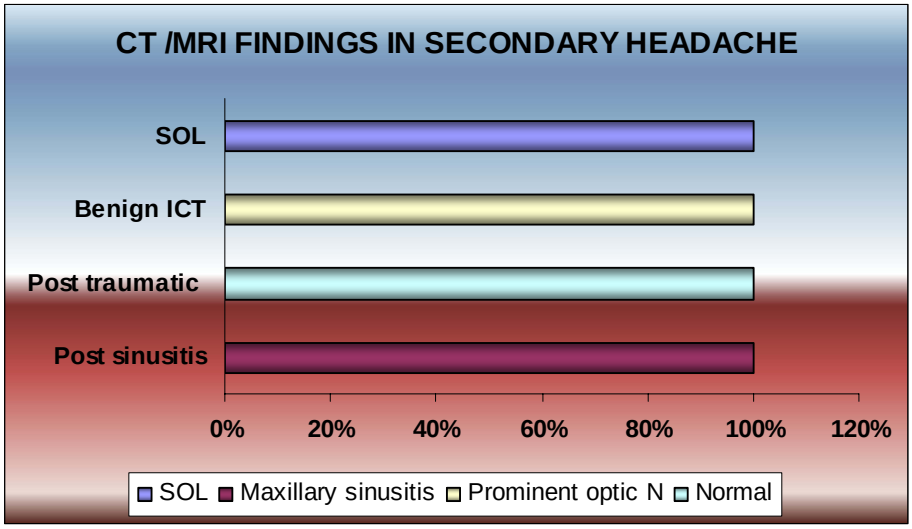


DISTRIBUTION OF DURATION OF HEADACHE



CT FINDINGS IN PATIENTS WITH PRIMARY HEADACHE





DISCUSSION

In the present study, out of 100 patients with Chronic headache, primary headache was the predominant type accounting for 82% as compared to Secondary headache seen only in 18%. (Table – 1) This correlated well with the study done by AP Jain et al 2007 in which majority of patients presented with primary headache accounting for 92.5% and the remaining 7.5% with secondary headache. Even similar results were obtained in the study done by Ade C longe et al 1998, where primary headache was diagnosed in 88% of patients with chronic headache and secondary headache in remaining 12%.

Of the 100 target population in the current study , 49% presented with Migraine and its variants, 23% with tension type , 4% with Mixed (Tension +Migraine), 5% with NDPH, 1% with cluster headache, 2% with post traumatic, 5% with post sinusitis , 5% with drug overuse , 1% with SOL and about 1% with Benign intracranial hypertension .(Table-2)

This correlated well with the study done by Ade C Longe et al 1998, where about 43% presented with Migraine and its variants, 38% with tension type, 6.7% with Mixed migraine and tension type , 2.2% with post traumatic and about 2.2% with with pseudo tumour cerebri.

Among the 82 patients with primary chronic headache , in the current study , Chronic Migraine was the most common type seen in 49 patients

(60%) followed by CTTH in 23 patients (28%)and about 5 patients(6%) presented with NDPH.(Table-3) This was comparable to the earlier study done by Chakravarthy et al 2003, who reported that 82% of the patients suffered from Migraine followed by 16% from CTTH and about 1.5% with NDPH. This was in contrast to the study done by Ravi et al 2007 who has reported CTTH as the most prevalent type.

This profile of headaches in our study was in inverse correlation with many international studies .Epidemiological evidence from around the world suggests TTH is the most common cause of primary headache. This variance is attributed to self treatment of tension type headaches by the general population.

Out of a total of 82 patients with chronic primary headache, 23 (28%) patients had chronic tension type headache , of which 2 (9%) patients presented with pericranial tenderness and the remaining 21 (91%) patients without pericranial tenderness (Table-5)

Among the 100 patients with chronic headache, current study revealed female predominance accounting for 66% (Table- 7) . Both in the primary and secondary headache group, females outnumbered males. Even there was no significant difference in the gender distribution among the patients with primary and secondary headaches. (Table-7a).

Present study also revealed that females outnumbered males in number of cases of both tension type and migraine headache whereas male predominance among patients with NDPH and mixed type headache. Among the migraine patients, women seem to be more affected than men in a ratio about 2.3 :1. (Table – 7b) This was similar to the study done by Jain et al who showed a female dominance in both patients with migraine and tension type headache.

In this study , majority of the headache problems were observed in the population aged 21 to 40 years , the most productive age group which included patients of Migraine (59%) and Tension headache(74%) (Table-8) Even in the hospital based study done by A.P.Jain et al , majority involved the young population aged 21 to 40 years. About 76.4% of the migraine and 65.2% of the tension type headache patients were within this age group .

Current study revealed that almost 100% of the migraine patients presented with headache which was pulsating / throbbing in nature whereas 96% of the tension headache with pressing (non- throbbing) type of headache. (Table -9)This was comparable to the study done by Young chou et al 1996 where 87% of the migrainous patients presented with throbbing headache and about 72% of the patients with tension headache had headache which was non-throbbing in nature.

Inspite of the chronification of the headache , in this study, the intensity of pain was more severe in about 98% of patients with Migraine headache whereas 69% of patients with CTTH had pain of moderate intensity.(Table -10) This was comparable to the study done by Young Chul Choi et al, where majority of migrainous patients presented with headache of severe intensity and majority of tension headache patients had pain of moderate intensity.

In this study, 51% of the migraine patients presented with bilateral headache and the remaining 49% with unilateral headache. Majority of patients with tension and other primary headache disorder (except the patient with cluster headache) , presented with bilateral headache as per the diagnostic criteria of ICHD-II. (Table -11) Whereas in the study done by Young Chul Choi al 1996 , 70% of the migrainous patients presented with unilateral pain and about 63% of tension headache patients had bilateral pain.

Majority of the migrainous and tension headache patients presented with headache at the fronto temporal area accounting for about 55% and 60% respectively. (Table -12) In the study done by A P Jain et al 2007, frontotemporal area is the most common site of migraine headache whereas tension type headache was situated all over the scalp.

The majority of chronic headache patients were suffering from headache for around 15-25 days / month. However patients with NDPH presented with headache throughout the month .(Table-13) This explains the morbidity of chronic headache and also explains the poor quality of life in the sufferers.

In the current study, stress is the major aggravating factor in patients with chronic tension type headache constituting about 78% . This is comparable to the study done by Chakravarthy et al , in which stress has been implicated as the aggravating factor for the development of CTTH in about 72.7% whereas 43.8% of the chronic migrainous patients had stress as the aggravating factor.

Among the patients with migraine headache in the present study , stress has been implicated as the major aggravating factor in about 71% followed by noise , smell and lighting in about 16% , 2% and 14% respectively .(Table-14)

Symptoms such as nausea , vomiting , photophobia , blurring of vision were commonly associated with migraine headache . Among the patients with TTH , few had nausea, vomiting and blurring of vision . Lacrimation and rhinorrhea were commonly associated with cluster headache.(Table-15)

In the current study, CT Brain reveals normal study in almost majority of patients with chronic primary Headache . CT Brain of few patients

revealed calcification and age related atrophy which are insignificant.(Table-17) As per ICHD-2 criteria primary headache is usually not associated with any structural abnormalities in the brain.

In this study , among the patients with secondary headache , CT Brain was normal in patients with medication overuse headache and post traumatic headache whereas CT PNS revealed sinusitis in almost all patients with post sinusitis headache.

CT Brain in the patient with Benign intracranial hypertension showed dilated ventricles and CT of a 55year old male revealed a space occupying lesion on the right temporoparieto occipital region.(Table-18)

SUMMARY

- Among the 100 patients with Chronic headache , **Chronic primary headache** was the most common type accounting for 82%
- **Chronic migraine headache** was the most common type of chronic headache constituting 42% of the target population.
- There was a female preponderance accounting for 66% of the target population. Females outnumbered males in both primary and secondary headache.
- Majority of the study population belonged to the age group of 21 -40 years.
- Out of the 82 patients with chronic primary headache , 49 patients (60%) presented with Chronic migraine followed by 23 patients (28%) with Chronic tension type headache. Mixed
- **Chronic migraine** was the most common primary headache disorder . Majority of the patients presented with typical characteristics of headache such as Pulsatile/ throbbing quality (100%) , severe intensity (98%) , Frontotemporal in location(55%), presenting around 15-25 days/ month (98%) with out any structural lesions in CT brain.
- **CTTH** was the second most common type of primary headache which commonly presented with pressing quality , moderate intensity,

bilateral in nature, fronto temporal in location with normal study on CT brain.

- STRESS was the most common aggravating factor in patients with chronic migraine as well as CTTH.
- **NEW DAILY PERSISTENT HEADACHE** was accounted for 6% of primary headache which commonly presented with Bilateral, moderate intensity with pressing quality which presents daily and remaining through out the day with out structural lesions in CT brain.
- Out of 18 patients , medication overuse headache and post sinusitis headache were the most common type of secondary headache
- Secondary headache most commonly seen in the age group of 21-40 years with characteristic bilateral location, moderate to severe intensity with specific aggravating factors according to the type of headache with structural lesions noted in CT brain & paranasal sinuses.

CONCLUSION

Headache in India is as important as any other neurological problem , and yet it is neglected . The study of headache is relatively simple and cheap compared to other neurological disorders. Prevalence data are essential.

This study documents the profile of chronic headache patients and highlights the characteristics of headache and factors that predict headache associated morbidity. Although most patients suffered from primary chronic headache requiring only clinical evaluation , CT scan is also necessary to reassure patients for the absence of an ominous structural intracranial lesions.

To prevent misconceptions among doctors and to promote research , headache must be given greater importance in the medical curriculum.

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CLINICAL PROFORMA

NAME

AGE

SEX

OCCUPATION

CHIEF COMPLAINTS:-

HEADACHE

SITE

U/L OR B/L

MODE OF ONSET

THROBBING/PULSATING

INTENSITY

DURATION

RADIATION

LAST EPISODE

PERIODICITY

DAY/NIGHT

NO OF ATTACKS/MONTH

ASSOCIATED SYMPTOMS:-

BLURRING OF VISION

PHOTOPHOBIA / PHONOPHOBIA

DIZZINESS

NAUSEA & VOMITING

AURA SYMPTOMS

LACRIMATION

RHINORRHOEA

AGITATION

LOC

SEIZURES

NECK STIFFNESS

FOCAL NEUROLOGICAL DEFICIT

MOTOR/ SENSORY

AGGRAVATING FACTORS:-

LIGHTING

STRESS

NOISE

SMELLS

ALCOHOL

CAFFEINE

COUGH& COLD

H/O TRAUMA

H/O NECK PAIN

H/O FEVER

H/O REFRACTORY ERROR

H/O SINUSITIS

H/O DENTAL CARIES

H/O DEPRESSION/MOOD SWINGS

PAST HISTORY

H/O TYPE2 DM

H/O CVA

H/O SHT

H/O DRUG INTAKE

H/O HEAD INJURY

H/O SUBSTANCE ABUSE

H/O FEVER WITH NECK STIFFNESS

H/O SYSTEMIC ILLNESS

MENSTURAL HISTORY

EXAMINATION

GENERAL CONDITION:-

HIGHER FUNCTIONS:-

CRANIAL N:-

FUNDUS

SPINO MOTOR SYSTEM:

POWER

REFLEXES

PLANTAR

SENSORY

CEREBELLAR

BOWEL& BLADDER

SPINE & CRANIUM

SCALP TENDERNESS

ENT OPINION

OPTHAL OPINION

PSYCHIATRY OPINION

INVESTIGATIONS

CT BRAIN

CT PARANASAL SINUS

MRI BRAIN

IMPRESSION:-

ABBREVIATIONS

CDH	Chronic Daily Headcahe
CM	Chronic Migraine
CTTH	Chronic Tension type Headache
NDPH	New Daily Persistent Headache
IHS	International Headache Society
ICHD	Internation Classification of Headache Disorders
SSRI	Selective Serotonin Reuptake Inhibitors
CT	Computed Tomography
MRI	Magnetic Resonance Imaging
EEG	Electroencephalogram
PET	Positron Emission Tomography
ICP	IntraCranial Pressure
PNS	Paranasal Sinus
DHE	Dihydro ergotamine
NSAID	Non Steroidal Anti Inflammatory Drugs
CSF	Cerebro Spinal Fluid
cGRP	Calcitonin gene related peptide
CBF	Cerebral Blood Flow
SOL	Space Occupying Lesion