PRIMARY HEADACHE:

BY: S.KAVIYA SRI

ABSTRACT:

Headache is usually benign in nature. Headache commonly occurs as a symptom of neurological conditions when it causes severe disabilities. Headaches are of two types among them we are going to discuss about primary headaches. Different medications can be used for primary headaches. Common types of primary headache are Migraine (with or without aura), Tension type headache and Cluster headache. For chronic cases of migraine Triptans or Triptans related drugs can be used. In case of tension type headache ibuprofen and aspirin can be used, and for cluster headache verapamil and lithium can be used

INTRODUCTION:

Migraine is a ubiquitous familial disorder characterized by periodic, commonly unilateral, often pulsatile headaches that begin in childhood, adolescence, or early adult life and recur with diminishing frequency during advancing years. Two closely related clinical syndromes have been identified, the first called migraine with aura and the second, migraine without aura the first syndrome was referred to as classic or neurologic migraine and the second as common migraine. Each patient displays a proclivity for the pain to affect one side or the other of the cranium , but not exclusively , so that some bouts are on the other side.

The heritable nature of classic migraine is apparent from its occurrence in several members of the family of the same and successive generations in 60 to 80 percent of cases ; the familial frequency of common migraine is slightly lower. Twin and sibling studies have not revealed a consistent Mendelian pattern in either the classic or common form .Certain special forms of migraine such as familial hemiplegic migraine , appear to be monogenic disorders but the role of these genes, most of which code for ion channels, in classic and common migraine is speculative.One third of migraineurs have more than three attacks monthly if untreated and many require bed rest or severe curtailment of daily activities.

Migraine may have its onset in childhood but usually begins in adole-scence or young adulthood ; in more than 80 percent of patients,the onset is before 30 years of age, and the physician should be cautious in attributing headaches that appear for the first time after this age to migraine, although there are exceptions. In younger women, the headaches often tend to occur during the pre-menstrual period; in approximately 15 percent of such migraineurs, the attacks are exclusively perimenstrual. Menstrua migraine, had been considered to be solely related to the with-drawal of estradiol. It is now acknowledged that the influence of sex hormones on headache is more complex.

Migraine tends to cease during the second and third trimesters of pregnancy in 75 to 80percent of women, and in others they continue at a reduced frequency ;less often, attacks of migraine or the associated neurologic symptoms first appear during pregnancy, usually in the first trimester. Although migraine commonly diminishes in severity and frequency with age , it may actually worsen in some postmenopausal women, and estrogen therapy may either increase or, paradoxically, diminish the incidence of headaches. The use of birth control pills is associated with an increased frequency and severity of migraine and in rare instances has resulted in a permanent neurologic deficit.

ETIOLOGY:

Most of the time etiology for migraine is idiopathic ,but sometimes it can be due to:

* Hormone changes
* Stress
* Foods
* Skipping meals
* Caffeine
* Sense

Symptoms of migraine:

Vision disturbances or aura are considered a warning sign that a migraine is coming.The aura occurs in both eyes and may involve any or all of the following:

* A temporary blind spot
* Blurred vision
* Eye pain
* Seeing stars or zigzag lines
* Headache
* Hallucinations
* Other focal neurologic symptoms, much less common than visual ones, include numbness and tingling of the lips, face, and hand.
* Slight confusion of thinking
* Weakness of an arm or leg
* Mild aphasia or dysarthria, dizziness
* Uncertainty of gait or drowsiness.
* Nausea and vomiting may occur.

Pathogenesis:

For many years ,our thinking about the pathogenesis of migraine was dominated by the views of Harold Wolff and others - that the headache was caused by the distention and excessive pulsation of branches of the external carotid artery. Certainly , the throbbing , pulsating quality of the headache and its relief by compression of the common carotid artery supported this view , as did the early obser-vation of Graham and Wolff that the headache and amplitude of pulsation of the extra-cranial arteries diminished after the intravenous administration of ergotamine .

The importance of vascular factors continues to be emphasized by more recent findings but not in the way envisaged by Wolff . For example , in a group of 11 patients with classic migraine , Olsen and colleagues, using the xenon inhalation method ,noted a regional reduction in cerebral circulation spreading forward from the occipital region during the period when neurologic symptoms appear . they concluded that the reduction in blood flow was consistent with the cortical spreading depression syndrome. In a subsequent study, Woods and colleagues described a patient who ,during positron emission tomography , fortuitously had an attack of common migraine with blurred vision.

In reference to the extracranial vessels, Iversen and associates , by means of ultra-sonography , documented a dilatation of the superior temporal artery on the side of the migraine during the headache period. The same dilatation in the middle cerebral arteries has been inferred from observations with transcranial doppler insonation.

The complication of cerebral infarction is also in keeping with a vascular hypothesis, but it involves only a tiny proportion of migraineurs . The vascular hypothesis must be regarded as uncertain , but , clearly , there is frequently a reduction in posterior cortical blood flow during an aura . What is not established is whether the blood flow changes are fundamental or simply the result in a reduction in cortical activity.

The original opinion expressed by Wolff that a vascular element is responsible for the cranial pain of migraine is also unconformed that a vascular element is responsible for the cranial pain of migraine is also uncon-formed.

The relationship between the vascular changes and evolving neurological symptoms of migraine are noteworthy. Lashley , who plotted his own visual aura , calculated that the cortical impairment progressed at the rate of 2 to 3 mm/ min over the surface of the brain. Similarly , during the aura, there is a regional reduction in blood flow , as noted above. It begins in one occipital lobe and extends forward slowly as a wave of “spreading oligemia” that does not respect arterial boundaries . both of these events are intriguingly similar to the above mentioned phenomenon of “spreading cortical depression”, first observed by Leavo in experimental animals.

He demonstrated that a noxious stimulus applied to the rat cortex was followed by vasoconstriction and slowly spreading waves of inhibition of the electrical activity of cortical neurons , moving at a rate of approximately 3mm/min.

An alternative , but not necessarily exclusive hypothesis links the aura and the painful phase of migraine through a neural mechanism originating in the trigeminal nerve as proposed by Moskowitz. This is based on the innervation of extracranial and intracranial vessels by small unmyelinated fibers of the trigeminal nerve that subserve both pain and autonomic functions. This model provides an explanation for migraine pain in the trigeminal ganglion. Activation of these fibers releases substances P, calcitonin gene related peptide (CGRP), and other peptides intothe vessel wall , which serves to sensitize the trigeminal system to the pulsatility of cranial vessels,and to increase their permeability,thereby promoting an inflammatory response.

The small molecules released from nerve endings adjacent to the cortex would then incite spreading depression in this model. Against this hypothesis is the occurrence of headache as often as not on the side opposite the side of generation of the aura and the lack of clinical effect of drugs that work in this experimental model. Most likely, both neural and vascular mechanisms are operative and they interact.

In part to address the action of the serotonin agonist drugs on migraine, a body of evidence has been assembled that serotonin acts as a humoral mediator in the neural and vascular components of migraine headache . Serotonin is discharged from platelets at the onset of headache and the headache is reduced by the injection of 5-HT.

This led to the development by Humphrey of sumatriptan , which acted selectively on 5-HT1B /D receptors so as to reduce side effects.this was the forerunner of the large group of “triptans”. More recently, nitric oxide generated by endothelial cells has been implicated as the cause of the pain of migraine headache , but the reason for its release and the relationship to changes in blood flow is unclear.

Investigation:

* Imaging studies detect a significant abnormality in <0.5% patients with migraine and a normal neurological examination,are not usually indicated.
* MRI scan in migraine patients with or without auras, may reveal small nonspecific white matter lesions in 30% of individuals under the age of40 years.
* Magnetic resonance venographies (MRV) are useful in identifying acute arterial occlusion, aneurysm and venous sinus thrombosis.

Clinical features of Migraine:

Prodrome :

Vague premonitory symptoms that begin from 12 to 36 hours before the aura and headache. It lasts for about 15 to 20 minutes.

Symptoms

* Yawning
* Excitation
* Depression
* Lethargy
* Craving or distaste for various foods

Types of migraine:

1. Migraine with aura
2. Migraine without Aura
3. Basilar migraine
4. Retinal migraine
5. Hemiplegic migraine

1.Migraine with aura: Migraine with aura, the term now used to denote classic migraine, is ushered in by an evident disturbance of nervous function, most often visual, followed in a few minutes by hemicranial or, in about one-third of cases, by bilateral headache, nausea, and sometimes vomiting, all of which last for hours or as long as a day or two. Symptoms of migraine are headache ,throbbing ,unilateral ,worsened by bright light and relieved by sleeping in a dark room.

Migraine with aura frequently has its onset soon after awakening, but it may occur at any time of day. During the preceding day or so, there may have been mild changes in mood (sometimes a surge of energy or a feeling of well-being), hunger or anorexia, drowsiness, and frequent yawning).

2.Migraine without Aura:

2a. Sensitivity to light and noise attends both types, and intensification with movement of the head is common

* It is paroxysmal headache without vomiting but no aura.
* Scalp may be tender during episodes.
* Preferences is to in dark and quit environment.

3 . Basilar migraine :

A less common form of the migraine syndrome which affects both visual fieldS (temporary cortical blindness may occur) and associated vertigo, staggering, incoordination of the limbs, dysarthria, and tingling in both hands and feet and sometimes around both sides of the mouth. These symptoms last 10 to 30 min and are followed by headache, which is usually occipital.

There is an alarming period of coma or quadriplegia. basilar migraine, though more common in children and adolescents, affects men and women more or less equally over a wide age range, and that the condition is not always benign and transient

4. Ophthalmoplegic and Retinal Migraine:

Ophthalmoplegic are recurrent unilateral headaches associated with weakness of extraocular muscles. A transient third-nerve palsy with ptosis, with or without involvement of the pupil, is the usual picture; rarely, the sixth nerve is affected. This disorder is more common in children The paresis often outlasts the headache by days or weeks.

Retinal migraine, or, more accurately, ocular migraine, since either the retinal or the ciliary circulation may be involved. It is well to remember that in adults the syndrome of headache, unilateral ophthalmoparesis, and loss of vision may have more serious causes, including temporal (cranial) arteritis

5.Hemiplegic Migraine:

* Hemiplegic Migraine, is also called as” migraine variant ”. Adults have episodes of unilateral paralysis that may long outlast the headache. Hemiplegic means paralysis on one side of the body.
* A person with hemiplegic migraine will experience a temporary weakness on one side of their body as part of their migraine attack. This involves face , arm or leg and be accompanied by numbness or pins and needles.
* The patient may experience speech difficulties, vision problems or confusion.Symptoms are similar to those of a stroke and TIA and need a careful differential diagnosis.

Triggering factors of Migraine attack:

* Dietary items particularly chocolate, cheese
* Fatty foods
* Oranges, tomatoes, and onions

Some of these foods are rich in tyramine, which has been incriminated as a provocative factor in migraine. Alcohol, particularly red wine or port, regularly provokes an attack in some persons;in others, headaches are fairly consistently induced by exposure to glare or other strong sensory stimuli, sudden jarring of the head (“footballer’s migraine”), or by rapid changes in barometric pressure. A common trigger is excess caffeine intake or withdrawal of caffeine. Light is irritating and may be painful to the globes, or it is perceived as overly bright and strong odors are disagreeable.

Conclusion :

Between attacks, the migrainous patient is normal. In the past, it was believed thata migrainous personality existed, characterized by tenseness, rigidity of attitudes and thinking, meticulousness, and perfectionism. Further analyses, however, have not established a particular personality type in the migraineur.

A relationship of migraine to epilepsy in general is also tenuous; however, the incidence of seizures is slightly higher in migrainous patients and their relatives than in the general population, and there are syndromes that encompass both disorders.

Some patients note that their attacks of migraine tend to occur during the “let-down period”, after many days of hard work or tension.

Moreover, as appreciated by Graham, migraine has a lifetime profile and is a familial disease that includes some or many of the following : colic in infancy, motion sickness, episodic abdominal pain, fainting, alcohol sensitivity, exercise-induced headaches, “sinus headaches”, “tension headaches”, and menstrual headaches.

These are fairly dependable markers of the disease, and their absence in the patient or family members should at least cause the consideration of alternative explanations for facial pain.

2)TENSION TYPE HEADACHE

INTRODUCTION

Tension headaches are also called as stress headaches, which commonly occurs in adult. The major trigger point of this type of headache is either stressful situation or overloaded pressure which causes stress for a long period of time. Additionally this headache occurs due to lack of sleep, weather changes and even some times an after effect of drinking alcohol.

Patients those who have tension headache will feel as a dull pain or band like pressure around the head, usually both the sides. Two types of headache occurs here, if headache occurs less than 15 times in a month it is termed as episodic tension headache whereas if it occurs 15 or more than 15 times in same one month then it is termed as chronic tension headache. Both will last more than 30 minutes to more days .

Patients will have variation in pain, like they will have mild tension headache in the morning continued by severe pain in the evening for a whole day.

CAUSES:

1. Stress and anxiety
2. Lack of water
3. Wastage of muscles in the neck
4. Visual effects like bright sunlight
5. Posture disability

SYMPTOMS:

1. Mild to moderate pain in the front, top or sides of our head
2. Feeling mostly dizzy
3. Trouble in concentrating
4. When they see light or hear some vigorous noise.
5. Muscular aches

DIAGNOSIS:

Tension headache can be diagnosed by attempting to identify the triggering points which may involve questionnaire containing questions regarding the lifestyle and their medical history

Circumstantial factors like

* A head injury happened within or before 3 months.
* Headache along with high fever
* Headaches accompanied by confusion
* Headache accompanied by nausea or vomiting.

TREATMENT:

PAINKILLERS:

Pain killers will be more effective compared to other medications but it should not be often used and could be used only when the pain is untoleratable. commonly used painkillers are paracetamol, AI MEDICATIONS and Aspirin.

1. Paracetamol: it is available in all pharmacies and normal stores as it is the most common type of painkiller. It is more effective on the onset of headache and can be used as a second dose after four hours.
2. Anti-inflammatory medication: Ibuprofen is a type of anti-inflammatory medication available over the counter whereas the other naproxen requires prescription prescribed by doctors. But it has side effect such as stomach pain.
3. Aspirin: they can be effective in relieving pain but may cause stomach related side effects. So certain doctors will not provide prescription for aspirin.

PREVENTION FOR TENSION TYPE HEADACHE:

1. A headache diary: in dairy the patients should note when and which part headache occurs along with how severe the pain is so it will be helpful in identifying the headache triggers.
2. Stress relief and lifestyle changes : Relaxation techniques like meditation, yoga or other methods like acupuncture are effective can be used for relieving stress whereas regular exercise and a healthy balanced diet can be useful as lifestyle changes.
3. Medication: Amitriptyline, an antidepressant can be used for frequent and severe headaches as preventive measures which should be used only when described by doctors.

CLUSTER HEADACHE

INTRODUCTION:

Cluster headache is also called as bouts of frequent attacksmay last for weeks to months, usually it follows long period of relief during remission, attacks may stop for months or years. An cluster headache person might experience bouts of 1-8 headaches per day, also termed as cluster periods.

Although cluster headache is very painful it is not life threatening and with treatment can reduce the severity of headache. Short time treatment for this headache is specialized painkillers or oxygen therapy whereas long time treatment for cluster headache is lifestyle changes and prevention.

SYMPTOMS:

1. Cephalalgia: Is a type of dull, often occurring, moderate intense pain in the head region.
2. Trigeminal: Refers to trigeminal nerve which is responsible for sensation in the face and action such as biting and chewing. Cluster headache can affect either the left or right side of the head .
3. Autonomic: here the clustered headache will have its own body activities automatically for example- runny nose during cold and teary eyes during eye irritation or when dust particles fall on eyes.
4. Shrinking of the pupil in the eye
5. A drooping or swollen eyelid
6. Other symptoms like:

- A pale or flushed face

- Spreading of the pain to the face , head, and neck

TYPES OF CLUSTERED HEADACHE:

Episodic cluster headaches will occur every day for periods of weeks or months. Mostly if it occurs six to 12 weeks it is considered normal, for 80%of the patients. Rest of the time patient having episodic cluster headaches will experience remission with no cluster headache. Mostly patients or person affected by cluster headache will have it experienced in spring and fall.

Chronic cluster headaches will occur for 20% of patients. It means that the headache occurs whole year rather than occurring in periods or seasonal cycles, but period of remission will last for less than one month.

TRIGGERS:

* Seasonal changes: cluster headache is somewhat related to circadian rhythmicity and it occurs according to adjustment of body observing more or less light during day time.
* Weather changes
* Alcohol consumption
* Tobacco usage
* Stress
* Strong smells such as paint, petrol and perfume
* Temperature changes
* Preserved meats
* Elevated levels of histamine and serotonin

TREATMENT:

* Short term treatment is relieving headache attacks themselves.
* Long term treatment is to reduce the frequency of headache attacks.

Short term treatment:

Specialized treatment for cluster headache is to relieve pain in 15-30 minutes. it is called as abortive treatments because they relieve pain more quickly than other painkiller. They can be provided through nose by breathing which is called as vaporization or by injection into muscle or vein.

Oxygen therapy:

The treatment involves pure oxygen breathed in through a mask at the start of attack. The mask and cylinder of oxygen will be provided or given by the doctor. it is usually advised to breathe in 15 liters of oxygen per minute at the start of the attack and decrease it to six to eight liters per minute when the symptoms reduces. Even after the attack has got over the patient should breathe oxygen continuously for five to ten more minutes in order to prevent from the return of attack.

LONG TERM TREATMENT FOR CLUSTER HEADACHE

1. Transcutaneous vagus nerve stimulation

This treatment involves giving the patient small handheld device which they will use to treat cluster headache. This device works by emitting a low voltage electric current which when applied to the skin on the neck area near the vagus nerve it will stimulate it relieving tension and pain.

2.Sphenopalatine ganglion stimulation:

This sphenopalatine ganglion is a nerve bundle which is localized behind the nose region. In patients affected by cluster headache, low voltage electrical stimulation of the sphenopalatine ganglion can relieve pain of the cluster headache.general anaesthesia is used to implant this device, and a small incision is made in the upper gum on the side most affected . this implantation procedure takes around one hour and no visible scarring is present. The sphenopalatine ganglion will be stimu;lated not only during attacks but also between attacks to reduce the frequency of cluster headache.

PREVENTION:

* Mostly giving up tobacco products may therefor redue a persons chace of developing chs.
* Make lifestyle choices which would avoid most of the triggering factors.
* Preventive medications like Corticosteroids are used for two to three weeks compared to other therapies that take longer to start their effect , but this cannot be long used because of its side effects like increased sugar level, thinning of bones.
* Healthy life choices like relaxation, resting well, exercising regularly and staying dehydrated and quitting tobacco and cocaine related products.

REFFERENCES:

1. Migraine. “Headache Types.” August, 2014.
2. Mayo Clinic. “Headache: Definition.” May 4, 2016..
3. Migraine. “What is the difference between a primary and secondary headache?” March 18, 2011WebMD.
4. Mayo Clinic. “Migraine - Symptoms and causes.” April 26, 2017
5. Cluster headaches.” NHS Choices. 22 May 2017. Accessed: 14 March 2018.
6. “Cluster headache.” American Migraine Foundation. 28 November 2016
7. “Cluster headache.” Headache Australia. 2018..
8. “Cluster headaches.” Cleveland Clinic. 22 July 2014. Accessed: 14 March 2018.