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MIGRAINE : AN OVERVIEW

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ABSTRACT :

Migraine affects over one billion people annually across the planet, and isOne of the foremost common medicine disorders, with a high prevalence and morbidity,Especially among young adults and females. Migraine is related to a good vary ofComorbidities, that vary from stress and sleep disturbances to suicide. The advanced and for the most part unclear mechanisms of megrim development have resulted within the proposal of various social and biological risk factors, like secretion imbalances, genetic andEpigenetic influences, also as vas, medical specialty, and response diseases.This review presents a comprehensive review of the foremost up-to-date literature on theEpidemiology, pathophysiology , and risk factors, Diagnosis, Acute treatment and preventive treatment as well as lightness the gaps in our data.

KEYWORDS : Migraine, Epidemiology, Risk factors, Diagnosis, Types of Migraine, Acute treatment, preventive treatment

INTRODUCTION :

Migraine is a common condition, with a point prevalence of 20% in women and 8% in men. Prophylactic treatment may be helpful if the patient experiences frequent debilitating migraine headaches. Beta blockers, tricyclic antidepressants, and anticonvulsants have the best evidence of efficacy. Calcium channel blockers and non-steroidal anti-inflammatory drugs are also popular because they are well tolerated and inexpensive. We review migraine treatments with an emphasis on prevention.Therefore, guidelines for treating migraine attacks and preventing them with medication or behavioral therapy are very practically important. The purpose of this guideline is to optimize the treatment of acute migraine attacks and the prevention of migraine. The guidelines are evidence-based and take into account the clinical experience of the guideline authors and are a further development of the following guidelines and recommendations.

Deutsche Gesellschaft fur Neurologie (GermanSociety for Neurology; DGN) and DeutscheMigraine (GermanMigraine and Headache Society; DMKG) Guide-line Therapy of Migraine (2012) (Diener et al., 2012)

DMKG Guideline: Relaxation procedures andbehaviour-therapeutic interventions in the treatment of migraine. (Meyer B et al., 2017)

European Federation of Neurological Societies(EFNS) Guideline (2009) (Evers et al., 2009)

Report of the Quality Standards Subcommittee ofthe American Academy of Neurology and theAmerican Headache Society (2012) (Holland et al., 2012, Silberstein SD et al., 2012)

Guidelines of the Canadian Headache Society(2012) (pringsheim T et al., 2012)

Guidelines of the French Headache Society (2014)(Valade et al., 2014)

DEFINITION :

Migraine is a complex neurobiological disorder that has long been known. Migraine headaches are characterized by moderate to severe, often unilateral, throbbing headache attacks that increase in intensity with physical activity. (Headache classification Committee of the International headachesociety 2018) One-third of patients suffer from total cranial headache. Individual seizures include loss of appetite (most often), nausea (80%), vomiting (40-50%), photophobia (60%), sensitivity to noise (50%), and hypersensitivity to specific odors (10%) is accompanied. Signs of parasympathetic activation are seen in up to 82% of patients and are most commonly minor lacerations. (Riesco et al., 2016) For unilateral headaches, the side may be switched during or after each seizure. The strength of the attack can vary greatly from attack to attack. According to the definition of the International Headache Society (HIS), the attack time is 4 to 72 hours. In children, the seizures are shorter, there is no headache, and only severe nausea, vomiting, and dizziness may appear. (Maytal J. et al., 1997) Headache localization is more often bilateral.

HISTORY :

Migraine and associated pain have long plagued humans. (Friedman AP 1972) Many people suffer from migraines, including Julius Caesar, St. Paul, Thomas Jefferson, and Charles Darwin (Jones et al., 1999). 3000 year BC Anonymous Sumerian poet described a condition in which the headache was severe and the patient was ill and wanted to relieve the pain (Friedman AP. 1972, Silberstein et al., 2004). In Mesopotamia, it was believed that the evil spirits of Thie attacked the victims and caused severe headache . The skull was discovered during archaeological excavations, suggesting that doctors

recommend removing part of the patient's skull to relieve severe headaches (Friedman AP. 1972, Jones et al., 1999). Ancient Egyptian medicine (since 3000 BC) clearly described migraine and neuralgia, as well as sudden and severe pain (Silberstein et al., 2004). Hippocrates (460-370 BC), the father of medicine, was the first to scientifically record clinical observations of migraine, thereby freeing the disease from superstition . Hippocrates explained the onset of a bright glow in the right eye followed by severe head pain, eventually spreading throughout the area (Silberstein et al., 2005, Huntsinger et al., 1927). Aretaeus (AD 30–90) provided one of the first formal classifications of headaches (Stevens et al., 1993)

He divided headaches into three major types: (a) Cephalagia Headaches - mild and transient headaches, (b) Cephalea-types of chronic and severe headaches, and (C) heteroclania - unilateral headaches, Often accompanied by sweating, nausea, vomiting, photophobia, eye discomfort, sweating, and altered perceived scent (Stevens et al., 1993, Magiorkinis et al., 2009). Galen (AD 129-216) writes about severe pain affecting almost half of the head (Guerrero—petal et al., 2014, Trompoukis et al., 2007). Galen, like Hippocrates, believed that migraine headaches were caused by steam rising from the stomach to the head (Guerrero—petal et al., 2014, Trompoukis et al., 2007). A renowned Iranian doctor , Razi (also known as the Latin name Rhazes, AD 854–925)(Salazar C 2017, Meyer hot 1935)), dedicated the entire chapter of his book, Headache, which wrote about the symptoms and treatment of migraine. His study details one of the various types of headaches currently known in modern medicine as migraine clusters, and also suggests that severe sudden headaches may be the first sign of a stroke.(Shoja et al., 2007).

Ibn Sina, also known in the West as Avicenna (980-1037), another well-known Iranian doctor (Gohiman 1986, Afshar et al., 2020)) wrote a chapter on headaches in his book "The Canon of Medicine". The whole head, this type of headache was severe, exacerbated by activity, and was also characterized by photophobia and noise phobia (Zargaran et al., 2016, Avicenna et al., 2005). In the 17th century, Thomas Willis (1621–1675) published the hypothesis that "immigrants" were due to the dilation of blood vessels in the head (the first expression of blood vessel theory) (Rose FC 2009). In 1873, Edward Living suggested that migraine headaches were due to "nerve storms originating from the thalamus" (Centurion et al., 2003). In 1898, Mobius stated that "the substance is the master and the circulatory system is the servant," and that both brain and vascular dysfunction are required to cause a migraine attack (Centurion et al., 2003, Edmeads J 1991)

EPIDEMIOLOGY :

Migraine is one of the most common types of headache. The annual prevalence of migraine is between 10% and 15%. (Lipton RB et al., 2007) The one-year prevalence of migraine is 3-7%. (Loder et al., 2015, Sivaraman et al., 2010) Boys and girls are affected at about the same rate. The highest prevalence is found between the ages of 20 and 50. At this stage of life, women are affected up to three times more often than men. The difference in prevalence between men and women is largest around the age of 30. (Campbell et al., 2010)

In 2019, the national age-standardized incidence rates ofMigraine ranged from 692.6 to 1,528.4 cases per a hundred,000, withItaly [1,528.4 (95% UI: one,345.4–1,709.3)] and Kingdom of Norway [1,515.7(95% UI: 1,333.8–1,693.4)] having the very best rates. In distinction,The lowest rates were discovered for Yaltopya [692.6 (95% UI:605.2–776.7)] and Djibouti [737.2 (95% UI: 623.5–848.9)]. The global prevalence of head ache has redoubled wellOver the last 3 decades. Per the world BurdenOf sickness (GBD) 2019 study, the calculable world prevalence ofMigraine redoubled from 721.9 million (95% UI: 624.9–833.4) in1990 to 1.1 billion (95% UI: zero.98–1.3) in 2019. The proportionChange within the world age-standardized prevalence rate and yearsLived with incapacity (YLDs), from 1990 to 2019, were 1.7 (95%UI: 0.7–2.8) and 1.5 (95% UI: −4.4 to 3.3), severally. During this era, the very best will increase within the age-standardizedPrevalence per a hundred,000 population were in East Asia (7.9% (95%UI: 4.3–12%) and range of mountains geographical area [6.7% (95% UI: two.1–11.9%)], whereas the most important decreases were in High-income NorthAmerica [−2.2% (95% UI: −5.3 to 1.1%)] and geographic region[−2.2% (95% UI: −3 to −1.4%)] (7). Moreover, the age-Standardized YLD rate of head ache additionally redoubled from 517.6(95% UI: eighty two.0–1169.1) in 1990 to 525.5 (95% UI: seventy eight.8–1194.0)In 2019.

The prevalence of head ache was higher in females, than inMales, across all age teams. In 2019, the world age-standardizedPrevalence rate in females and males were seventeen,902.5 (95% UI:15,588.3, 20,531.7) and 10,337.6 (95% UI: eight,948.0, 12,013.0) per100,000 populations, severally. The highest incidence rate and range of incident cases ofMigraine were within the cohort 10–14 years, in each femalesAnd males. In 2019, whereas the amount of YLDs startedIncreasing from birth, they peaked within the 30–34 cohort andThen bit by bit declined for each sexes. Socioeconomic standing didn't appear to be related toThe burden of head ache, since no clear association was reported between the socio-demographic index (SDI) and also the head acheYLD rate. (Safiri S,et al.,2022)

PATHOPHYSIOLOGY OF MIGRAINE -

Regarding the pathophysiology of migraine (Fukuchi et al. 1993), vascular theory has traditionally been widely believed, in which vasoconstriction occurs during the aura and then dilation leads to headache. However, two theories about the pathophysiology of migraine have recently been proposed and are drawing attention. One is nerve theory (Olesen et al., 1981), which states that hyper excitability of nerve cells in the cerebral cortex is the cause of migraine. The other is the trigeminal neurovascular theory, which focuses on the relationship between the trigeminal nerve and the intracranial blood vessels.

Moskowitz (Moskowitz M A 1984) emphasized the relationship between the trigeminal nerve and intracranial blood vessels, especially the dural blood vessels, and pointed out that myelinated C fibers derived from the trigeminal ganglion are distributed in the dural blood vessels. In addition, they have shown that when the trigeminal nerve is electronically or chemically stimulated, neurological inflammation occurs in the dural blood vessels. Therefore, they thought that neurological inflammation due to the trigeminal neurovascular system could serve as a migraine model. As a result, the trigeminal neurovascular theory was proposed.A summary of this theory is shown in Fig.

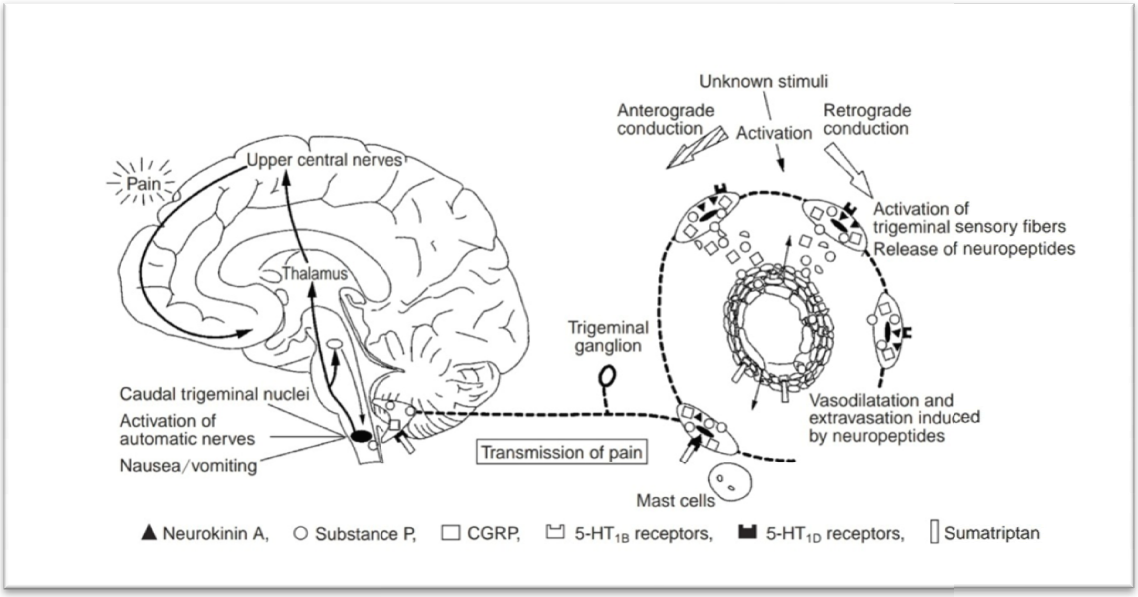

Fig. Pathophysiology of Migraine

In other words, certain stimuli include the trigeminal axon around the dural blood vessels, and vasoactive neuropeptides (substance P (SP), neurokinin A (NKA), calcitonin gene-related peptide (CGRP), etc.) Affects. Will be released. These then cause neurological inflammation (vasodilation, plasma protein leakage and mast cell degranulation). This causes both anterograde and retrograde conduction in the trigeminal nerve.

According to this theory, anterograde conduction reaches the trigeminal nucleus and is relayed to the thalamus and cerebral cortex. This message is felt as pain. Retrograde conduction, on the other hand, further activates the release of vasoactive neuropeptides around the trigeminal nerve. In addition, sumatriptan has been shown to act on 5HT1B receptors in intracranial vascular smooth muscle to constrict blood vessels. It also suppresses the release of neuropeptides by binding to 5HT1D receptors on the trigeminal nerve around blood vessels. In addition, Moskowitz published an article in which this unknown stimulus is believed to be the inhibition of cortical transmission discovered during the aura. This theory seems to be an organic combination of traditional vascular theory and neurological theory. (Goadsby PJ, 2000)

DIAGNOSIS :

The Diagnosis is based on a medical history and discreet neurological examination findings (see the guideline “Diagnosis of Headache and Additional Technical Examinations” for more information). Headaches with an abnormal clinical appearance (eg, to rule out subarachnoid hemorrhage) and headaches with prominent persistent neurological or psychopathological features require additional diagnostic procedures, especially imaging procedures.

COMPLICATION:

Status Migrainosus :

Most migraines sometimes linger between four and seventy two hours. Standing migrainosus, on the opposite hand, could be a relentless attack that lasts for quite three days. It will leave you feeling drained or perhaps disabled. The pain and nausea will keep you from obtaining enough sleep or cause you to dehydrated from throwing up. You will would like care at the hospital. This sort of head ache typically comes on once you are taking an excessive amount of headache medication.

Migrainous Infarction :

Also referred to as a migrainous stroke, this can be a rare complication that happens principally in younger ladies. Blood vessels to the brain will get narrowed and bring to a halt the atomic number 8 provide. A migrainous stroke will hit suddenly associated is an emergency. It continually happens with associate aura, a collection of bizarre sensations like flashes of sunshine, blind spots, and tingling hands or face.Women beneath forty five UN agency smoke and take contraception pills are possibly to own a stroke with a cephalalgia.

Persistent Aura Without Infarction :

One in four individuals with migraines will have aura. However typically it lingers for over per week once associate degree attack. Rarely, you'll be able to have aura and symptoms like bother respiratory and symptom for months or perhaps years.The signs will appear on the point of those of a stroke, or haemorrhage within the brain, however with none actual haemorrhage. Pathology is another word for stroke.

Migraine-Triggered Seizure:

This rare case will seem like AN convulsion. It happens throughout or presently when a cephalalgia with aura. Encephalopathy and cephalalgia generally go along. However researchers don't absolutely perceive why.

RISK FACTOR :

Extensive epidemiological studies have identified several risk factors for migraine attacks. For example, in a web-based study of 15,133 migraine patients and 77,453 controls, insomnia, depression, anxiety, digestive ulcer disease, gastrointestinal bleeding, angina, and epilepsy were more migraine than controls. It was shown to be significantly more common in patients ($p < 0.001$) ($p < 0.001$) (Buse et al., 2020). The intensity and frequency of pain is associated not only with inflammation, but also with biological and psychological disorders (Buse et al., 2020). Given the complex nature of migraine, it is difficult to distinguish between migraine risk factors, triggers, and consequences (Lipton et al., 2005). Educating patients about the causes and exacerbations can reduce the frequency and severity of seizures. In addition, some interventions such as B. aerobic exercise, reduce migraine attack duration and pain intensity, reduce migraine days / month. However, based on current knowledge, migraine has a variety of risk factors, triggers, and comorbidities (Lipton et al., 2005). Different risk factors associated with migraine development and progression.

Biological Factors :

1. Hormonal Imbalances- EstrogenDysregulation, Cortisol Dysregulation
2. Demographic Factors – Advancing age, Female sex
3. Metabolic factors – Obesity, Diabetes, Hypertension
4. Genetic and epigenetic Factors –MTDH gene, MEF2D gene, PRDMI6 gene

Psychological Factors : Anxiety

1. Phobia
2. Panic, Stress

Miscellaneous : Lower level of education

1. Lower socioeconomic status

COMMONMIGRAINE TRIGEERS:

Alcohol,Artificialsweeteners (e.g., aspartame),Caffeine (overconsumption or acute,Withdrawal from regular use),Delayed/missed meals,Exercise, Foods (e.g., chocolate, soft cheese),Light,Odours (e.g., perfumes),Oral contraceptives,Sleep disturbances (e.g., obstructive Sleep apnea, insomnia),Stress,

TYPESOF MIGRAINE :

1)Migraine Without aura
2)Migraine with aura
3)Episodic Migraine
4)Chronic Migraine
5)Medication overuse headache.

Migraine Without aura :

A. At least five attacks fulfilling criteria B, C, and D
B. Attack lasting 4 to 72 hours (untreated or unsuccessfully treated).
C. Having at least two of these characteristics: aggravation by orCausing avoidance of routine physical activity (e.g., walking or Climbing stairs), moderate or severe pain intensity, pulsating quality, unilateral location
D. Having at least one of these conditions during the headache:Nausea and/or vomiting, phonophobia or photophobia

Migraine with Aura :

A. At least two attacks fulfilling criteria B and C
B. Having one or more of these fully reversible aura symptoms: Brainstem, motor, retinal, sensory, speech and/or language, visual
C. Having at least two of these characteristics: at least one aura symptoms spreads gradually over at least 5 minutes and/or two or more Symptoms occur in succession; each individual aura symptom lasts 5 to 60 minutes; at least one aura symptom is unilateral; the aura is Accompanied or followed within 60 minutes by headache.

Episodic Migraine :

Characterized by those with migraine who have zero to 14 headache Days per month.

Chronic Migraine-

A. Headaches at least 15 days per month for more than 3 months And fulfilling criteria B and C.
B. Occurring in patients with at least five attacks fulfilling criteria in The Migraine with Aura or Migraine without Aura sections.
C. For at least 8 days per month for more than 3 months, fulfills any Of the following:
    1. Criteria C and D for Migraine without Aura section
    2. Criteria B and C for Migraine with Aura section
    3. Believed by the patient to be migraine at onset and relieved by Triptan or ergot derivative.

STRATEGIESOF MIGRAINE TREATMENT –

Treatment can be acute or prophylactic. Acute treatment is started during the seizure to relieve pain and disability and stop the progression of the seizure. Prophylactic treatment is maintained for months or years to reduce the frequency, severity, and duration of seizures. (Silberstein et al., 2002)

ACUTE MIGRAINE TREATMENT :

Many medicines are available to treat migraine, and the choice depends on the severity and frequency of the headache. Drugs in these categories include specific and non-specific treatments. Specific treatments such as ergotamine-containing compounds, DHE, and triptans are only effective in treating migraine and related disorders. Non-specific treatments are effective treatments for all pain disorders and include the combination of nonsteroidal anti-inflammatory drugs (NSAIDs), analgesics, opioids, neuroleptics / antiemetics, and corticosteroids. (Diener et al., 1998)

TRIPTANS :

Triptans as a class represent significant advances in the therapeutic treatment of migraine. These agents have been described as receptor-specific agonists for serotonin or 5HT receptors. In particular, they are selective 5HT1B / 1D agonists that have the greatest affinity for these receptors. Blocking 5HT1 receptors has been shown to rapidly relieve migraine headaches. (SilbersteinSD, 2000). Triptans provide a rapid onset of action (15 minutes to 1 hour, depending on the formulation) compared to non-specific therapies including analgesics and NSAIDs, and are very effective in relieving migraine symptoms. It is effective and has a favorable side effect profile. All active ingredients in this class have proven therapeutic effects. In the majority of patients, side effects are mild and short-lived. Side effects include chest tightness, hot flushes, dizziness, light-headedness', and nausea. Patients at risk for coronary artery disease, diabetes, obesity, severe uncontrolled hypertension, or hypercholesterolemia should be evaluated prior to administration of triptans. (Silberstein et al., 1998).As a group, triptans may affect migraine by several proposed mechanisms, including: 1) meninges, dural, and brain that are dilated and edematous during migraine attacks: Vasoconstriction of the 5HT1B receptor in the arterial vascular smooth muscle. 2) Activation of the 5HT1 receptor at the peripheral end of the caudal trigeminal nucleus results in neurosuppression and blocking of vasoactive neuropeptide release in the trigeminal sensory nerve. Suppression occurs by direct action on 5HT1D receptors at the central trigeminal nerve endings. (Hargreaves et al., 1999)

There are several triptans currently available in the United States. The first is sumatriptan (Imitrex®). The oral formulation is available in 25mg, 50mg and 100mg doses as either an oral tablet or a rapidly dissolving tablet. Since its introduction in the early 1990s, over 400 million doses of sumatriptan have been given.

• Zolmitriptan (Zomig®), the second product in the triptan class, has a longer half life than Sumatriptan and a more rapid Tmax. It can be used as a tablet, a tablet that collapses in the mouth, and a nasal spray.

• Naratriptan (Amerge®) has a longer half-life and a lower recurrence rate than sumatriptan. Naratriptan is less effective than sumatriptan and is thought to have minimal side effects.

• Rizatriptan (Maxalt®) has a faster onset of action and a shorter Tmax of 1 hour than oral sumatriptan. This product is available in the form of tablets and meltable wafers.

• Almotriptan (Axert®) is available as an oral tablet and has a good side effect profile.

• Frovatriptan (Frova®) is available as a tablet. Like naratriptan, it has a long half-life, a low incidence of side effects, and a low recurrence rate.

• Eletriptan (Relpax®) is available as an oral tablet. (Ferrari et al., 1999)

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS :

According to Cochrane's review, ibuprofen is superior to placebo at all doses of 200 mg to 600 mg, and pain relief in 2 hours in patients with moderate to severe early painful acute migraine. And there is continuous pain relief in 24 hours. The NNT to achieve painless results in 2 hours was 9.7 at 200 mg and 7.2 at 400 mg. (Rabbie et al., 2013)

Naproxen has also been found to provide 2 hours of pain relief for patients with acute migraine compared to placebo. The painless NNT in 2 hours was 11. Results did not change at doses of 500 mg and 825 mg. (Derry et al., 2013)

Diclofenac potassium 50 mg has a relative benefit over placebo, relative risk (RR) 2.0 (95% confidence interval (CI) 1.6-2.6), NNT = 8.9 for relief from pain after 2 hours in patients with acute migraine. (Derry et al., 2013)

Naproxen and ibuprofen were also more effective in alleviating migraine-related symptoms (such as nausea, photophobia, phonophobia, and dysfunction) compared to placebo. (Rabbie et al., 2013, Derry et al., 2013)

Ibuprofen is the only NSAID which is licensed for patients with acute migraine. Ibuprofen (400 mg) is recommended as first-line treatment for patients with acute migraine. If Ineffective, the dose should be increased to 600 mg. (Sachaefer et al., 2014)

ASPIRIN :

A Cochrane evaluation of thirteen studies (4,222 participants) pronounced that aspirin 900 mg and aspirin 1,000 mg had been powerful in accomplishing ache loose at hours as compared to placebo (NNT=8.1). For sustained ache alleviation at 24 Hours aspirin 1,000 mg had an NNT of 6.6 as compared to placebo.(Kirthi et al., 2013)

Aspirin by myself had comparable efficacy to sumatriptan 50 mg, and sumatriptan a hundred mg became advanced to aspirin And metoclopramide combined.(Kirthi et al., 2013)

Associated signs and symptoms of nausea, vomiting, photophobia (NNT=7.7) and phonophobia (NNT=6.6) had been decreased with the aid of using aspirin while as compared to placebo. The addition of metoclopramide in addition decreased nausea (NNT=2.6) and vomiting.(Kirthi et al., 2013)

Aspirin is a capacity gastrointestinal irritant and can motive ulcers or gastrointestinal bleeding, but unfavourable outcomes from short-time period use are often slight and transient.(Kirthi et al., 2013)

Aspirin must now no longer be utilized in sufferers below sixteen years of age because of the hazard of Reye`s syndrome. The use of aspirin for the duration of pregnancy, in particular of intermittent excessive doses, must be avoided.Aspirin is contraindicated for the duration of the 0.33 trimester of Pregnancy.(Joint Formulary Committee 2017)

Aspirin (900 mg) is recommended as first-line treatment for patients with acute migraine. Aspirin, in doses for migraine, is not an analgesic of choice during pregnancy and should not be used in the third trimester of Pregnancy (JointFormulary Committee 2017)

ANTIEMETICS :

Metoclopramide 10 mg (oral) in combination with aspirin 900 mg had similar efficacy to 100 mg sumatriptan in achieving the outcome of pain free at two hours.(Kirthi et al., 2013) Similar results were found for paracetamol 1,000 mg Combined with metoclopramide 10 mg versus sumatriptan.(Derry et al., 2013) However, aspirin and metoclopramide provided Significantly better relief of associated symptoms, with an NNT of 2.6 (95% CI 2.1 to 3.1). It was particularly Beneficial in reducing vomiting, NNT=2.1 (95% CI 1.5 to 3.7)(Kirthi et al., 2013).

Metoclopramide (10 mg) or prochlorperazine (10 mg) can be considered in the treatment of Headache in patients with acute migraine. They can be used either as an oral or parenteral formulation depending on presentation and setting. Metoclopramide (10 mg) or prochlorperazine (10 mg) should be considered for patients presenting with migraine-associated symptoms of nausea or vomiting. They can be used either as an oral or parenteral formulation depending on presentation and setting.Metoclopramide should not be used regularly due to the risk of extrapyramidal side effects.(Friedman et al., 2011)

Preventive treatment -

BETA BLOCKERS:

A well-conducted systematic review identified a large number of trials on the use of beta blockers for prophylaxis of migraine, mostly from the 1980s. The individual trials were rated as low quality and of short Duration (<3 months).46 Propranolol (80–160 mg) reduced the frequency of episodic migraine by ≥50% compared to placebo (NNT=4, 95% CI 3 to 7). (Shamliyan T A. et al., 2013) Metoprolol (200 mg daily, slow release) reduced migraine severity, but no consistent benefits in reduction of migraine frequency or use of acute analgesics was shown. Atenolol 50–200 mg daily was reported to reduce frequency of episodic migraine and use of acute therapies. (Shamliyan T A. et al., 2013) Propranolol (80–160 mg daily) is recommended as a first-line prophylactic treatment for patients With episodic or chronic migraine.(Shamliyan T A et al., 2013)

TOPIRAMATE:

Three systematic reviews reported the efficacy of topiramate and placebo in patients with temporary and chronic migraine headaches. Many patients have a 50% or greater reduction in headache frequency (RR 2.02, 95% CI 1.57 to 2.60; NNT = 4, 95% CI 3 to 6), a reduction in headache every 28 days, and a reduction in quality of life. Reported an improvement in quality. (Linde M et al., 2013) In patients with chronic migraine, low-quality

evidence is more effective because topiramate reduces the frequency of symptoms associated with the number of days of migraine each month and reduces monthly migraine attacks by 25% compared to placebo. It suggests that it is a target. Topiramate also improved quality of life and migraine-related disability scores. (Shamliyan TA et al., 2013)

Topiramate (50-100 mg daily) is recommended as a prophylactic treatment for patients with temporary or chronic migraine headaches. Before starting treatment, women of childbearing potential may seek advice on the risks associated with topiramate during pregnancy, the need for effective contraception, and migraine prevention during pregnancy or planning. You need to get counseling.(Shamliyan TA et al., 2013)

TRICYCLIC ANTIDEPRESSANTS:

According to a systematic review, patients with temporary migraine (mean 4.7 migraine per month) treated with tricyclic antidepressants (TCA) had a 1.4 reduction in headache per month. (Jackson et al., 2010) The study period varied from 4 to 24 weeks. It is classified as a success with a high risk of bias. (Jackson et al., 2010) The average dose of TCA used was 50% of the maximum dose (eg,the dose range of amitriptyline was 10 mg to 150 mg and the average pooled dose was 80 mg). In most studies, the dose was titrated. There was some evidence that higher doses provided greater benefit, but the difference between higher and lower doses was not significant. Patients with temporary migraine taking TCA had an 80% chance of improving their headache by 50% compared to placebo (RR 1.80, 95% CI 1.24 to 2.62). Continued treatment with TCA reduced the frequency of headaches slightly and persistently. (Jackson et al., 2010)

Amitriptyline (25-150 mg per night) should be considered as a prophylactic treatment for patients with temporary or chronic migraine headaches. Patients who are intolerant to amitriptyline should consider tricyclic antidepressants, which have less sedative effects. (Jackson et al.,2010)

CANDESARTAN:

A systematic review identified two moderate-quality RCTs demonstrating the efficacy of candesartan (16 mg)(Jackson et al., 2015) One study reported a 26% reduction in the number of days of headaches. (Tronvin et al., 2015) Other studies have shown that candesartan is as effective as propranolol 160 mg with the side effect of reducing migraine days by more than 50% (percentage of respondents: 43% for candesartan, 40% for propranolol, 23% for placebo). Candesartan is generally well tolerated, and early study data suggest that the incidence of side effects is not increased compared to the placebo rate. (Tronvin et al., 2015)

SODIUM VALPROATE:

In patients with temporary migraine, sodium valproate is more effective than placebo, reducing the frequency of headaches by more than 50% in 8-12 weeks (RR 2.83, 95% CI 1.27-6.31; NNT = 3.95% -KI 2-9). Pooled data from two small studies (n = 63) at doses ranging from 400 to 1500 mg daily. (63) In a pooled analysis of two small studies, there was no difference in efficacy compared to flunaridine, and sodium valproate 500 mg was not as effective as high-dose topiramate (400 mg)(Linde M et al. 2013)

Sodium valproate (400-1,500 mg daily) can be considered as a prophylactic treatment for patients with temporary or chronic migraine headaches. (Linde M et al., 2013)

CONCLUSION :

In conclusion, we've seen that headache is neurovascular headache.A Significant geneticomponent to predisposition to head ache exists, and therefore the purposeful consequences of the biological science square measure getting down to be explored. The biology of headache is healthier though not utterly understood. This exaggerated understanding offer nice opportunities to still improve treatment. The complicated and complex nature of headache is mirrored. In the presence of a range of risk factors and triggers agents. The choice of a drug ought to be based mostly upon level of proof for effectivity, adverse impact profile, and patient comorbidities so as to treat multiple disorders at the time to enhance adherence.

REFERENCE:

1. Diener HC. Leitlinien fu¨ r Diagnostik und Therapie in Der neurologie: herausgegeben von der Kommission Leitlinien Der DGN. Stuttgart: Georg Thieme Verlag, 2012.
2. Kropp P, Meyer B, Dresler T, et al. Entspannungsverfahren Und therapeutische Interventionen zur Behandlung der Migra¨ne. Leitlinie der Deutschen Migra¨ne- und Kopfsch-Merzgesellschaft (German). [Relaxation techniques andBehavioural therapy for the treatment of migraine: guidelinesFrom the german migraine and headache society]. DerSchmerz [Schmerz] 2017; 31(5): 433–447.
3. Evers S, Afra J, Frese A, et al. EFNS guideline on the drug treatment of migraine – revised report of an EFNS task force.Eur J Neurol 2009; 16(9): 968–981.
4. Holland S, Silberstein SD, Freitag F, et al. Evidence-basedGuideline update: NSAIDs and other complementary treatments' for episodic migraine prevention in adults: report of the quality standards subcommittee of the American academy of neurology and the American headache society. Neurology2012; 78(17): 1346–1353.
5. Silberstein SD, Holland S, Freitag F, et al. Evidence-based guideline update: pharmacologic treatment for episodic migraine prevention in adults: report of the quality standards subcommittee of the American academy of neurology and theAmerican headache society. Neurology 2012; 78(17):1337–1345.

6. Pringsheim T, Davenport W, Mackie G, et al. Canadian headache society guideline for migraine prophylaxis. Can J Neurol Sci 2012; 39(2 Suppl 2): S1–S59.
7. Lanteri-Minet M, Valade D, Geraud G, et al. Revised FrenchGuidelines for the diagnosis and management of migraine inAdults and children. J Headache Pain 2014; 15: 2.
8. Headache Classification Committee of the International headache Society (HIS). The International Classification of Headache Disorders ICHD-3, 3rd edition. The Committee Cephalalgia 2018; 38(1): 1–211.
9. Riesco N, Perez-Alvarez AI, Verano L, et al. Prevalence ofCranial autonomic parasympathetic symptoms in chronicMigraine: usefulness of a new scale. Cephalalgia 2016;36(4): 346–350.
10. Maytal J, Young M, Shechter A, et al. Pediatric migraine and the International Headache Society (HIS) criteria.Neurology 1997; 48(3): 602–607
11. Friedman AP. The headache in history, literature, and legend. Bull N Y AcadMed. (1972) 48:661.
12. Jones J. Great pains: famous people with headaches. Cephalalgia.(1999)19:627–30.Doi:10.1046/j.14682982.1999.019007627.x
13. Silberstein SD, Young WB. Migraine and Other Headaches. New York, NY:Demos Medical Publishing (2004).
14. Silberstein SD. Historical aspects of headache. Atlas Mig Other Head. (2005)2005:14-5. Doi: 10.1201/9780203696569
15. MC CLURE CW, Huntsinger ME. Observations on migraineBostMed surgj. (1927)196:270–3.Doi: 10.1056/NEJM192702171960703
16. Merikangas KR, Stevens DE, Angst J. Headache and personality: results ofA community sample of young adults. J Psychiatric Res. (1993) 27:187–96.Doi: 10.1016/0022-3956(93)90007O
17. Magiorkinis E, Diamantes' A, Mitsikostas D-D, Androutsos G. Headaches In antiquity and during the early scientific era. J Neurol. (2009) 256:1215. Doi: 10.1007/s00415-009-5085-7
18. Guerrero-Peral ÁL, de Frutos González V, Pedraza-Hueso MI. Galeata: Chronic migraine independently considered in a medieval headache Classification. J Headache Pain. (2014) 15:1–5. Doi: 10.1186/1129-2377-15-16
19. Trompoukis C, Vadikolias K. The "byzantine classification" of Headache disorders. Headache J Head Face Pain. (2007) 47:1063–8. Doi: 10.1111/j.1526-4610.2007.00813.x
20. Salazar C. Continuity and innovation in paul of aegina's Chapters on headaches and migraines. Coll Rec. (2017) 4:175. Doi: 10.1515/9781501502538-009 24. Meyerhof M. Thirty-three clinical observations by Rhazes (circa 900 AD). Isis. (1935) 23:321–72. Doi: 10.1086/346968
21. Tubbs RS, Shoja MM, Loukas M, Oakes WJ. Abubakr muhammad ibn Zakaria razi, rhazes (865–925 AD). Child Nerv Syst. (2007) 23:1225–6. Doi: 10.1007/s00381-007-0368-8
22. Gohlman WE. The Life of Ibn Sina. Suny Press (1986).
23. Afshar A, Steensma DP, Kyle RA. Ibn sina (Avicenna): The Prince of physicians. Mayo Clin Proc. (2020) 95:e31–e32. Doi: 10.1016/j.mayocp.2020.01.023
24. Zargaran A, Borhani-Haghighi A, Faridi P, Daneshamouz S, Mohagheghzadeh A. A review on the management of migraine In the avicenna's canon of medicine. Neurol Sci. (2016) 37:4718.Doi: 10.1007/s10072-016-2498-9
25. Avicenna H. Al-Qanon fi al-Tibb (Canon on medicine.), vol. 2. BeirutLebanon: Alalami Library Publication (2005).
26. Abokrysha N. Ibn Sina (Avicenna) on pathogenesis of migraine comparedWith the recent theories. Headache J Head Face Pain. (2009) 49:923–7.Doi: 10.1111/j.1526-4610.2009.01394.x
27. Rose FC. An historical overview of British neurology. Handbook Clin Neurol.(2009) 95:61328. Doi: 10.1016/S0072-9752(08)02139-8
28. Villaln C, Centurin D, Valdivia L, de Vries P, Saxena PR. Migraine:Pathophysiology, pharmacology, treatment and future trends. Curr VascPharmacol. (2003) 1:71–84. Doi: 10.2174/1570161033386826
29. Edmeads J. What is migraine? Controversy and stalemate in migrainePathophysiology. J Neurol. (1991) 238:S2–5. Doi: 10.1007/BF01642898
30. Lipton RB, Bigal ME, Diamond M, et al. Migraine prevalence, disease burden, and the need for preventive therapy.Neurology 2007; 68(5): 343–349.
31. Stovner L, Hagen K, Jensen R, et al. The global burden ofHeadache: a documentation of headache prevalence and disability worldwide. Cephalalgia 2007; 27(3): 193–210.
32. Yoon MS, Katsarava Z, Obermann M, et al. Prevalence of Primary headaches in Germany: results of the GermanHeadache Consortium Study. J Headache Pain 2012; 13(3):215–223.
33. Stovner LJ and Andree C. Prevalence of headache in Europe: A review for the Eurolight project. J Headache Pain 2010; 11(4): 289–299.
34. Burch RC, Loder S, Loder E, et al. The prevalence and burden of migraine and severe headache in the United States: Updated statistics from government health surveillance studies. Headache 2015; 55(1): 21–34.
35. Abu-Arafeh I, Razak S, Sivaraman B, et al. Prevalence ofHeadache and migraine in children and adolescents: a systematic review of population-based studies. Dev Med Childneurol 2010; 52(12): 1088–1097.
36. Victor TW, Hu X, Campbell JC, et al. Migraine prevalence by age and sex in the United States: a life-span study. Cephalal-Gia 2010; 30(9): 1065–1072.
37. Araki, N. and Fukuuchi, Y.: Pathophysiology of migraine. Internal Medicine 1993; 82: 19–23. (in Japanese)
38. Olesen, I., Larsen, B. and Lauritzen, M.: FocalHyperemia followed by spreading ooligemia and impaired activation of rCBF in classicMigraine. Ann Neurol 1981; 9: 344–352.
39. Moskowitz, M.A.: The neurobiology of vascular head pain. Ann Neurol 1984; 16: 157–168.
40. Goadsby, P.J.: The pharmacology of headache.Prog Neurobiol 2000; 62: 509–525.

41. Buse DC, Reed ML, Fanning KM, Bostic R, Dodick DW, Schwedt TJ, et al.Comorbid and co-occurring conditions in migraine and associated risk of increasing headache pain intensity and headache frequency: results of the migraine in America symptoms and treatment (MAST) study. J HeadachePain. (2020) 21:23. Doi: 10.1186/s10194-020-1084-y
42. Lipton RB, Bigal ME. Migraine: epidemiology, impact, and risk factors for progression. Headache J Head Face Pain. (2005) 45(Suppl. 1):S3–13. doi: 10.1111/j.1526-4610.2005.4501001.x
43. Silberstein SD, Goadsby PJ. Migraine: preventive treatment. Cephalalgia. 2002;22(7):491-512.
44. Diener HC, Kaube H, Limmroth V. A practical guide to the management and prevention of migraine drugs. 1998;56(5):811-824.
45. Silberstein SD. Practice parameter: evidence-based guidelines for migraine headache (an evidence- Based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology. 2000;55(6):754-762.
46. Silberstein SD, Lipton RB, Goadsby PJ. Headache in Clinical Practice. Oxford, England: Isis Medical Media; 1998.
47. Hargreaves RJ, Shepheard SL. Pathophysiology of migraine—new insights. Can J Neuro Sci.1999;26(suppl 3):S12-S19.
48. Ferrari MD, Roon KI, Lipton RB, Goadsby PJ. Oral triptans (serotonin 5-HT(1B/1D) agonists) in acute migraine treatment: a meta-analysis of 53 trials. Lancet. 2001;358(9294):1668-1675. Worldwide Product Safety and Pharmacovigilance Document. December 1999.
49. Rabbie R, Derry S, Moore RA. Ibuprofen with or without an Antiemetic for acute migraine headaches in adults. Cochrane Database of Systematic Reviews 2013, Issue 4.
50. Law S, Derry S, Moore RA. Naproxen with or without an antiemetic For acute migraine headaches in adults. Cochrane Database of Systematic Reviews 2013, Issue 10.
51. Derry S, Rabbie R, Moore RA. Diclofenac with or without an Antiemetic for acute migraine headaches in adults. Cochrane Database of Systematic Reviews 2013, Issue 4.
52. Schaefer C, Peters PWJ, RK. M. Drugs During Pregnancy and Lactation. 3rd edition. London: Academic Press; 2014.
53. Kirthi V, Derry S, Moore RA. Aspirin with or without an antiemetic For acute migraine headaches in adults. Cochrane Database of Systematic Reviews 2013, Issue
54. Joint Formulary Committee. Guidance on prescribing. In: British National Formulary (online) London: BMJ Group and Pharmaceutical Press. [cited 22 Nov 2017].
55. Derry S, Moore RA. Paracetamol (acetaminophen) with or without An antiemetic for acute migraine headaches in adults. Cochrane Database of Systematic Reviews 2013, Issue 4.
56. Friedman BW, Mulvey L, Esses D, Solorzano C, Paternoster J, Lipton RB, et al. Metoclopramide for acute migraine: A dose-finding Randomized clinical trial. Ann Emerg Med 2011;57(5):475-82.
57. Friedman BW, Esses D, Solorzano C, Dua N, Greenwald P, Radulescu R, et al. A Randomized Controlled Trial of Prochlorperazine Versus Metoclopramide for Treatment of Acute Migraine. Ann Emerg Med 2008;52(4):399-406.
58. Shamliyan TA, Kane RL, Taylor FR. Migraine in adults: preventive pharmacologic treatments. Rockville, MD: Agency for Healthcare Research and Quality (AHRQ); 2013. [cited 22 Nov 2017].
59. Linde M, Mulleners WM, Chronicle EP, McCrory DC. Topiramate for the prophylaxis of episodic migraine in adults. Cochrane Database of Systematic Reviews 2013, Issue 6.
60. Jackson JL, Shimeall W, Sessums L, Dezee KJ, Becher D, Diemer M, Et al. Tricyclic antidepressants and headaches: systematic review and meta-analysis. BMJ 2010;341:c5222.
61. Jackson JL, Cogbill E, Santana-Davila R, Eldredge C, Collier W, Gradall A, et al. A Comparative Effectiveness Meta-Analysis of Drugs for the Prophylaxis of Migraine Headache. PLoS ONE 2015;10(7):e0130733.
62. Tronvik E, Stovner LJ, Helde G, Sand T, Bovim G. Prophylactic Treatment of migraine with an angiotensin II receptor blocker: a Randomized controlled trial. JAMA 2003;289(1):65-9.
63. Linde M, Mulleners WM, Chronicle EP, McCrory DC. Valproate (valproic acid or sodium valproate or a combination of the two) for the prophylaxis of episodic migraine in adults. Cochrane Database of Systematic Reviews 2013, Issue 6.