

Neurological Disorder Migraine

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1. Abstract :-

The second most common cause of disability worldwide is still migraine. Imaging is typically not required for diagnosis because it is dependent on the patients history and clinical examination Based on the frequency of headaches and the presence or absence of an aura, Migraine can be classified Whether a patient has chronic Migraine or episodic Migraine depends on the number of headache days. It is possible to treat Migraine in order to both alleviate the headache and stop it from happening again With the most recent data, we tackle the Migraine from a practical standpoint in this review

2. Keywords:-

Clinical features, management Migraine

3. Introduction:-

A complicated illness with genetic influences Migraine is typified by episode of moderate-to severe headache, usually unilateral, and is typically accompanied by nausea & enhanced light and sound sensitivity.

The Greek word “Hemikrania” which was then translated into Latin as “hemigranea”, is where the word Migraine originates.

One of the most common causes of impairment and missed work is Migraine. Complex brain events, Migraine episodes can last for hours or even days and are a persistent problem. Seventy-five percent of Migraine cases are of the aura free variety.

The international society’s headache classification committee has divided Migraines into subtypes. These variations consist of :-

An aura free Migraine:- A repeated headache episode lasting four to seventy -two hours is known as a Migraine without aura. These attacks are usually unilateral, pulsing ,moderate to severe in intensity, increased by physical exertion , and accompanied by light & sound sensitivity (photophobia & phonophobia)as well as nausea.

Migraine with aura:- Recurrent, fully reversible attacks of Migraine with aura often last minutes & involve one or more of the following unilateral symptoms: motor , brainstem, visual, sensory, speech & language retinal and headache.

Chronic Migraine:- A chronic Migraine is a headache that lasts longer than three month’s, lasting atleast 15 days per month, and having Migraine symptoms on at least eight of those days

4. Etiology :-

Inheritance & genetics:-

There's a major hereditary component to Migraine. Relative of sick subjects have a threefold increase incidence of Migraine compare to those of relatives of healthy subjects; nevertheless, no pattern of Inheritance the seen. The Etiology of Migraine is complex, with multiple genetic sources at different genomic locations acting in concert with environmental factors to confer susceptibility disease characteristics in affected individuals it is unclear which loci & genes are involved in pathogenesis. When a Migraine suffers genes are identified, the appropriate preventive medication may be predicted.

Triggers:-

A retrospective studies revealed that 76% patients identified triggers.

Some are probable contributing factors, while others are only possible or unproven.

These include 70% stress

Hormones changes 65% during menstruation,

Ovulation, and

Pregnancy,

Skipped meal 57%,

Whether changes 53%,

Excessive or insufficient sleep 50%,

Odours 48%,

Exposure to light 38%,

Alcohol ingestion 38%,

Late sleep 32%,

Heat 33%,

Food 27%

5. Treatment / management:-

Acute treatment:-

Acute treatment aim to stop the progression of the headache. It has to be treated quickly and with a large single dose.

Nsaid:- non steroidal anti inflammatory drug

Ibuprofen 400-600 mg

Naproxen 275-825 mg

Diclofenac 65mg

Aspirin 900-1000

Triptans :- administered as subcutaneous injection of 6 mg,

Nasal spray of 20-40 mg over 24 hours,

A Nasal powder of 10-30 mg over 24 hours

. To avoid overused of medicine, triptans should be limited to less than 10 days of use within a month.

Antiemetics :- metoclopramide , chlorpromazine they are generally used as adjunctive therapy with Nsaid

Calcitonin gene-related peptide antagonist:-

Rimegepant 75 mg a single dose

Preventive treatment agent are the following

Beta blockers:- metoprolol propranolol

Antidepressants ,

Anticonvulsant,

Calcium Channel blockers.

6 Alternative treatment:-

Lifestyle changes

Regular exercise

Yoga

Relaxation training

Cognitive behavioral therapy

Reduction of trigger

Differential diagnosis:-

Tension type headache

Cluster headache

Dissection syndrome

Meningitis

Reference:- NIH national institute of health .

1. Rose FC. The history of migraine from Mesopotamian to Medieval times. *Cephalalgia*. 1995 Oct;15 Suppl 15:1-3.
2. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. *Cephalalgia*. 2018 Jan;38(1):1-211.
3. Merikangas KR, Risch NJ, Merikangas JR, Weissman MM, Kidd KK. Migraine and depression: association and familial transmission. *J Psychiatr Res*. 1988;22(2):119-29.
4. Devoto M, Lozito A, Staffa G, D'Alessandro R, Sacquegna T, Romeo G. Segregation analysis of migraine in 128 families. *Cephalalgia*. 1986 Jun;6(2):101-5.
5. de Vries B, Anttila V, Freilinger T, Wessman M, Kaunisto MA, Kallela M, Artto V, Vijfhuizen LS, Göbel H, Dichgans M, Kubisch C, Ferrari MD, Palotie A, Terwindt GM, van den Maagdenberg AM., International Headache Genetics Consortium. Systematic re-evaluation of genes from candidate gene association studies in migraine using a large genome-wide association data set. *Cephalalgia*. 2016 Jun;36(7):604-14.
6. Riant F, Ducros A, Ploton C, Barbance C, Depienne C, Tournier-Lasserre E. De novo mutations in ATP1A2 and CACNA1A are frequent in early-onset sporadic hemiplegic migraine. *Neurology*. 2010 Sep 14;75(11):967-72.
7. Jen JC, Kim GW, Dudding KA, Baloh RW. No mutations in CACNA1A and ATP1A2 in probands with common types of migraine. *Arch Neurol*. 2004 Jun;61(6):926-8.
8. Costa C, Prontera P, Sarchielli P, Tonelli A, Bassi MT, Cupini LM, Caproni S, Siliquini S, Donti E, Calabresi P. A novel ATP1A2 gene mutation in familial hemiplegic migraine and epilepsy. *Cephalalgia*. 2014 Jan;34(1):68-72.
9. Ebrahimi-Fakhari D, Saffari A, Westenberger A, Klein C. The evolving spectrum of PRRT2-associated paroxysmal diseases. *Brain*. 2015 Dec;138(Pt 12):3476-95.
10. Jarvis SE, Zamponi GW. Masters or slaves? Vesicle release machinery and the regulation of presynaptic calcium channels. *Cell Calcium*. 2005 May;37(5):483-8.

11. Suzuki M, Van Paesschen W, Stalmans I, Horita S, Yamada H, Bergmans BA, Legius E, Riant F, De Jonghe P, Li Y, Sekine T, Igarashi T, Fujimoto I, Mikoshiba K, Shimadzu M, Shiohara M, Braverman N, Al-Gazali L, Fujita T, Seki G. Defective membrane expression of the Na(+)-HCO₃(-) cotransporter NBCe1 is associated with familial migraine. *Proc Natl Acad Sci U S A*. 2010 Sep 07;107(36):15963-8.
12. Lee HN, Eom S, Kim SH, Kang HC, Lee JS, Kim HD, Lee YM. Epilepsy Characteristics and Clinical Outcome in Patients With Mitochondrial Encephalomyopathy, Lactic Acidosis, and Stroke-Like Episodes (MELAS). *Pediatr Neurol*. 2016 Nov;64:59-65.
13. Hansen JM, Lipton RB, Dodick DW, Silberstein SD, Saper JR, Aurora SK, Goadsby PJ, Charles A. Migraine headache is present in the aura phase: a prospective study. *Neurology*. 2012 Nov 13;79(20):2044-9.
14. Stam AH, Kothari PH, Shaikh A, Gschwendter A, Jen JC, Hodgkinson S, Hardy TA, Hayes M, Kempster PA, Kotschet KE, Bajema IM, van Duinen SG, Maat-Schieman MLC, de Jong PTVM, de Smet MD, de Wolff-Rouendaal D, Dijkman G, Pelzer N, Kolar GR, Schmidt RE, Lacey J, Joseph D, Fintak DR, Grand MG, Brunt EM, Liapis H, Hajj-Ali RA, Kruit MC, van Buchem MA, Dichgans M, Frants RR, van den Maagdenberg AMJM, Haan J, Baloh RW, Atkinson JP, Terwindt GM, Ferrari MD. Retinal vasculopathy with cerebral leukoencephalopathy and systemic manifestations. *Brain*. 2016 Nov 01;139(11):2909-2922.
15. Martin VT, Behbehani MM. Toward a rational understanding of migraine trigger factors. *Med Clin North Am*. 2001 Jul;85(4):911