

## A CASE REPORT ON MIGRAINE TREATED WITH BACILLUS MORGAN (BACH)

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### ABSTRACT

Migraine affects more than one billion individuals each year across the world, and is one of the most common neurologic disorders, with a high prevalence and morbidity, especially among young adults and females. Migraine is associated with a wide range of comorbidities, which range from stress and sleep disturbances to suicide. The complex and largely unclear mechanisms of migraine development have resulted in the proposal of various social and biological risk factors, such as hormonal imbalances, genetic and epigenetic influences, as well as cardiovascular, neurological, and autoimmune diseases.

**KEYWORDS:** Migraine, Epidemiology, Risk factors, Bacillus morgan, Homoeopath, Cardiovascular, Neurological & Autoimmune diseases.

### INTRODUCTION

Migraine is a genetically influenced complex disorder characterized by episodes of moderate-to-severe headache, most often unilateral and generally associated with nausea and increased sensitivity to light and sound. The word migraine is derived from the Greek word "hemikrania," later converted into Latin as "hemigranea." The French translation of such a term is "migraine".<sup>[1]</sup> Migraine is a common cause of disability and loss of work. Migraine attacks are complex brain events that unfold over hours to days in a recurrent manner. The most common type of migraine is without aura.

Migraines can be classified into subtypes according to the headache classification committee of the International Headache Society.<sup>[2]</sup> These subtypes are:

*Migraine without aura* is a recurrent headache attack of 4 to 72 hours; typically unilateral in location, pulsating in quality, moderate to severe in intensity, aggravated by physical activity, and associated with nausea and light and sound sensitivity (Photophobia and Phonophobia).

*Migraine with aura* has recurrent fully reversible attacks, lasting minutes, typically one or more of these unilateral symptoms: visual, sensory, speech and language, motor, brainstem, and retinal, usually followed by headache and migraine symptoms.

*Chronic migraine* is a headache that occurs on 15 or more days in a month for more than three months and has migraine features on at least eight or more days in a month.

### **Complications of migraine**

- ❖ Status migrainosus is a debilitating migraine attack that lasts more than 72 hours.
- ❖ Persistent aura without infarction is an aura that persists for more than one week without evidence of infarction on neuroimaging.
- ❖ Migrainous infarction is one or more aura symptoms associated with brain ischemia on neuroimaging during a typical migraine attack.
- ❖ Migraine aura-triggered seizure occurs during an attack of migraine with aura, and a seizure is triggered.
- ❖ Probable migraine is a symptomatic migraine attack that lacks one of the features required to fulfil the criteria for one of the above and does not meet the criteria for another type of headache.
- ❖ Episodic syndromes that may be associated with migraine
- ❖ Recurrent gastrointestinal disturbances are recurrent attacks of abdominal pain and discomfort, nausea, and vomiting that may be associated with migraines.
- ❖ Benign paroxysmal vertigo has brief recurrent attacks of vertigo.
- ❖ Benign paroxysmal torticollis is recurrent episodes of head tilt to one side.

### **Etiology**

#### ***Genetics and Inheritance***

Migraine has a strong genetic component. The risk of migraines in ill relatives is three times greater than that of relatives of non-ill subjects, but no inheritance pattern was identified.<sup>[3,4]</sup>

The genetic basis of migraine is complex, and it is uncertain which loci and genes are the ones implicated in the pathogenesis; it may be based on more than one genetic source at different genomic locations acting in tandem with environmental factors to bring susceptibility and the

characteristics of the disease in such individuals.<sup>[5]</sup> Identifying these genes in an individual with migraines could predict the targeted prophylactic treatment.

### ***Familial hemiplegic migraine***

Hemiplegic migraine can occur in families or sporadically (One individual, as the first member of the family to have a hemiplegic migraine).<sup>[6]</sup>

### **Channelopathies cause the primary three types**

**Type 1** is caused by mutations in the CACNA1A gene (calcium voltage-gated channel alpha 1A subunit) on chromosome 19p13.<sup>[7]</sup>

**Type 2** is caused by mutations in the ATP1A2 gene (ATPase, Na<sup>+</sup>/K<sup>+</sup> transporting alpha two subunit) on chromosome 1q23.<sup>[8]</sup>

**Type 3** is caused by mutations in the SCN1A gene (sodium voltage-gated channel Type 1 alpha subunit).

Mutations in the PRRT2 (Proline-Rich transmembrane 2) gene are recognized as a possible cause.<sup>[9]</sup> PRRT2 gene encodes a protein that interacts with the SNAP25 (Synaptosomal nerve-associated protein 25), which may pose a role in voltage-gated calcium channel regulation.<sup>[10]</sup>

Mutations in the SLC4A4 (solute carrier family four member 4) gene have also been associated with familial forms of migraine.<sup>[11]</sup>

### **Melas**

It is a syndrome of mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes, a multisystemic disorder by maternal inheritance that can present recurrent migraine headaches.<sup>[12]</sup>

### **Cadasil**

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) angiopathy by autosomal dominant inheritance, caused by mutations in the NOTCH3 gene (notch receptor 3) on chromosome 19 that can present migraine with aura (prodrome in 80%) in nearly 50% of carriers.<sup>[13]</sup>

### **RVCL**

Retinal vasculopathy with cerebral leukodystrophy is angiopathy by C-terminal frame-shift

mutations in TREX1 (three prime repair exonuclease 1) presents almost 60% of the cases.<sup>[14]</sup>

### **Hihratl**

Hereditary infantile hemiparesis, retinal arteriolar Tortuosity and Leukoencephalopathy.

### **Herns**

Hereditary endotheliopathy with retinopathy, nephropathy and stroke.

### **Triggers**

Withdrawn or exposure to several factors contribute to the development of migraine headaches<sup>[15]</sup>

- Stress
- Hormonal changes during menstruation, Ovulation and Pregnancy
- Skipped meals
- Weather changes
- Excessive or insufficient sleep
- Odors (Perfumes, Colognes, Petroleum distillates)
- Neck pain
- Exposure to lights
- Alcohol ingestion
- Smoking
- Late sleeping
- Heat
- Food (Aspartame and Tyramine and Chocolate)
- Exercise
- Sexual activity

### **Epidemiology**

Migraine is highly prevalent, affecting 12% of the population, attacking up to 17% of women and 6% of men yearly.<sup>[16,17&18]</sup> Among children, it tends to happen more in girls than boys.<sup>[19]</sup>

The adjusted prevalence of migraine is highest in North America, followed by South America, Central America, Europe, Asia, and Africa. It is ranked as the second leading cause of disability worldwide.<sup>[20]</sup> Migraine tends to run in families.<sup>[16]</sup> There is a reported risk of 40% if one parent has a history of migraine, which increases to 75% when both parents have a migraine history.

It is consistently the fourth or fifth most common reason for emergency visits accounting for an annual 3% of all emergency visits.<sup>[21]</sup> Its prevalence increases in puberty but continues to increase until 35 to 39 years of age, decreasing later in life, especially after menopause.<sup>[17]</sup> Moreover, it is considered the second major cause of disability after back pain with respect to years of life lived with disability.<sup>[19]</sup>

### Phases of migraine attacks

Four phases have been identified in migraine attacks.<sup>[22]</sup>

#### 1). *Prodrome*

- Premonitory symptoms associated with hypothalamus activation (Dopamine)<sup>[23&24]</sup>
- Around 77% of patients suffer prodromic symptoms for up to 24 to 48 hours before headache onset. It is more common in females than males.
- Frequent symptoms are yawning, mood change, lethargy, neck symptoms, light sensitivity, restlessness, difficulties in focusing vision, feeling cold, craving, sound sensitivity, sweating, excess energy, thirst, and edema.

#### 2). *Aura*

- Changes in cortical function, blood circulation, and neurovascular integration occur in some cases.<sup>[2,13,25,26]</sup>
- It can precede the headache, or it can present simultaneously.
- They are typically gradual, less than 60 minutes in duration, more often visual, and have positive and negative symptoms.
- Positive symptoms are caused by active release from central nervous system neurons (Bright lines or shapes, Tinnitus, Noises, Paresthesias, Allodynia, or Rhythmic movements).
- Negative symptoms indicate a lack or loss of function (Reduction or loss of vision, hearing, sensation, or motion).
- They have to be fully reversible.
- It usually consists of tingling sensations on one side of the face or a limb. They are considered paraesthesia's.
- The most common positive visual symptom is the scintillating scotoma (an area of absent vision with a shimmering or glittering zigzag border).
- The most common negative visual symptom is visual field defects.
- Visual auras are the most frequent ones.

- Sensory auras are also common. They can follow visual symptoms or occur without them.
- Language auras are not frequent. They consist of transient dysphasia.
- Motor auras are rare. They consist of complete or partial hemiplegia involving limbs and the face.

### 3). *Headache*

- Additional changes in blood circulation and function of the brainstem, thalamus, hypothalamus, and cortex.
- Often unilateral, generally with a pulsatile or throbbing feature and increasing intensity within the first hours.
- The intensity can correlate to nausea, vomiting, photophobia, phonophobia, rhinorrhoea, lachrymation, allodynia, and osmophobia.
- It can take place over hours to days.
- Patients may seek relief in dark places, as the pain usually resolves in sleep.

### 4) *Postdrome*

- Persistent blood changes with symptoms after headache termination
- This phase consists of a movement-vulnerable pain in the same location as the previous headache.
- Common symptoms can be exhaustion, dizziness, difficulty concentrating, and euphoria.

## Evaluation

The *diagnosis of migraine* is based on patient history, physical examination, and fulfilment of the diagnostic criteria. The necessary information that must be gathered consists of these simple questions:

- ✓ Demographic features of the patient: age, gender, race, profession
- ✓ When did the headache start?
- ✓ Where does it hurt? Location, irradiation.
- ✓ What is the intensity of the pain?
- ✓ How is the pain? Which are the qualitative characteristics of pain?
- ✓ How long does the pain last?
- ✓ At which moment of the day does the pain appear?
- ✓ How has it evolved since it started?

- ✓ What is the frequency of appearance?
- ✓ What are the triggering situations?
- ✓ Simultaneous symptoms?
- ✓ Is it related to sleep?
- ✓ How does it get better or worse?

### **Case profile**

*Present complaints:-* A 36 -year-old Female presented at Sophia Homoeopathic Medical College and Hospital & Research Institute, Gwalior (M.P.) in June 2023 with the recurrent complaint of the Headache with throbbing pain since 10 to 15 years. Pain more prominent in temporal & parietal region and Left Side. Frequency of attack remain 3 to 4 days. Agg < Warmth, Stress, Talking, Travelling, Sunlight, Spicy & Oily Food.

*Associated complaints:-* Pain in eyes, Nausea, Vertigo, Vomiting, Black Spots before eyes, Eructation & Flatulence.

*History of present complaints:-* Patient was apparently well when she gradually develop her complaints. She used to take allopathic medicines for pain frequently.

*Past medical history & previous treatment history:-* Take allopathic medicines for pain.

*Family history:-* Headache present in mother.

*Obs/gynae history:-* Hysterectomy done

*Obstetric History:-* G10 P7 A3 L7

*Personal history:-*

Addiction- Nothing Significant Diet- Not Good

Habits- Nothing Significant

*Childhood history [MILESTONES-* Teething/Talking/Walking all milestones are appear on time.

### **Physical generalities**

Appetite: - Decrease

Desires/Craving: - Salty

Aversion/Dislikes: - Sweets

Thirst:- Normal

Thermal:- Ambithermal

Taste:- Bland

Stool: -Satisfactory

Urine: - D4N2, NAC

Perspiration: - Profuse

Sleep:- Decrease

Dream:- Nothing Significant

Male/ Female Sexual function:- Dyspareunia

Sensitivity (Noise, Odour, sun, light, atmosphere changes, Neck tight):- Nothing Significant

*Mental General symptoms:-* Tense because of family matters & wants to live alone.

### A) Physical examination

Apperance-	Looking Weak	Complexion- Wheatish	Built- Mesomorphic
Nutrition-	Normal	Height- 5.2	Weight- 57 Kg
Anaemia-	Absent	Jaundice- Absent	Cyanosis-Absent
Oedema-	Absent	Skin- Normal	Clubbing- Absent
Blood Pressure-	128/90 mmhg	Pulse rate- 80/Min	Temperature- A/F
Respiratory rate-	20/Min	Tongue- Clean& Moist	Lymphadenopathy- Absent

### B) Systemic examination

Respiratory system: - Nothing abnormal detected

Cardiovascular system: - Nothing abnormal detected Central Nervous system: - Nothing abnormal detected Gastro-intestinal system: - Nothing abnormal detected Genito - urinary system :- Nothing abnormal detected Musculo-skeletal system:- Nothing abnormal detected.

### Analysis and Evaluation of the case

Symptoms	MG/PG/Particulars	Intensity
Tense because of family matters.	Mental general	3+
Wants to live alone	Mental general	2+
Appetite decrease	Physical general	3+
Desire for salty	Physical general	2+
Aversion for sweets	Physical general	2+
Profuse perspiration	Physical general	3+
Headache since 10-15 yrs. with pain eyes, nausea, vertigo, vomiting, black spots before eyes, eructation, and flatulence	Particular	3+

### Prescription (Medicine, Potency, Dose, Repetition)

Bacillus morgan 202/2 doses Placebo 30/3 for 7 days.



**Auxillary measures**

- ❖ Avoid stress.
- ❖ Take nutritious and adequate food.
- ❖ Avoid oily, spicy food.

**Follow UP**

Date	Symptoms	Prescription
23/06/2023	Slight relief in headache	Rubrum 200/4 doses Sac lac 30/3 for 7 days
30/06/2023	Slight relief in headache and appetite	Sac lac 30/3 for 7 days Rubrum 200/4 doses
07/07/2023	Headache improved, and other symptoms like pain eyes, vertigo	Sac lac 30 /3 Rubrum 200/ 4 doses for 7days
14/07/2023	Improvement in headache and other associated symptoms like nausea, vomiting, pain eyes,	Rubrum 200 / 4 doses Sac lac 30/3 for 7 days
	appetite, spots before eyes etc.	
21/07/2023	All symptoms improved	Rubrum 200/4 dose Sac lac 30/3 for 7 days
28/07/2023	All symptoms improved	Rubrum 200/4 dose Sac lac 30/3 for 7 days

**CONCLUSION**

There are many more remedies for Migraine in Homoeopathy. Bacillus Morgan (Bach) belongs to bowel nosodes. It is prepared by attenuating the cultures of non-lactose fermenting bacilli of intestinal flora. It is most frequently found in stool and it has the greatest number of associated remedies compared to other types on list. Bacillus morgan can be very useful in cases of Migraine.

**REFERENCES**

1. Rose FC. The history of migraine from Mesopotamian to Medieval times. Cephalalgia, 1995; 15, 15: 1-3. [PubMed]
2. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia, 2018; 38(1): 1-211. [PubMed]
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. [PubMed]
4. Devoto M, Lozito A, Staffa G, D'Alessandro R, Sacquegna T, Romeo G. Segregation

- analysis of migraine in 128 families. *Cephalalgia*, 1986; 6(2): 101-5. [PubMed]
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; 36(7): 604-14. [PubMed]
  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; 14, 75(11): 967-72. [PubMed]
  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; 61(6): 926-8. [PubMed]
  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; 34(1): 68-72. [PubMed]
  9. Ebrahimi-Fakhari D, Saffari A, Westenberger A, Klein C. The evolving spectrum of PRRT2-associated paroxysmal diseases. *Brain*, 2015; 138(Pt 12): 3476-95. [PubMed]
  10. Jarvis SE, Zamponi GW. Masters or slaves? Vesicle release machinery and the regulation of presynaptic calcium channels. *Cell Calcium*, 2005; 37(5): 483-8. [PubMed]
  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(+)-HCO<sub>3</sub>(-) cotransporter NBCe1 is associated with familial migraine. *Proc Natl Acad Sci U S A*, 2010; 107, 107(36): 15963-8. [PMC free article] [PubMed]
  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; 64: 59-65. [PubMed]
  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; 13, 79(20): 2044-9. [PMC free article] [PubMed]
  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, Ferrari MD. Retinal vasculopathy with cerebral leukoencephalopathy and systemic manifestations. *Brain*, 2016; 01, 139(11): 2909-2922. [PMC free article] [PubMed]
  15. Martin VT, Behbehani MM. Toward a rational understanding of migraine trigger factors.

- Med Clin North Am, 2001; 85(4): 911-41. [PubMed]
16. Lipton RB, Stewart WF, Diamond S, Diamond ML, Reed M. Prevalence and burden of migraine in the United States: data from the American Migraine Study II. *Headache*, 2001; 41(7): 646-57. [PubMed]
  17. Lipton RB, Bigal ME, Diamond M, Freitag F, Reed ML, Stewart WF., AMPP Advisory Group. Migraine prevalence, disease burden, and the need for preventive therapy. *Neurology*, 2007; 30, 68(5): 343-9. [PubMed]
  18. Vetvik KG, MacGregor EA. Sex differences in the epidemiology, clinical features, and pathophysiology of migraine. *Lancet Neurol*, 2017; 16(1): 76-87. [PubMed]
  19. MacGregor EA. Migraine. *Ann Intern Med*, 2017; 04, 166(7): ITC49-ITC64. [PubMed]
  20. GBD 2016 Headache Collaborators. Global, regional, and national burden of migraine and tension-type headache, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet Neurol*, 2018; 17(11): 954-976. [PMC free article] [PubMed]
  21. Burch R, Rizzoli P, Loder E. The Prevalence and Impact of Migraine and Severe Headache in the United States: Figures and Trends From Government Health Studies. *Headache*, 2018; 58(4): 496-505. [PubMed]
  22. Charles A. The evolution of a migraine attack - a review of recent evidence. *Headache*, 2013; 53(2): 413-9. [PubMed]
  23. Karsan N, Goadsby PJ. Imaging the Premonitory Phase of Migraine. *Front Neurol*, 2020; 11: 140. [PMC free article] [PubMed]
  24. Laurell K, Artto V, Bendtsen L, Hagen K, Häggström J, Linde M, Söderström L, Tronvik E, Wessman M, Zwart JA, Kallela M. Premonitory symptoms in migraine: A cross-sectional study in 2714 persons. *Cephalalgia*, 2016; 36(10): 951-9. [PubMed]252.
  25. Hansen JM, Charles A. Differences in treatment response between migraine with aura and migraine without aura: lessons from clinical practice and RCTs. *J Headache Pain*, 2019; 06, 20(1): 96. [PMC free article] [PubMed]
  26. Van Dongen RM, Haan J. Symptoms related to the visual system in migraine. *F1000Res*, 2019; 8. [PMC free article] [PubMed]