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# HEMIFACIAL SPASM AND ITS HOMOEOPATHIC MANAGEMENT – CASE REPORTS

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## ABSTRACT:

Borders Neurovascular Compression (NVC) syndromes are the most common abnormalities due to the Anatomical aberrant positions of the vessels causing Irritations of the Nerves and the supplied muscle common in the Cranial nerves V, VII, VIII and occasionally in others Nerves. The common Vessels involved are Anterior Inferior Cerebellar Artery (AICA), Posterior Inferior Cerebellar Artery (PICA) and Vertebral Artery (VA). Hemifacial Spasm is compression of Facial Nerve is due to the compression by the Anatomical aberrations, due to the cystic compression or any Space occupying lesions. These compress at the Root exit zone and Transitional zone causing demyelination resulting in conduction abnormalities, Ephaptic transmissions, and Abnormal Muscles responses. Chronic compressions causing symptoms like twitching, facial spasms, frequent Blinking, Headaches, Occular irritation, Rhinopyema.etc can be detected by MRI which has high accuracy. Conventional treatment like micro vascular decompressions surgery or Botox injections may have side effects of muscle weakness, Facial palsy etc. Homoeopathic Remedies are very helpful in such conditions for those unwilling to opt for surgical Managements without any side effects. Though it cannot change the anatomical positions of the aberrant vessels and its compression affects it relieves the pain and spasmodic effects. Here two cases of Primary Hemifacial spasm are discussed with the miraculous improvement in the Facial Spasms and twitching with Silicea and Calcarea carb selected on the basis of totality and Repertorisation

**Keywords**: Neurovascular compression, Hemifacial spasm, Homoeopathic Management.

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# Introduction:

Neurovascular compression syndromes are defined as direct contact of Blood vessels with Cranial Nerves causing Mechanical irritation and compression leading to abnormal contractions of the musculature. The most common cases are Trigeminal Neuralgia, Hemifacial Spasm, Vestibulocochlear Neuralgia and Glossopharyngeal Neuralgia.1.

The long Blood vessels running near the Brainstem may be elongated and coils into loops as a part of ageing or Degenerative changes and Hypertensive changes as well causing compression symptoms.2

**Incidence**: The Hemifacial spasm is wrongly diagnosed in majority of cases and few of the cases were being mismanaged. The Worldwide estimates for the prevalence of HFS are 14.5 per 100,000 women and 7.4 per 100,000 men.3

The majority of HFS cases occur unilaterally with an estimated 0.6% to 5% occurring bilaterally. It is more commonly involved on the left side (56% approximately) and few patients had bilateral involvement. HFS was associated with Trigeminal Neuralgia in 5% of the cases and 5.7% with History of Bell's palsy. Primary HFS is more prevalent and the age of onset is in the 5th decade of life with a female preponderance and Right side left side ratio was 1.07:1 with Hypertension was the most prevalent systemic disease in the background.

**ANATOMY:** Vascular contact with the Facial Nerve root-exit zone or at the internal auditory canal was present in 96% of patients with Hemifacial spasm. The vessels responsible were the vertebral artery (VA) posterior Inferior Cerebellar artery (PICA) & anterior Inferior Cerebellar arteries (AICA).4

**The Transition Zone:** Most of the nerves are attached to the Neuraxis by rootlets. The fascicles of Cranial Nerve axons leave the Peripheral Nervous System (PNS) to enter the Central Nervous System (CNS), through the so-called PNS-CNS transitional zone. This transition zone between the Central and Peripheral myelin is the most vulnerable region for Neurovascular compressions causing symptoms. The CNS–PNS transitional zone (TZ) is a very irregular interface, consisting of the astrocytic tissue comprising the central component and central to this, Myelin sheaths are formed by Oligodendrocytes with Astrocytic and peripheral to it, sheaths are formed by Schwann cells which are enveloped in Endoneurium.5

The Transition zones of various Nerves are as follows: In symptomatic Trigeminal Neuralgia, the Transition zone is 4 mm, with Neurovascular compression typically in proximal end. In

Symptomatic Hemifacial spasm the transition zone is 2.5 mm with proximal end involvement. In Vestibular Paroxysmia the transition zone is 11 mm at the internal auditory canal. The lateral transitional zone of the Facial Nerve has a mean length of 1.9 mm and the root detachment point of the Facial Nerve at the medial side was located very close to the beginning of the medial transitional zone. In more than 80% of the nerves that were examined, vascular structures compressed the central glial myelin of the nerve.6

**Histology:** The Voxel-Based Morphometry (VBM) analysis showed that patients with HFS has reduced Grey Matter volume (GMV) in the Right Inferior Parietal lobule and decreases with the disease duration when compared with healthy subjects.7 Even in patients with Trigeminal Neuralgia, the Cisternal CN V shows focal demyelination in the region of Vascular compression, and it has been observed to be more frequent in proximal (<3 mm) than in distal sites.

**ETIOLOGY:** Walter Dandy first described contact of a Vessel loop with the trigeminal root in patients with trigeminal neuralgia, since then the theory of neurovascular compression has been widely accepted. Studies suggest that distal Neurovascular compression can be classified by pure Neurovascular compression in distal, cisternal portion by a Single artery [AICA] or Double compression (both REZ and the distal portion) by a single arterial loop or two different offending arteries (AICA and PICA/VA].7 The Meatal loop of the AICA (43%) is the most common vessel that compress the FN when the vessel passed between the Seventh and the Eighth Cranial nerve.[9]

The various possibilities of Neurovascular compression are: [10]

i) The Blood vessel twists itself into long loop and compresses the Nerve roots, or
ii) The nerve is compressed between the Arachnoid Trabeculae and Blood vessel or
iii) The perforating arteries from the compressing vessel pushes the vessel to the brainstem
iv) Nerve is caught between the compressing vessel and its branches or
v) Sandwich type, where the nerve is sandwiched between two different vessels, and
vi) One vessel compresses another vessel that pushes the nerve.

HFS is of two types. Primary and Secondary- Development of Primary HFS beyond the 5th decade can be attributed to the senile / loss of elasticity changes in the walls of the blood vessels resulting in an ectatic blood vessel with Hypertension as the most prevalent causation in background. Primary HFS was 10 times more common than secondary HFS and Facial/Bell's palsy was the leading cause of secondary HFS. Other important etiologies were CPA angle tumor/cyst and

inferior Cerebellar artery aneurysm.[11] Primary HFS occurs due to vascular compression of the Facial Nerve root entry zone in the posterior fossa by Anterior Inferior Cerebellar artery (AICA), Posterior Inferior Cerebellar artery (PICA), and Vertebral Artery (VA) whereas Secondary HFS occurs due to Neurovascular Compression by Tumors or Cysts, Post Palsy effects, etc. Anatomic variations in vasculature such as lateral deviation of one or both vertebral arteries occurred on the ipsilateral side of HFS in 86.4% cases, making these variations a HFS risk factor. Though AICA was said to be the most common artery involved, Intraoperative Endoscopic examination of a study shown Posterior Inferior Cerebellar artery as the offending vessel followed by Vertebral and AICA.12

**PATHOLOGY:** The Facial Nerve responsible for facial Expression is mostly being compressed by the Anterior Inferior Cerebellar Artery eventually causing spasm. If trigeminal neuralgia is preceded or accompanied by Hemifacial spasm, this may indicate that there is a tumor, aneurysm, or arteriovenous malformation compressing both the trigeminal (V) and Facial (VII) nerves. [13]

The Intracranial Segment of Normal facial Nerve Facial Nerve is ensheathed by Arachnid Membrane only and this segment shows demyelination in chronic Compression Syndromes causing Ectopic excitation, Ephaptic transmission between Facial nerve fibers, and Auto Excitation phenomenon.[14] Most of the demyelinated Nerves show Ectopic excitations and Abnormal Muscle Responses.

Ectopic excitations also occur which are impulses generated independently of the natural synapse when the excitation threshold is low in demyelinated conditions. Abnormal muscle response (AMR), is stimulation in Facial muscles innervated by other branches of the Facial nerve, is most specific for patients with HFS. The AMR consists of a constant response occurring about 10 ms after stimulus with auto excitation. Also the phenomenon of F-wave in facial muscles is a small recurrent discharge that propagates in the facial motonucleus via same axon. A study shown that there exist a linear correlation between AMRs elicited in the mentalis muscles by the zygomatic branch stimulation of the facial nerve and that of the F-waves in the mentalis muscles, which suggest that the AMR is an exaggerated F-wave.[15]

## RADIOLOGICAL INVESTIGATIONS: :

Non Invasive Procedures such as MRI and MRA detects the information on Vascular and Brain tissues defects and is a primary Neuroradiological investigation in HFS. It also supports findings of electrophysiological investigations indicating that local irritation of the facial nerve is the most

possible explanation for HS.[16]

Due to the limitations of CT in the visualization of posterior fossa structures, MR imaging should be considered the initial screening procedure in the assessment of patients with Hemifacial spasm.[17]

In most of the cases the MRI reveals that the transition zone overlaps the Root entry zone close to the brain stem in cranial nerves V, VII, and IX, yet it is more distal and does not overlap the root entry zone in cranial nerve VIII. It also revealed that the mean age of onset of the spasms in the primary HFS group was 49.26 ± 8.35 years, and in secondary HFS was 43.13 ± 12.12 years respectively and Facial nerve palsy was the most common cause (n = 13) of secondary HFS followed by Cerebellopontine angle (CPA) tumors. **[18, 19]**

**Clinical Signs and Symptoms**: [20] A close contacts between a major artery and a nerve trunk over a prolonged period of time may cause changes in the Nerve trunk, in the form of localized demyelination and initiates the phenomenon of ephaptic transmission causing clinical Symptoms and signs.

Hemifacial spasm is characterized by unilateral, intermittent contractions of the muscles of facial expression, typically beginning in the Orbicularis oculi and spreading to the other muscles. Primary HFS is triggered by NVC, whereas secondary HFS comprises all other causes of CN VII damage. The AICA (43%) is the most common vessel causing NVC.

HFS starts with tonic-clonic contractions of the orbicularis oculi muscle, resulting in involuntary eyelid closure and eyebrow elevation. Over time, the contractions progress to Frontal Region, Neck region, Platysma and orbicularis oris. Later gradually patient may develop sustained contractions of all involved muscles, causing a severe, disfiguring grimace with partial closure of the eyes and lifting of the mouth corners in the "tonus phenomenon". The majority of HFS cases occur unilaterally. Some patients report fatigue, anxiety, headaches, Rosacea, Occular irritation and Rhinopyma.

Few studies suggest that Glossopharyngeal Neuralgia, disabling positional vertigo, tinnitus, Geniculate Neuralgia, Spasmodic torticolis, essential Hypertension, Cyclic oculomotor spasm with paresis and superior oblique myokymia also may be initiated by vascular compression of the glossopharyngeal, cochleovestibular, intermediate, accessory, oculomotor and trochlear nerves or the ventrolateral medulla oblongata.

HFS starts with Tonic-Clonic contractions of the Orbicularis Oculi muscle, resulting in involuntary eyelid closure, eyebrow elevation and progressively affecting the muscles of the forehead, muscles of the neck and mouth. Causing a severe, disfiguring grimace with partial closure of the eyes and lifting of the mouth corners in the "Tonus phenomenon".

Lifting of the ipsilateral eyebrow with eye closure during the spasm, known as the Babinski-2 sign, is highly specific and sensitive for the condition.[21]

GRADING:

| Grade | Detailed description |
|---|---|
| I | Localized spasm around the periocular area |
| II | Involuntary movement spreads to other parts of the ipsilateral face and affects other muscle groups: the orbicularis oris, zygomaticus, frontalis or platysma muscle |
| III | Interference with vision because of frequent tonic spasms |
| IV | Disfiguring asymmetry: continuous contraction of the orbicularis oculi muscles affects opening of the eye |

**Fig. no.1**. *Published in Stereotactic and Functional Neurosurgery 2011; Using the New Clinical Grading Scale for Quantification of the Severity of Hemifacial Spasm: Correlations with a Quality of Life Scale.*

**Differential Diagnosis:** Hemifacial spasm needs to be differentiated from Blepharospasm, Oromandibular Dystonia, Facial Nerve Tic, Hemimasticatory Spasm, Focal seizures and Synkinesias after Facial Nerve Paralysis.[22]

**Management:** Though many treatment options available surgical repair of the Hemifacial spasm condition has the most successful outcome. Surgical management of the offending vessel was moved away from the site and the decompression was secured by inserting Teflon sponges, placed between the offending vessels and the acousticofacial bundle, ensuring that both the Teflon sponge and the offending vessels do not contact the root exit zone nor the transition zone, leading to facial spasm disappearance of the symptoms in immediate postoperative period. In few cases MR angiography performed 6 months after surgery confirmed the relapse of the conflict though the patient is free of symptoms.[23]

Though surgical option too has lot of success rate, commonest complications include Sensorineural

hearing loss and Facial Nerve Palsy are the main complications to be expected and also postoperative mild Transient Facial Weakness, mild sensory-neural hearing loss, delayed facial palsy, transient facial twitching, and paradoxical CSF rhinorrhea in few cases.[24]

Since vascular decompression reported many permanent complications, Botulinum toxin type A is the next most preferable treatment of choice for relief of Facial spasms, but on chronic use it cause degeneration of Muscle mass and flattening of the expressions.[25] Felbamate therapy is also another alternative to surgical approach in Hemifacial spasm.[26, 27]

**Homoeopathic Management:** A Homoeopathic remedy act dynamically on the vital force and relieves the suffering humanity. It is a successful alternative system of Medicine for the last many decades. It is beneficial where surgery is an option in those suffering from other systemic diseases, and the economically weaker sections.

In Primary Hemifacial spasms the Homoeopathic remedies although cannot modify the Anatomical aberrations nor elongated vascular loops or degenerative changes in arteries; its action at the dynamic vital Force can relieve the Spasms or twitching conditions arising there from so that the patient is relieved completely avoiding surgery.

Many well known Homoeopathic Remedies are proved to be successful in Neurological abnormalities such as Bells Palsy, Hemiplegia, Motor Neuron Diseases, etc.

In Primary Hemi facial Spasm the Remedies found under various rubrics are Causticum, Plumbum, Apis, Calcarea, Belladona, Cicuta, Argentum Noitricum,Pulsatilla etc.
Though these remedies are indicated in the particular condition, here are the two case reports successfully treated with Individualized Constitutional Homoeopathic Treatment selected upon Totality of Symptoms using Classical Repertorisation technique.

## CASE NO.1:

Patient of age 52 years female a resident of Kerala, visited OPD in Sep'2021 with complaints of Spasmodic Closure of Left Eyelids, accompanied by twitching of facial muscles of left side. It is also accompanied by Weakness of Left side facial muscles with Drooping of Left eyelid. The complaints are usually aggravated during talking any actions involving facial muscles. The twitching usually aggravates during evening and talking while.

Initially the complaints started as twitching of eyelids later it spread all over the face accompanied by sensation of pins on face. It is also accompanied by deviation of angle of mouth. These complaints got gradually aggravated. She took Allopathic treatment for 1 year and got relieved so then discontinued. The complaints got reappeared 3 months back and more severely aggravated since 1 month. Already a known case of Thyroidism since 3yrs, On Allopathic medication, Thyronorm, Dyslipidemia.

Past History of Hepatitis 10years back. Rheumatoid arthritis since 20years. Hysterectomised for fibroid uterus 10years back.

Physical Generals: Appetite: good; Bowels: Irregular, Loose stools. Ambithermal: Desires warm food and drinks; Aversions: Nothing specific;

Mental Generals: Sensitive, Anxious++, Emotional, Easily weeping. Consolation> Brooding over her troubles, Artistic, Loves Music; Stage fright +++, Reserved, Fear of Crowd, Desire to be taken care of by others always.

MRI - Left AICA looping in close Proximity to Lt.Vestibulo Facial Nerve complex in its Cisternal Segment.

Examination Findings: Spasmodic Closure of Left eye especially while talking or slightest emotional excitement. Sensations are intact. No facial weakness but on attempting to talk or on slightest stimulation

FIRST PRESCRIPTION: 22/09/21: SILICEA 200 WEEKLY ONE DOSE FOR 2 WEEKS
03/10/2021: Frequency of twitching of facial mucles only slight Improvement - SILICEA 1M 2DOSES WEEKLY ONCE FOR 2 WEEKS

22/10/21: Complaints persist; Anxiety about Disease++, Twitching of Left Eyelids++, Pain in Left Heel - Reduced; Frequency of twitching Increased; No other Complaints. SACLAC FOR 1 MONTH

19/11/21: Twitching of Left Eyelids Present constantly. Sensation of weakness of Left Side of the face < Talking; chewing.

Pain on Heel region of Rt.leg. Pain on Lower back < bending. - SILICEA 200 2 Doses

3/12/21: Complaints Aggravated Lowback ache, Heel pain reduced. Stress Incontinence Mildly better. 31/12/21: Twitching of Left side of face much reduced; Pain Low back ache - Much Better - Pulsatilla 200 -2d ; Once in 15 days; Saclac 15 days. Later the patient got entirely relieved of her Facial Spasm complaints, with No episodes of recurrences.

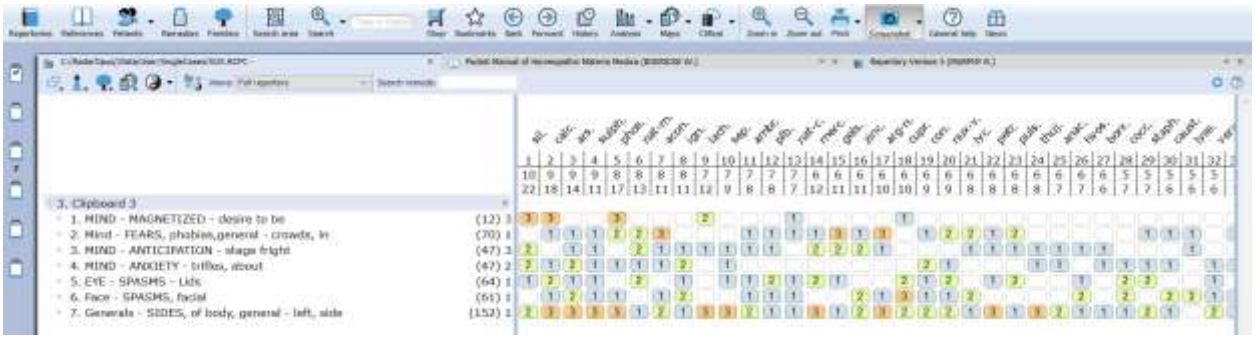


Fig. no.2: Repertorisation chart No. 1

## CASE NO.2:

Patient of age 66 years female a resident of Kerala visited OPD in Nov'2021 with complaints of frequent blinking of right eye since 2012. It is also accompanied with twitching of right side of face. Also she complains of pain all over the Right side of face and head. All the complaints are aggravated by talking. **H/o Presenting Complaints**: The complaints started as frequent Blinking of Right Eye with twitching of right side of Face. There is also pain along the Right side of head on and off extending upto the right lower part of face.

Past History: H/O Hepatitis in the past. Underwent Surgery for Osteopenia of spine in 10years back.

Physical Generals: Desires Pungent Aversion: Milk Thirst: Increased prefers for warm water;

Perspiration: More on Axillary region; Thermally: Chilly Patient

Mental Generals: Sluggish, More Laziness to do works. Extroverted and prefers to be in company always.

Examination Findings: Frequent blinking of Right eye especially while talking or slightest emotional excitement. Twitching on right side of face while, Sensations are intact. No facial weakness but on attempting to talk or on slightest stimulation all the complaints increase.

## FIRST PRESCRIPTION:

10/11/21: Calcarea Carb 200 WEEKLY ONE DOSE FOR 2 WEEKS and SACLAC BD for 15days.
24/11/2021: Frequency of blinking much better for 1week but again complaints aggravated with accompanying headache. - Calcarea Carb 200 2 Doses Weekly Once For 2 Weeks and Saclac Bd for 15days.

08/12/21: Blinking of Eye much reduced, but recurring after exertion/eye strain. Twitching of Right side of face was also reduced. Calcarea carb 200 1 dose and Saclac BD for 15days.

After 1month Patient had a Telephonic conversation saying that "twitching of right side of face and Blinking of eyes were totally relieved with No recurrence. But due to Covid Constraints the patient did not follow up again.


Fig. no.3: Repertorisation chart No. 2

## DISCUSSION:

Neurovascular syndromes are the most common causing weakness of the muscles supplied by particular Cranial Nerve. Especially it is common in the Cranial nerves V, VII, VIII and occasionally in others. As per the above the common Vessels involved are Anterior Inferior Cerebellar Artery(AICA), Posterior Inferior Cerebellar Artery(PICA) and Vertebral Artery(VA). These vessels compress the Nerves especially at the Root entry zone and Transitional zone causing Demyelination resulting in Delayed conduction, Ephaptic transmissions, and Abnormal Muscles responses. The Primary HFS is caused by Neurovascular compression whereas Secondary type is attributed to compression by Cysts, Tumors, and secondary to Bells/Facial Palsy injuries. Though most of the cases are asymptomatic the Chronic compression of the Nerves causes symptoms like twitching, Facial spasms, frequent Blinking, Headaches, Occular irritation, Rhinopyema.etc

In all the Symtpomatic cases MRI is the most preferable choice of investigation with high accuracy.

Though Surgical Management had high success rates via microvascular decompression surgery, still many of the cases have post operation Facial weakness, and other complications. Homoeopathic Remedies which act dynamically upon the vital force helps in relieving the Symptoms to a large extent though it cannot change the anatomical positions of the aberrant vessels and its compression effects. Here in the above cases Silicea and Calcarea carb selected on the basis of totality and Repertorisation, had succesfully relieved the frequency and intensity of Hemi Facial Spasms.

## CONCLUSION:

The cases of Primary Hemifacial Spasms caused by Neurovascular Compressions of prolonged duration were relieved symptomatically with no recurrence within few months of Homoeopathic treatment. The present case report serves as a valuable clinical observation although conclusions cannot be drawn from isolated experiences.

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