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The Role Of Homoeopathy In Managing Charcot-Marie-Tooth Disease: Integrating Vitalist And Biomedical Perspectives

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Abstract:

Charcot-Marie-Tooth (CMT) disease is a complex, inherited neurological disorder affecting peripheral nerves, leading to muscle weakness and sensory impairments. Despite advances in conventional medicine, Homoeopathic approaches have gained attention for their holistic and patient-centered methodologies. This article explores the potential role of Homoeopathy in managing CMT, comparing it with the biomedical model. The article reviews the vitalist principles of Homoeopathy, specifically how they align with genetic theories such as gene expression and epigenetics. It highlights how Homoeopathy's concept of miasms—deep-seated imbalances—parallels epigenetic modifications seen in conventional medicine, where environmental factors trigger gene expression changes. The comparison reveals overlaps between the two models, suggesting a complementary approach to managing CMT. Furthermore, the article discusses current treatments and the role of PXT3003, a pharmacological treatment for CMT, alongside Homoeopathic remedies aimed at balancing the vital force. The possibility of integrating Homoeopathy with modern treatments presents a promising approach for improving quality of life in CMT patients.

Keyword- CMT, vitalist principle, peripheral neuropathy, genetic expersion.

INTRODUCTION-

Charcot-Marie-Tooth (CMT) disease, also known as hereditary motor and sensory neuropathies. It is the most common inherited neuromuscular disorder. CMT was first described as a clinical entity in 1886 by physicians Jean-Marie Charcot, Pierre Marie, and Howard Henry Tooth, who referred to it as "progressive muscular atrophy."[1] CMT exhibits a wide range of clinical, electrophysiological, genetic, and pathological characteristics. Traditionally, it was divided into two main types based on neurophysiological data: the demyelinating form, known as CMT type 1 (CMT1), and the axonal form, referred to as CMT type 2 (CMT2). However, with the advent and growing use of next-generation gene sequencing (NGS) technologies, the way CMT is classified and diagnosed has evolved. [2][3]

CMT is a disorder that affects nerves in a length-dependent manner, leading to gradually worsening foot deformities (commonly pes cavus), sensory impairment, weakness in the legs, and diminished or absent deep tendon reflexes. Most people with CMT start showing symptoms during childhood or adolescence, typically experiencing a slow onset of weakness that begins in the lower limbs and eventually progresses to the upper limbs..[4] The clinical features of CMT can resemble those of other neurodegenerative disorders. CMT and its related conditions are genetically inherited, involving mutations in over 100 different genes. Approximately 80% to 90% of these genetic abnormalities result from copy number variations (CNVs) in the peripheral myelin protein 22 (PMP22) gene, along with mutations in the GJB1, MPZ, and MFN2 genes. A smaller number of families are affected by mutations in other genes, while 18% to 50% of cases remain unresolved, possibly due to mutations in noncoding regions of DNA or structural variations. Structural variations refer to genomic alterations that affect chromosomal organization, including duplications, deletions (also known as CNVs), insertions, inversions, and translocations.

[5].

There are several types of CMT, each differing in genetics, inheritance, and whether the myelin sheath or the nerve axon is affected:

1. CMT1 (Myelin Sheath Dysfunction)

- **Cause:** Damage to the myelin sheath. dominant mode of inheritance, early onset, distal motor weakness, and moderate slowing of NCVs (>15 to ≤35 m/s).
- **Key Types:**
  - **CMT1A:** Caused by a duplication of the *PMP22* gene, leading to muscle weakness, atrophy in the lower legs, hand weakness, and foot deformities, progressing slowly from childhood.
  - **CMT1B:** Caused by mutations in the *MPZ* gene, affecting myelin protein zero (P0) and presenting similar symptoms to CMT1A.
  - **Other CMT1 Forms:** Include mutations in genes like *SIMPLE (LITAF)*, *EGR2*, *NEFL*, and *PMP22*.

2. CMT2 (Axonal Damage)

- **Cause:** Direct damage to the axon of peripheral nerves. onset typically in the second or third decade, distal motor weakness, and normal or slightly slowed NCVs (>40 m/s).
- **Symptoms:** Generally milder than CMT1, with less sensory loss. Onset is typically in childhood or adolescence. Some subtypes can also affect speech and breathing.

3. CMT3 (Dejerine-Sottas Disease) CMT3 has its onset in infancy with symptoms such as hypotonia, feeding difficulties, severe sensory and motor impairments, and profound slowing of NCVs (≤15 m/s)

- **Cause:** Severe demyelination due to mutations in genes like *PMP22*, *MPZ*, and *EGR2*.
- **Symptoms:** Early onset with severe muscle weakness, sensory loss, and spinal deformities.

4. CMT4 (Autosomal Recessive Types) CMT4 presents with progressively severe sensory and accompanied by motor symptoms and moderate slowing of NCVs (>15 to ≤35 m/s)

- **Cause:** Rare mutations affecting the myelin sheath or axon, passed through autosomal recessive inheritance.
- **Symptoms:** Often starts with leg weakness in childhood, leading to significant disability by adolescence.

5. CMTX (X-linked Inheritance) CMTX follows an X-linked mode of inheritance with onset typically in the first or second decade. Symptoms are generally more severe in males than females, featuring gait impairment and mild slowing of NCVs (>35 to ≤45 m/s)

- **CMTX1:** The second most common form, caused by mutations in the *connexin-32* gene. Males experience moderate to severe symptoms, while females often have milder or no symptoms.

These types of CMT vary in severity, progression, and the specific nerve structures affected, depending on the underlying genetic mutation.

Epidemiology

CMT is a neuropathic ailment that affects people of various ethnic backgrounds and is both clinically and genetically diverse. It is a global illness. In general, 1 in 2500 individuals are thought to experience CMT.[6]
A nerve biopsy, performed with informed consent, can help determine the genetic cause in sporadic cases of CMT and differentiate it from acquired conditions like chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). It can also clarify the significance of genetic findings, such as "variants of uncertain significance" or novel mutations. By examining morphological and ultrastructural changes in axons, myelin, nodes of Ranvier, and mitochondria, the biopsy offers insights into the role of mutated genes and the mechanisms driving disease progression. Prolonged demyelination and remyelination can cause basal lamina growth or concentric Schwann cell cytoplasm, which can result in the formation of multilayered "onion bulbs."[7] In CMT1, axonal loss advances with time, although demyelination stays constant. Sometimes, "tomacula"—a defining sign of hereditary neuropathy with a predisposition to pressure palsies—may be discovered during a nerve biopsy in a patient with CMT1A.[7] Tomacula are longitudinally oriented multifocal hypermyelinating processes that resemble a "chain of sausages."[8]

Physical examination

A thorough neurological examination is essential during the physical assessment of CMT. Common signs across all CMT types include distal, symmetrical weakness in the feet and legs, muscle atrophy, diminished or absent tendon reflexes, and skeletal deformities. Gait evaluation is particularly important, as muscle weakness can lead to foot drop and a high-stepping gait. Proximal muscle weakness in the upper limbs is rare, but hand weakness often presents as difficulty with tasks like buttoning, zipping, or writing. Approximately 20% to 30% of CMT patients experience musculoskeletal pain rather than neuropathic pain, while paresthesia and other sensory symptoms are less common.
A physical examination may identify foot deformities like high arches (pes cavus) or hammer toes, along with subtle wasting of the hypothenar muscles in patients with prolonged disease, caused by weakness in the small muscles of the hand. Muscle atrophy in the lower legs and thighs can result in what is referred to as a "stork leg deformity." Weakness, loss of proprioception, and skeletal deformities, such as pes cavus and hammer toes, can lead to an abnormal gait and instability.[9]. Symptoms of dysautonomia, such as urinary urgency and incontinence, orthostatic hypotension, and excessive sweating (hyperhidrosis), have also been observed in individuals with CMT[10]. Restless legs syndrome is more prevalent in patients with CMT as compared to the general population.[11] . Regardless of the CMT subtype, axonal loss heightens the excitability of primary sensory nerves in the leg muscles, resulting in creeping sensations. [12] Additional rare features include hyperkeratosis, loose skin (hyperlaxity), joint contractures (arthrogryposis), cognitive impairment, learning disabilities, lip or chin muscle twitching (myokymia), breathing difficulties, tremors, nystagmus, lack of coordination (ataxia), muscle fasciculations, cold-induced cramps, hip dysplasia, and Ehlers-Danlos syndrome. [13]

Homoeopathy correlation with genetic theory

Now we will see the how their is correlation between two theory, vital principle, which is believed to regulate health and the genome, which governs biological functions. Both phenomena—vitalism and genetic expression—are described as complex, dynamic and self-organizing systems. The vital force, responsible for maintaining bodily functions and balance, is compared to the genome (exome and epigenome), which stores biochemical information for protein production and vital processes.

Key correlations include:

1. **Non-linearity**: Both the Homoeopathic principle and gene expression operate in a non-linear way, where small inputs (like diluted Homoeopathic remedies or genetic mutations) can cause disproportionately large effects.
2. **Self-organization**: The vital force and genome both exhibit emergent behavior from local interactions within the body, forming complex networks that regulate health and disease.
3. **Dynamism**: Both systems are dynamic, adapting to internal and external stimuli, which is reflected in how Homoeopathic remedies and gene expressions respond to changes in the environment or the body's state.

According to Palazzo,the vital force is related to ‘vortex’, the emerging phenomenon that indicates the presence of a force that comes from within the system, the result of the very masses in circular motion that animate the phenomenon, experimentally evident in the cell membrane. [14] This emerging phenomenon (vortex) corresponds to the concept of force centres or *chakras* (‘wheels’ in Sanskrit) from Traditional Hindu Medicine, according to which energy vortices that are spread across the body's surface vibrate continuously and distribute vital energy or *prana* to the physiological systems and body components.[15]

This discussion on the "vortex" model, used to describe the vital force and genetic information transmission, suggesting that the vital force or genetic expression is a force emerging from within the system. This concept of a self-emergent, internal force is linked to the idea of how genes or the vital principle modulate physiological and health outcomes.

- Charcot-Marie-Tooth Disease (CMT): A Link between Homoeopathic Vitalism and the Biomedical Model

CMT is a genetic disorder that affects the peripheral nerves, leading to muscle weakness and sensory problems. It occurs due to mutations in certain genes that control the function of the peripheral nerves, affecting either the nerve fibers (axons) or the myelin sheath, which protects and insulates these nerves. The disorder is inherited and symptoms gradually progress over time, often leading to mobility issues.

- Biomedical Model and CMT

In the biomedical framework, diseases like CMT are understood primarily as genetic disorders. Mutations in genes like PMP22, MPZ, or GJB1 alter protein production in a way that disrupts nerve function. These genetic errors directly impact the exome (the part of DNA that codes for proteins) and are regulated by the epigenome (the non-coding portion of the genome that controls gene expression).

In this context, CMT manifests due to the interaction of genetic mutations with environmental influences that may alter how these genes are expressed. The biomedical approach acknowledges that while the inheritance of faulty genes causes CMT, factors like diet, stress, and exposure to toxins may modify the severity and progression of symptoms through epigenetic changes.

- Homoeopathic Vitalist Model and CMT

From a Homoeopathic perspective, disease arises when the vital force—the non-material, dynamic principle that maintains health—becomes imbalanced. In the case of CMT, this vital disturbance is seen as a chronic miasm that predisposes individuals to nerve degeneration. Miasms, in Homoeopathy, are thought to be inherited energetic disturbances that weaken the body's natural defenses, leading to chronic diseases.

In this framework, CMT is viewed not just as a result of genetic mutations but as a reflection of a deep-seated vital imbalance passed down through generations. External factors such as emotional stress, trauma or unhealthy habits might trigger the activation of this latent miasm, similar to how environmental triggers influence epigenetic expression in the biomedical model.

- Comparison: Miasms and Epigenetic Modifications in CMT

Both Homoeopathy and modern biomedicine recognize that external factors can influence the manifestation of hereditary diseases. In Homoeopathy, miasms—energetic imbalances inherited across generations—are believed to be triggered by lifestyle or emotional factors, leading to chronic disease. In biomedicine, epigenetic modifications alter the expression of disease-causing genes, influenced by external stimuli such as stress, diet or toxin.

- Integrating Homoeopathy and Biomedicine in Understanding CMT

Both Homoeopathic vitalism and the biomedical model offer valuable insights into diseases like Charcot-Marie-Tooth disease (CMT). Homoeopathy focuses on the energetic imbalance inherited across generations, while biomedicine focuses on genetic mutations and their regulation by the epigenome. Both recognize the influence of environmental factors on disease progression, with Homoeopathy emphasizing the restoration of vital balance and biomedicine focusing on gene expression and regulation.

Treatment and management

New developments in science and medicine have shown that drugs like PXT3003, which combines naltrexone, baclofen, and d-sorbitol, are capable of reducing the harmful consequences of PMP22 overexpression in humans as well as animals. When compared to placebo, subjects treated with PXT3003 did not exhibit any decline or improvement in the 10-meter walk test, conduction velocities, overall neuropathy limitations scale (ONLS) or CMT neuropathy score (CMTNS). PXT3003 was safe and well-tolerated. [16]

An interprofessional healthcare team is essential in managing patients with Charcot-Marie-Tooth (CMT) disease who are at risk of limited mobility, contractures and skeletal deformities. Physical therapy focuses on stretching, grip exercises, aerobic conditioning, resistance training and using orthotic devices such as supportive shoes to correct foot drop and assist with walking. Some patients may also need canes or forearm crutches for stability. Physical therapy aids in maintaining muscle strength, enhancing joint flexibility, improving balance and promoting cardiovascular fitness.

PROGNOSIS

Charcot-Marie-Tooth (CMT) is a group of slowly progressing disorders that cause gradual weakness and muscle wasting in the limbs, affecting mobility and the daily activities. The rate at which the condition advances differs depending on the type of CMT. While life expectancy typically remains normal, early onset can lead to more severe symptoms. Regular assessments and interventions from an interprofessional

rehabilitation team are crucial to support independence, ensure safe movement, and maintain functionality in daily tasks.

REPERTORIAL RUBRICS CAN BE OF USE IN CMT

[KENT] Extremities –inversion;feet- calc, cic, lyc,merc, nux v, petr, PSOR rhus t,sec, sil, sol-n, sulph

[SYNTHESIS] Extremities; give away; feet: carb-an cic croto-t nit-ac sulph
[SYNTHESIS] Extremities; hammer toes: prot
[SYNTHESIS] Extremities; shortened muscles and tendons; feet: ***CAUST***
[SYNTHESIS] Extremities; unsteadiness; feet: ***acon*** agar camph carbn-s gall mag-m merc PHYS plb sumb

[PHATAK] [PHATAK A-Z]FEET:ARCHES: (2) 1 Ruta, 1 Sil

[BOENNING] [SENSATION AND COMPLAINTS IN GENERAL]EMACIATION:ATROPHY IN GENERAL: (71)

4 ARS, **4 CHIN**, **4 GRAPH**, **4 IOD**, **4 LYC**, **4 NAT-M**, **4 NUX-V**, **4 STANN**, **4 SULPH**, **3 Ambr**, **3 Arg-m**, **3 Bar-c**, **3 Bry**, **3 Calc**, **3 Canth**, **3 Caust**, **3 Cham**, **3 Clem**, **3 Cocc**, **3 Cupr**, **3 Ferr**, **3 Guai**, **3 Ign**, **3 Ip**, **3 Lach**, **3 Merc**, **3 Nit-ac**, **3 Op**, **3 Petr**, **3 Ph-ac**, **3 Phos**, **3 Plb**, **3 Puls**, **3 Rhus-t**, **3 Sars**, **3 Sec**, **3 Sil**, **3 Stront**, **3 Verat**, *2 Alum*, *2 Anac*, *2 Ant-c*, *2 Bor*, *2 Cina*, *2 Con*, *2 Dros*, *2 Dulc*, *2 Hep*, *2 Kali-c*, *2 Mag-c*, *2 Mez*, *2 Nat-c*, *2 Ruta*, *2 Samb*, *2 Sel*, *2 Sep*, *2 Staph*, *2 Stram*, 1 Am-c, 1 Arg-n, 1 Arn, 1 Calc-p, 1 Carb-v, 1 Dig,,etc..

[BOERICKE] [GENERALITIES]MARASMUS (EMACIATION, ATROPHY, WASTING): (62)

3 Abrot, **3 Acet-ac**, **3 Arg-n**, **3 Ars**, **3 Bar-c**, **3 Calc**, **3 Calc-p**, **3 Calc-sil**, **3 Ferr-p**, **3 Hep**, **3 Hydr**, **3 Iod**, **3 Merc-c**, **3 Nat-m**, **3 Ol-j**, **3 Ph-ac**, **3 Plb**, **3 Plb-acet**, **3 Samb**, **3 Sanic**, **3 Sars**, **3 Sil**, **3 Sulph**, **3 Syph**, **3 Thuj**, **3 Tub**, **3 Verat**, *2 Ant-i*, *2 Arg-m*, *2 Ars-i*, *2 Carb-an*, *2 Carb-v*, *2 Caust*, *2 Cetr*, *2 Cinch*, *2 Clem*, *2 Ferr*, *2 Fl-ac*, *2 Glyc*, *2 Helon*, *2 Kali-i*, *2 Kali-p*, *2 Kreos*, *2 Led*, *2 Mang*, *2 Merc*, *2 Nat-c*, *2 Op*, *2 Phos*, *2 Phyt*, *2 Plb-i*, *2 Psor*, *2 Rhus-t*, *2 Ricin*, *2 Sec*, *2 Sel*, *2 Stann*, *2 Staph*, *2 Ter*, *2 Uran*, *2 Vanad*, *2 Zinc-m*

[BOGER] [GENERALITIES]EMACIATION, ATROPHY, ETC: (21)

3 Ars, **3 Calc-p**, **3 Iod**, **3 Plb**, *2 Calc*, *2 Chin*, *2 Lyc*, *2 Nat-m*, *2 Nit-ac*, *2 Sars*, *2 Sulph*, 1 Arg-m, 1 Bar-c, 1 Caust, 1 Ferr, 1 Graph, 1 Nux-v, 1 Op, 1 Phos, 1 Sil, 1 Tub

[KENT] EXTREMITIES; NUMBNESS, INSENSIBILITY; FEET:

abrot acet-ac ACON acon-f ***act-sp*** adan agar aids ***ALUM*** ALUM-MET alum-p ALUM-SIL ALUMN AM-C AMBR ANT-C ANT-T APIS ARG ***ARG-N ARS asar*** BAPT BERB ***bry CALC*** CALC-P CAMPH CANN-I CARB-AN CARB-V CARBN-S CAUST ***CHAM*** c CIMX ***COCC COLOC*** com ***CON electr euph ferr*** ferr-ar ferr-i form gado-n galph GLON ***GRAPH*** ham hell HELO HYPER ign iod jug-c KALI-AR kali-bi KALI-C lat-m ***laur lepro*** lept lim ***LYC*** m-arct m-aust ***mag-m*** MAG-S maland MANG mang-acet ***MILL*** morg morph musc-d naja-h naja-m nat-ar NAT-C ***nat-m*** NIT-AC ***NUX-V*** OLND petr ***PH-AC PHOS*** PHYS pic-ac pitu-a ***plac*** PLAT PLB ***plb-acet*** psor ***PULS*** rhod RHUS-T ros-g sabad sapin sapo ***SEC SEP*** SIL sphing staph

stict STRAM ***sul-ac*** sulfa ***SULPH*** sumb syph tax thal thal-s ***THUJ*** tung ulm-c upa verat verat-v vip xan ***ZINC*** zinc-p zing

[BOERICKE] [LOCOMOTOR SYSTEM]GAIT:STAGGERING, UNSTEADY, DIFFICULT WALKING: (44)

3 Agar, **3 Arg-n**, **3 Bell**, **3 Carbn-s**, **3 Caust**, **3 Cocc**, **3 Con**, **3 Gels**, **3 Lath**, **3 Mygal**, **3 Nux-v**, **3 Oxyt**, **3 Phos**, **3 Sec**, **3 Zinc-m**, *2 Acon*, *2 Agro*, *2 Ang*, *2 Asar*, *2 Aster*, *2 Astra-mo*, *2 Calc-p*, *2 Colch*, *2 Dubin*, *2 Helo*, *2 Ign*, *2 Lac-ac*, *2 Lil-t*, *2 Lol*, *2 Mang*, *2 Merc*, *2 Morph*, *2 Mur-ac*, *2 Nat-c*, *2 Nux-m*, *2 Onos*, *2 Paeon*, *2 Ph-ac*, *2 Rhus-t*, *2 Sep*, *2 Stram*, *2 Sulfon*, *2 Tab*, *2 Trion*

[PHATAK] [PHATAK A-Z]GAIT:STAGGERING: (10)

Alum, 1 Calc, 1 Helo, 1 Lil-t, 1 Mur-ac, 1 Nat-c, 1 Onos, 1 Pic-ac, 1 Puls, 1 Verat-v

[SYNTHESIS REPERTORY]- EXTREMITIES PARALYSIS;PROGRESSIVE- caust.,con. *Lath. Plb.*

[SYNTHESIS] ; EXTREMITIES ;CONTRACTION of muscles and tendon; Feet- acon. alum. am-m. berb. bism. Cann-s. cann-xyz. carb-an. Caust. cham. Colch. Etc.,

Conclusion:

Homoeopathy offers a unique and promising approach to managing Charcot-Marie-Tooth (CMT) disease, emphasizing the balance of the vital force as a key component of health. Unlike conventional medicine, which focuses on genetic mutations and physical symptoms, Homoeopathy addresses the underlying energetic disturbances, or miasms, that predispose individuals to chronic diseases like CMT. By focusing on individualized treatment and the dynamic relationship between mind, body, and environment, Homoeopathy seeks to restore balance at a deeper level than symptomatic relief.

Homoeopathic remedies, tailored to each patient's constitution, offer a potential pathway to mitigate the progression of CMT by correcting vital imbalances. This approach, coupled with the recognition of environmental triggers similar to epigenetic factors in the biomedical model, presents a more holistic way of managing CMT. While further clinical studies are required to establish Homoeopathy's efficacy in treating CMT, it holds promise as a complementary therapy that could enhance the quality of life for patients by addressing both physical and energetic aspects of this complex condition. The integration of Homoeopathy into a comprehensive care plan could mark a significant step forward in personalized treatment for those living with CMT.

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