Salient Visionary Publications

Neural Therapy in Migraine: Clinical Evidence for a Holistic Therapeutic Approach – Analysis of 464 Cases

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ABSTRACT

Background: Migraine is one of the most common neurological disorders and significantly impairs quality of life. While conventional therapies may alleviate symptoms, they often fail to address the underlying causes. Neural therapy is a therapeutic approach in which local anesthetics such as procaine or lidocaine are not primarily used for analgesia but rather to modulate the autonomic nervous system, particularly its sympathetic branch. This method aims to restore functional balance and stabilize dysregulated networks through segmental and systemic reflex pathways, thereby supporting a holistic regulatory effect.

Objective: This retrospective study aims to evaluate the effectiveness of neural therapy in 464 migraine patients using a holistic, regulatory medical approach.

Methods: Patient history forms were analyzed for hormonal dysregulation, intestinal dysbiosis, temporomandibular dysfunction, C2 vertebral blockage, hydration status, and interference fields. Additionally, clinical treatment progressions were statistically assessed.

Results: Over 91% of patients demonstrated clinical improvement, and 60% became symptom-free. The most frequently observed contributing factors were intestinal dysbiosis (89%), hormonal imbalance (71%), and temporomandibular/C2 dysfunctions (43% each).

Conclusion: Neural therapy is an effective and regulatory therapeutic option for migraines, particularly in chronic, multifactorial cases. Its integration into a holistic treatment strategy may yield substantial clinical benefits.

Keywords: Neural therapy; Autonomic nervous system; Local anaesthetic (Procaine, Lidocaine); Migraine; Regulatory approach

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1. Introduction:

Migraine is a complex, genetically influenced neurological disorder characterized by episodic moderate to severe headaches. These headaches are often unilateral and pulsating and frequently accompanied by nausea, vomiting, photophobia, and phonophobia. The term "migraine" is derived from the Greek word hemicrania ("half of the skull"), which entered medical terminology through the Latin hemicrania and the French migraine.[1,2,3,5,9,10]

Migraine is among the leading causes of disability worldwide and substantially impairs quality of life and productivity.[1,3,5] The World Health Organization (WHO) classifies severe migraine attacks as one of the most disabling medical conditions, with considerable impact on occupational performance and daily functioning (WHO, 2024). It is not merely a “headache” but a dynamic, multifaceted neurological disorder that can evolve over hours to days and often becomes a recurrent chronic condition for many patients.[1,5,7,12,13,14]

2. Types of Migraine:

The International Headache Society (IHS) classifies migraines into several subtypes.[1,13,15,16,17]

2.a. Migraine without aura:

This is the most common form, accounting for approximately 75% of all cases. It is characterized by recurrent headache episodes lasting between 4 and 72 hours without preceding neurological symptoms. These headaches are typically intense and accompanied by common vegetative symptoms such as nausea, photophobia, and phonophobia.[1,2,3,9,11,18]

2.b. Migraine with aura:

This form includes transient neurological symptoms that precede or accompany the headache phase. These symptoms are fully reversible and may involve visual disturbances, sensory alterations, speech difficulties, motor weakness, or retinal symptoms. [1,2,3,9,11,15,18]

2.c. Chronic migraine:

It is defined as a headache occurring on 15 or more days per month for more than three consecutive months, of which at least eight days meet the diagnostic criteria for migraine. [1,9,11,15]

2.d. Probable migraine:

Refers to migraine-like episodes that do not fulfill all diagnostic criteria or overlap with features of other primary headache disorders. [1,4,9,19,20]

3. Phases of a Migraine Attack:

Migraine often follows a four-phase progression, each characterized by distinct clinical symptoms and pathophysiological mechanisms. Recognizing these phases enables the development of individualized treatment strategies and targeted patient support. [1,2,3,5,18,65]

Prodromal Phase: This phase typically begins 24 to 48 hours before the onset of headache and affects approximately 75% of patients. Common prodromal symptoms include:[1,3,9,11,18,64,65]

- Irritability, mood swings, depressive tendencies
- Fatigue, yawning, thirst, neck stiffness
- Sensitivity to light and noise
- Difficulty concentrating, restlessness
- Cravings for specific foods (e.g., sweets or salty snacks)

These symptoms suggest hypothalamic network activation, particularly involving dopaminergic pathways, highlighting their potential as therapeutic targets.

Aura Phase: Approximately 25% of migraine patients experience an aura, which reflects transient changes in cortical activity and blood flow. Auras are predominantly visual (e.g., flashing lights, zigzag lines, scotomas) but can also be sensory, motor, or speech-related. They usually last less than one hour and are fully reversible. [1,2,3,5,9,18]

Headache Phase: The headache phase represents the core feature of a migraine attack. It typically begins as a unilateral, pulsating pain, intensifies progressively, and may last several hours to days.[1,2,3,5,9]

Associated symptoms include:

- Nausea and vomiting
- Sensitivity to light, sound, and odors
- Tearing, nasal congestion
- Cutaneous allodynia (sensitivity to touch)
- Withdrawal into dark, quiet environments; relief often achieved through sleep

Postdromal Phase: Following headache resolution, patients may experience fatigue, difficulty concentrating, residual pressure at the site of pain, or emotional changes (e.g., exhaustion and occasionally euphoria) lasting from several hours to days. [1,2,3,5,9,18]

4. Episodic Syndromes Associated with Migraine:

Migraine may present not only as a typical headache but also in the form of episodic syndromes, especially in pediatric populations:

- Recurrent gastrointestinal disturbances: Periodic abdominal pain, nausea, or vomiting in association with migraine episodes.[1,3,11,18]
- Benign paroxysmal vertigo: Recurrent episodes of vertigo, mainly observed in children.[1,3,4,18]
- Benign paroxysmal torticollis: Temporary tilting of the head, occurring in infants and young children.[1,2,3,4,19]

5. Complications of Migraine:

In addition to the disabling burden of migraine itself, several complications may arise:[1,2,3,18,19]

- Status migrainosus: A debilitating migraine attack persisting for more than 72 hours.[9,11,18,20]
- Persistent aura without infarction: Aura symptoms lasting more than one week without radiological evidence of cerebral infarction.[18,20,21]
- Migrainous infarction: A stroke occurring in association with a typical migraine aura.[12,22]
- Migraine-triggered seizure (migralepsy): A seizure provoked by a migraine aura.[3,7,8,17]

6. Diagnosis and Management of Migraine

The diagnosis of migraine requires a thorough patient history, detailed physical examination, and application of the diagnostic criteria defined by the International Headache Society (IHS).[7,14] Given migraine's heterogeneous nature, a personalized and multimodal treatment strategy is essential for effective management.

Acute Therapy: Acute pharmacological treatment aims to relieve symptoms during an ongoing migraine attack. First-line medications include triptans, non-steroidal anti-inflammatory drugs (NSAIDs), and antiemetics, which can be used individually or in combination depending on symptom severity and individual response.[1,3,16]

Prophylactic Therapy: Prophylactic strategies are indicated for patients with frequent or severely disabling migraine attacks. These include:

- Pharmacological interventions include beta-blockers, antiepileptic drugs, and calcitonin gene-related peptide (CGRP) inhibitors.
- Lifestyle modifications (e.g., regular exercise, hydration, regulated sleep patterns).
- Non-pharmacological approaches include relaxation training, biofeedback, and physical therapy.[1,3,17]

Individualized Treatment Strategies: A personalized approach is critical in migraine management and may include:

- Identify and avoid individual triggers (e.g., stress, specific foods, hormonal fluctuations).
- Dietary adjustments, including the elimination of known migraine-provoking substances.
- Stress management techniques, such as mindfulness or cognitive behavioral therapy.
- Implement proper sleep hygiene practices to reduce attack frequency and intensity.[1,9,10,18]

This integrative management paradigm highlights the importance of addressing migraine's acute and long-term dimensions, focusing on patient-specific factors and comorbidities.

7. Etiology of Migraine

A thorough understanding of the underlying causes of migraine is essential for accurate diagnosis, effective prevention, and sustainable treatment strategies. Migraine is considered a multifactorial disorder arising from a complex interplay of genetic predispositions, neurological dysregulation, and environmental influences. Central to its pathophysiology is the concept of abnormal neuronal activity, which leads to impaired nerve impulse processing, altered neurotransmission, and dysregulated vascular dynamics within the central nervous system.[1,2,3,4,5,9,11,12]

7.1. Genetics and Heredity

The genetic basis of migraine is well established: individuals with a positive family history are approximately three times more likely to develop migraines than those without.However, the inheritance pattern is not monogenic but rather polygenic, involving multiple genes that contribute to susceptibility.[1,19,26] In addition, environmental factors influence both the individual risk and the frequency and severity of migraine attacks.[1,5,9,11,26]

7.2. Environmental and Neurological Triggers

Migraine attacks are often precipitated by individual-specific triggers that influence neuronal excitability, vascular tone, or neurotransmitter balance. In retrospective studies, approximately 76% of patients could identify at least one personal trigger.[1,3,9,11,18,26,34–39]

7.3. Commonly Reported Triggers:[1,3,5,11]

- **Stress:** The most frequently cited trigger (80%)
- **Hormonal fluctuations:** Approximately 65% report attacks associated with menstruation, ovulation, or pregnancy
- **Skipped meals / irregular eating:** Fasting or inconsistent meal patterns (57%)
- Weather changes: Especially fluctuations in barometric pressure (53%)
- **Bright or flickering lights:** (38%)
- **Alcohol consumption:** Particularly red wine (38%)

7.3. Other Potential Triggers:[1,2,3,18,62,63]

- Sleep disturbances: Both sleep deprivation and oversleeping (50%)
- Strong odors: Perfumes, solvents, exhaust fumes (40%)
- Neck pain or tension: (38%)
- Heat exposure: (30%)
- Specific foods: Suspected triggers include aspartame, tyramine, and chocolate

7.4. Rare or Inconclusive Triggers:[1,11,18,63]

- Smoking: Suspected in 36% of patients, though not conclusively proven
- Physical exertion: Trigger in approximately 22% of cases
- Sexual activity: Very rarely reported (<5%)

Conclusion: This broad overview of potential migraine triggers—from genetic and metabolic to environmental and lifestyle related factors—underscores the importance of a personalized and interdisciplinary treatment approach. Ongoing research into migraine's molecular and neurophysiological mechanisms continues to inform novel targeted therapeutic strategies. Regulatory therapies, such as neural therapy with local anesthetics (e.g., procaine), offer promising potential. These interventions aim to modulate the autonomic nervous system, contributing to acute symptom relief and long-term prophylaxis.[11,18,32,33,37–50]

8. Epidemiology of Migraine

Migraine is among the most prevalent and disabling neurological disorders worldwide. It is estimated that approximately 12% of the global population is affected, with significant variations based on sex, age, and geographic region.[2,3,11,19]

8.1. Global Prevalence

Migraine affects around 17% of women and 6% of men annually. The markedly higher prevalence in women is primarily attributed to hormonal fluctuations during menstruation, pregnancy, and menopause. In childhood, migraine is initially more common in boys, but this trend reverses after puberty, favoring girls. The peak prevalence is observed between the ages of 35 and 39, representing the most productive years of life. With increasing age—particularly after menopause—migraine frequency tends to decline.[1,2,3,5,9,11,19]

8.2. Regional Differences

The highest prevalence rates are reported in North and South America and Europe, while Asia and Africa show comparatively lower rates. Genetic factors, lifestyle, environmental exposures, and access to healthcare and diagnostic resources can explain these regional differences.[1,3,11,18,51]

8.3 Burden and Health Economics

Migraine is the second leading cause of years lived with disability (YLD) globally, second only to lower back pain. It accounts for approximately 3% of all annual emergency room visits and ranks among the top five reasons for seeking urgent medical care.[1,3,51]

Familial aggregation is notable: if one parent suffers from migraine, the risk for offspring is approximately 40%, increasing to up to 75% if both parents are affected.

8.4. Socioeconomic Impact

The economic burden of migraine is substantial. Lost productivity, work absenteeism, frequent medical consultations, and repeated medication use impose a significant strain on both individuals and the healthcare system. Due to its chronic and episodic nature, migraine not only impairs occupational functioning but also negatively affects social and family life.[1,2,3,19–21,64,65]

9. Therapeutic Considerations

Given the substantial individual and societal burden posed by chronic pain syndromes, there is a growing need for low-risk, cost-effective, and patient-centered treatment modalities. In this context, neural therapy is gaining recognition as a powerful and modern form of regulatory medicine.[18,27,32,33,42,43,52–54]

Neural therapy—an established and time-tested intervention—utilizes targeted injections of local anesthetics such as procaine or lidocaine to modulate autonomic reflex circuits and restore regulatory balance within the autonomic nervous system.[11,18,53–60] Through its central and peripheral mechanisms of action, neural therapy is effective in treating both acute and chronic conditions.[42,44,61]

The method is characterized by its simplicity, high degree of control, and excellent tolerability. In acute management and preventive care, neural therapy has proven highly effective, particularly in general medical, pain management, and integrative medicine settings. It is increasingly recognized as a scientifically grounded and resource-efficient standard therapy whose role in modern medicine deserves further expansion.[11,18,26,27,31–33,42–44,52–54,66,67]

Conclusion: Migraine is a dynamic and multifaceted neurological disorder with far-reaching consequences on quality of life. Although not every patient experiences all four phases, a detailed understanding of this progression supports the design of more individualized therapeutic approaches [18].

In addition to pharmacological and lifestyle-based interventions, neural therapy emerges as an effective, practical, and economically viable option. Restoring autonomic balance can play a valuable role in both providing acute relief and achieving long-term stabilization of migraine symptoms.[9, 11, 18, 32, 40, 42, 44, 49, 53–58]

MIGRAINE – A DIFFERENT PERSPECTIVE

Headaches and migraines rank among the most prevalent complaints in Western industrialized nations—with an alarming rise observed in younger populations. Already in 1997, data indicated that nearly 80% of schoolchildren in Germany experienced occasional to frequent headaches or even migraines.[11,18]

Multifactorial Origins – A Holistic View

Despite intensive research, the precise etiology of migraine remains incompletely understood. Multiple hypotheses have been proposed, including altered vascular regulation, neurotransmitter imbalances, and disturbances in sensory processing within the brainstem. Specific genetic variants—such as those found in familial hemiplegic migraine—also appear to contribute.[1–3,18]

Additionally, visual disturbances, neuralgias (e.g., trigeminal neuralgia), temporomandibular joint dysfunction, or brain tumors can produce similar symptoms. Conventional medical treatment currently focuses on symptomatic relief: triptans for acute episodes and beta-blockers or antidepressants for prophylaxis. However, a causal cure is not yet available.[9,11,49,55,57,58]

Regulatory and Neural Therapy Perspectives on Migraine

From the standpoint of regulatory medicine, migraine is not merely viewed as an isolated symptom but rather as a manifestation of an underlying functional imbalance in systemic regulation. The focus extends beyond identifying specific triggers to include a deeper understanding of neurovegetative interconnections. This leads to individualized treatment concepts beyond purely symptomatic management.[9,11,18,27–29,49]

1. The Musculoskeletal System – Posture, Biomechanics, and Structural Balance

Postural asymmetries, joint restrictions, or muscle imbalances—particularly in the cervical spine, temporomandibular joint (TMJ), or sacroiliac joint (SIJ)—can contribute to headaches via myofascial chains. While the body compensates for these dysfunctions over time, they often result in chronic tension and pain. Temporary manipulations (e.g., spinal adjustments) may offer short-term relief, but a sustainable effect typically requires regulatory interventions, such as neural therapy, osteopathic techniques, or targeted trigger point therapy.[11, 18, 26, 32, 33, 37, 38, 44, 45, 49, 66, 69–74]

1.1 Musculoskeletal Perspective: Functional Relationships in Headache and Migraine

Headaches and migraines are often not isolated phenomena but instead reflect underlying functional disturbances of the musculoskeletal system. The cervical and thoracic spine, jaw joint, and surrounding neck and shoulder musculature play a central role. Crucially, the focus should not be limited to identifying dysfunctions in these areas, but must also include understanding their development and systemic interconnections.[18, 32, 33, 35, 43, 50, 72–75]

The sacroiliac joint (SIJ) is a frequently overlooked yet clinically significant structure—a stable, L-shaped articulation between the sacrum and iliac bones.

Dysfunction in this region often remains subclinical, as compensatory muscle activity—especially in the pelvic floor, lumbar extensors, and thigh musculature—initially masks the restriction. However, strain patterns develop along myofascial chains over time, extending cranially through anatomical tension lines.[32, 33, 35, 71, 73–76]

These fascial pathways, such as the superficial back or spiral lines, transmit mechanical tension from the pelvis to the shoulder girdle, cervical spine, and craniomandibular system. This often results in chronic muscular tension, postural overload, and adaptive movement patterns in the upper back and neck. The TMJ frequently reacts to such deep-seated imbalances with bruxism or occlusal dysfunction, which in turn can trigger migraine episodes via trigeminal afferents. [18,32,44,50,67,72–77]

1.2 Beyond Symptom Relief – Addressing Structural Causes

Functional complaints of this kind typically respond only temporarily to symptomatic treatments such as massage or manual relaxation techniques. However, the underlying causes—deep-seated restrictions, asymmetrical muscle activation, and disrupted fascial gliding—often remain undetected and untreated.[18, 49, 72, 73, 75, 76]

Therefore, an integrative therapeutic approach should target the pain site and analyze movement patterns and fascial tension dynamics. Manual medicine, fascial release techniques, and regulatory modalities such as neural therapy can help interrupt these myofascial chains and promote long-lasting relief. Only by addressing the structural basis can headaches and migraines be effectively treated at their roots.[32,33,44,72–74,76]

1.3 Perspective: Cranio-Cervical Junction

The craniocervical junction—the anatomical region between the upper cervical spine and the skull—represents one of the human body's most sensitive and complex areas. It comprises not only bony structures such as the atlantooccipital joint (C0–C1), atlantoaxial joint (C1–C2), and the functionally important sphenobasilar junction but also includes central autonomic centers, muscle insertions, ligamentous connections, and the upper spinal cord along with cranial nerve structures. [71–74,76]

This area is closely associated with several cranial nerves, particularly the trigeminal nerve (CN V), the vagus nerve (CN X), and the accessory nerve (CN XI). These nerves traverse or originate close to the craniocervical junction and are key conduits for sensory, motor, and autonomic information. Notably, the trigeminal nerve, whose nuclei extend into the upper cervical spinal segments (C1–C3), plays a central role in the pathogenesis of migraine and trigeminal-autonomic cephalalgias.[71–76,78,79]

Dysfunctions in this region—such as muscle imbalances, joint restrictions, fascial adhesions, or chronic low-grade inflammation—can initiate autonomic reflex patterns. These disturbances may not only contribute to headaches and migraines but are also associated with complex conditions such as tinnitus, vertigo, chronic fatigue, sleep disturbances, or even functional cardiac arrhythmias. The vagus nerve, in particular, is highly relevant due to its cervical trajectory, which makes it susceptible to mechanical irritation, with direct implications for visceral regulation.[71,73,74]

Given this zone's functional density and neurological sensitivity, therapeutic interventions must be conducted with utmost care. Craniosacral osteopathy, for example, offers a gentle approach to regulation using minimal pressure (typically 5–20 grams), demonstrating efficacy in modulating this delicate region.[71–74]

Neural therapy also provides a highly valuable treatment option for the craniocervical junction. Through targeted injections—applied to paraspinal cervical segments, occipital nerve points, or along the sphenobasilar junction—it is possible to modulate autonomic tension patterns, disengage interference fields, and soothe chronic irritative states.[11,18,80–84]

An integrative understanding of this region—incorporating structural, fascial, neurovegetative, and bioregulatory perspectives—is essential for the sustainable management of headaches, migraines, and a broad spectrum of other functional cranial complaints. [18]

1.4 Perspective: The Temporomandibular Joint (TMJ)

Temporomandibular joint (TMJ) dysfunction often goes unnoticed in clinical practice despite its substantial role in the development of chronic headaches, migraine, and other functional disorders. Typical indicators of craniomandibular dysfunction (CMD) include clicking or grinding sounds during jaw movement, limited range of motion, or asymmetrical tension in the masticatory muscles (e.g., masseter, temporalis, pterygoideus). These muscular imbalances can propagate via fascial and myofascial chains to adjacent structures, particularly the cervical spine and the craniocervical junction.[72–74]

CMD exemplifies a functional disorder with multisystemic effects. As von Heymann (2011, 2013) emphasizes, the TMJ is intricately connected to the upper cervical spine, the trigeminal system, and autonomic regulatory centers through sensorimotor feedback loops. Persistent misalignments or irritations in the TMJ region may cause nephrogenic pain and contribute to tinnitus, vertigo, difficulty concentrating, neck tension, sleep disturbances, and even functional cardiac arrhythmias.

The close functional relationship with the trigeminal nerve, a major relay for nociceptive stimuli from the cranial and facial regions, helps explain the clinical manifestations of CMD-related imbalances.[11,18,72–74]

Targeted interventions—such as functional jaw corrections, occlusal splint therapy, manual release of masticatory musculature, and adjunctive neural therapy—have demonstrated high efficacy in many cases.[11,18,72–74,76,78,79,82] An integrative diagnostic approach, which considers the TMJ of the cervical spine, the autonomic nervous system, and myofascial connectivity, is essential for the comprehensive management of patients with chronic pain syndromes.

Additional Perspectives (Brief Overview):

A holistic understanding of migraine and headache disorders requires consideration of several additional contributing factors [11,18,67,69,81,84–90], including:

- Disturbances in gut microbiota
- Regulatory dysfunction in connective tissue
- Overload of the lymphatic system
- Autonomic nervous system dysregulation
- Allergies, pseudoallergies, and food intolerances
- Psychosomatic stress and emotional burdens
- Hormonal imbalances
- Chronic low-grade inflammation
- Deficiencies in trace elements, vitamins, and minerals
- Instability in blood glucose regulation
- Disruptions in acid-base and fluid balance

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2. Gut Health and Metabolic Regulation

There is a well-established connection between the intestinal environment, gut microbiota, and chronic conditions such as migraines. The concept of autointoxication—the internal reabsorption of fermentation and putrefaction by-products due to impaired digestion—can contribute to the development of migraine through inflammatory or autonomic mechanisms.[90,91] In addition, reactive hypoglycemia, a frequently overlooked drop in blood glucose, may provoke migraine-like symptoms. Targeted diagnostics, including oral glucose tolerance testing and microbiological stool analysis, are essential in identifying underlying contributors.[11,18,81,90,92]

3. Targeted Use of Vital Nutrients

Iron or magnesium deficiencies are frequently associated with migraines. Magnesium, in particular, has a muscle-relaxing effect, acting on both skeletal and vascular smooth muscle. In therapeutic doses, omega-3 fatty acids and B vitamins have also proven beneficial. However, effective treatment depends on comprehensive diagnostics and precise supplementation, ideally guided by healthcare professionals trained in regulatory or orthomolecular medicine.[85,87–89]

4. Chronic Inflammation

Persistent inflammatory for example, in the tonsils, paranasal sinuses, or dental regions—can contribute to or maintain migraine episodes via autonomic reflexes, local muscular tension, or mechanical blockages. In such cases, neural therapy may play a key role in focus regulation and autonomic relief, supporting broader therapeutic goals.[9,11,18,41–46,49,50,54,66]

5. Allergies and Pseudoallergies

In addition to classical IgE-mediated immediate-type allergies, delayed immune responses (Type III/IV) and pseudoallergies—mainly related to foods—are gaining clinical importance in migraine pathophysiology. Laboratory blood tests performed in specialized diagnostic centers can provide valuable insights. Elimination phases and targeted exclusion diets help clarify causal relationships and improve symptom control.[18,62,86,93]

6. Psychological Stress

Migraine may also be a manifestation of emotional tension or internal conflicts—whether due to chronic daily stress, perfectionism, or unresolved family dynamics. In many cases, the migraine attack serves as a "warning signal" from the body, prompting self-regulation. Psychosomatic support—including talk therapy, mindfulness-based techniques, and body-oriented methods—can be beneficial in managing these psychosocial factors.[3,11,18,22,64,65,6894,95]

THERAPY – INDIVIDUALIZED AND CAUSALLY ORIENTED

Neural therapy deserves special attention in this context: it is easily applicable, cost-effective, minimally invasive, and well-suited for both acute intervention and long-term regulation. It can seamlessly integrate into routine medical practice.[18,22,23,27–30]

Connective Tissue, Latent Acidosis, and Heavy Metal Load – An Underestimated Link in Migraine

A. Connective Tissue as a Regulatory Organ

Connective tissue serves far more than a passive structural function. It is a key component of the Pischinger extracellular matrix system, integrating nerve endings, blood and lymphatic vessels, immune cells, and the extracellular matrix. As a regulatory interface, it reacts sensitively to physical, toxic, and emotional stressors.[27–30,96–100]

In the presence of chronic inflammation or a toxic burden, connective tissue may lose its buffering, filtering, and regulatory functions. The result is impaired microcirculation, disrupted cellular communication, and autonomic irritability, all of which can contribute to the onset and maintenance of migraine.[96–103]

B. Latent Acidosis as a Functional Disturbance

Latent acidosis refers to chronic, subclinical tissue acidification, often caused by dietary imbalances (e.g., excessive animal protein, sugar, and caffeine), stress, lack of physical activity, or reduced renal buffering capacity.[11,18,35,36,63,69] This condition may not be detectable in blood tests, as the body tightly regulates serum pH—often at the expense of buffer reserves (e.g., calcium from bones, bicarbonate from cells).[31,35,36]

Tissue-level consequences include:

- Increased pain sensitivity
- Muscular tension
- Impaired cellular metabolism
- Hyperexcitability of autonomic centers

These factors promote the development of tension headaches and migraine. Therapeutic approaches include alkaline therapies, nutritional correction, micronutrient balancing, and, most notably, regulatory techniques such as neural therapy, aimed at restoring proper signal conduction.[11,18,33,35,36]

C. Heavy Metal Load – An Underestimated Trigger for Chronic Migraine

Chronic exposure to heavy metals such as mercury, lead, aluminum, or cadmium can profoundly affect the nervous system, immune response, and cellular environment.[18,33,35,36]

Pathophysiological mechanisms include:

- Neurotoxicity: Heavy metals impair synaptic transmission, mitochondrial function, and signal processing in the central and autonomic nervous systems.
- Mitochondrial dysfunction: Energy-deprived neurons are more susceptible to trigger stimuli—leading to increased migraine susceptibility.
- Pro-inflammatory effects: Heavy metals chronically activate the immune system and promote "silent" inflammation.
- Tissue deposition: Mercury and similar heavy metals, in particular, accumulates preferentially in connective and nervous tissue potentially leading to cellular damage, dysfunction, and chronic toxicity.

Patients with non-specific symptoms, therapy resistance, or multiple intolerances should be evaluated for potential heavy metal burden. Diagnostic tools include chelation provocation tests with urine analysis, whole-blood mineral assessments, and clinical history regarding dental amalgams, vaccinations, and environmental or occupational exposures.[27,31,35,36]

THERAPEUTIC APPROACH: INTEGRATIVE DETOXIFICATION AND REGULATION

A comprehensive treatment strategy for connective tissue-related migraine should integrate multiple therapeutic components to address both the symptoms and underlying regulatory dysfunctions:

- **Neural Therapy:** Segmental and interference field injections with local anesthetics help relieve autonomic nervous system overload and enhance microcirculation.[54,94,95]
- **Alkalizing Infusions or Oral Base Therapy** supports the restoration of tissue pH and the extracellular matrix environment.[26,27,44,48]
- Micronutrient Supplementation: Particularly with magnesium, zinc, selenium, and vitamin C, which support mitochondrial, immune, and neuromuscular function.
- Heavy Metal Detoxification: The use of chelating agents (e.g., DMPS, DMSA) under medical supervision to reduce the toxic metal burden.[18,35,36]
- Gut Rehabilitation and Liver Support: Since detoxification pathways primarily operate through the gut, liver, and kidneys, targeted organ support is essential.[35,63,91,92,104]
- **Nutritional Medicine:** Emphasis on an anti-inflammatory, micronutrient-rich diet (e.g., FX Mayr principles or alkaline-oriented nutrition).[62,63]

Active Trigger Points in Headache and Migraine – The Role of Neural Therapy

In tension-type headaches and migraines, specific skeletal muscles with active myofascial trigger points play a crucial role in symptom development. Particularly in chronic cases, these points often represent the primary cause or a major exacerbating factor.[9,11,18,37,38,43,45,93,105–108]

Commonly affected muscles include:

- Trapezius (descending part)
- Sternocleidomastoid
- Temporalis
- Occipitalis
- Splenius capitis and services
- Masseter
- Suboccipital muscles (deep neck flexors and extensors)

Active trigger points in these muscles may cause dull, pressing, or pulsating pain, frequently radiating to the forehead, temples, occiput, orbital region. Tension-type headaches are often the primary source of pain. In migraines, they may lower the pain threshold and facilitate the onset of attacks.[11,18,23,24,32,37,38,42–44, 49,50]

Neural Therapy as a Targeted Intervention

Neural therapy provides a practical and precise approach for addressing these myofascial components. Injections of local anesthetics such as procaine directly into affected trigger points can:

- Interrupt maladaptive autonomic reflex arcs
- Reduce pathological muscle tension
- Stabilize chronic pain patterns

This approach relieves symptoms and, when combined with manual diagnostic assessment, can serve as a causal therapy.[105–107]

Holistic Regulation Beyond Symptom Control

What sets neural therapy apart is its holistic regulatory perspective. Beyond addressing myofascial dysfunction, it also considers:

- Interference fields
- Autonomic dysregulation
- Structural imbalances

This integrative orientation presents a clear advantage over conventional pharmacological therapies, making neural therapy a compelling option for the sustainable treatment of migraine and chronic headache disorders.[52,66,67,69,83]

The Role of the Autonomic Nervous System in Migraine – Focus on Sympathetic Tone and Regulation

Migraine is far more than an episodic headache disorder; it represents a profound neurovascular and autonomic dysregulation. A growing body of research highlights the critical role of chronic sympathetic overactivity in the pathophysiology of migraine. Excessive sympathetic tone contributes to persistent vasoconstriction, microcirculatory disturbances, and neurogenic inflammation, resulting in heightened activation of the trigeminovascular system and the

pathological amplification of pain processing.[9, 11, 30, 33, 48, 54, 55, 57, 81, 84, 96–100]

This dysregulation of the autonomic nervous system (ANS) increases pain sensitivity and disrupts core homeostatic functions such as sleep, digestion, vascular tone, and immune response. As a result, a self-perpetuating cycle emerges, marked by chronic pain, systemic inflammation, and impaired perfusion. Therapeutic strategies aimed at restoring autonomic balance—particularly by downregulating excessive sympathetic activity—are crucial in breaking this vicious cycle.[9,11,30,32,33,40,42,44,54,55,57,81,84,96–100]

Interventions such as neural therapy, which modulate the ANS, have demonstrated promising clinical outcomes. Injections into interference fields and segmental reflex zones can restore autonomic homeostasis, improve blood flow, and reduce inflammatory activity. These effects are frequently associated with a significant reduction in the frequency and intensity of migraine attacks. Thus, the reorganization of autonomic function may represent a key therapeutic mechanism in effectively treating chronic migraine.[29,30,40,52,54]

Critique of Conventional Approaches – and the Added Value of Neural Therapy

Conventional medicine often focuses on the symptomatic treatment of structurally mediated headaches, primarily relying on pharmacological agents such as analgesics. However, it frequently fails to incorporate functional diagnostics, particularly in the musculoskeletal system, leaving underlying causes unaddressed and impeding the potential for lasting recovery.

In contrast, neural therapy offers a causally oriented and individualized treatment modality. Through targeted injections, it regulates autonomic reflex pathways, relieves interference fields, and disrupts chronic pain circuits. As an integral component of regulatory medicine, it provides a comprehensive and system-oriented solution, especially in chronic and multifactorial pain conditions, significantly improving patients' quality of life.[18, 30, 52, 54, 66, 67, 53, 94, 105, 109–112]

In clinical practice, neural therapy has also proven efficient, safe, and cost-effective, making it a valuable tool in modern, patient-centered medical care.

A HOLISTIC NEURAL THERAPY APPROACH IN MIGRAINE PATIENTS

Objective of the Study

The objective of this retrospective study is to present the results of a comprehensive clinical evaluation of 464 patients with a confirmed diagnosis of migraine, who were treated and followed up at our clinic between 2019 and 2024. The analysis focuses on both the therapeutic responses to neural therapy and the identification and interpretation of potential underlying causes of migraine.

Particular attention was given to multifactorial contributors, including hormonal dysregulation, intestinal dysbiosis, structural dysfunctions (e.g., cervical spine, temporomandibular joint), and autonomic nervous system imbalance. The aim was to deepen the understanding of migraine pathophysiology and to evaluate the clinical impact of a holistic, regulation-based therapeutic approach, especially in chronic and recurrent cases.

Materials and Methods

In this retrospective analysis, patient history forms were reviewed for individuals whose pain documentation included the keyword “migraine.”

The following parameters were evaluated:

- Hormonal dysfunction
- Disturbed intestinal microbiota (dysbiosis)
- History of dental interventions
- Temporomandibular dysfunction (CMD)
- Daily fluid intake
- Functional limitations of the upper cervical spine (C2 blockade)
- Sex and age

A total of 464 patient records were included in the evaluation, consisting of 384 women and 80 men.

Commentary and Interpretation

- Most patients had a normal body weight, indicating no significant correlation between migraine and overweight or obesity.

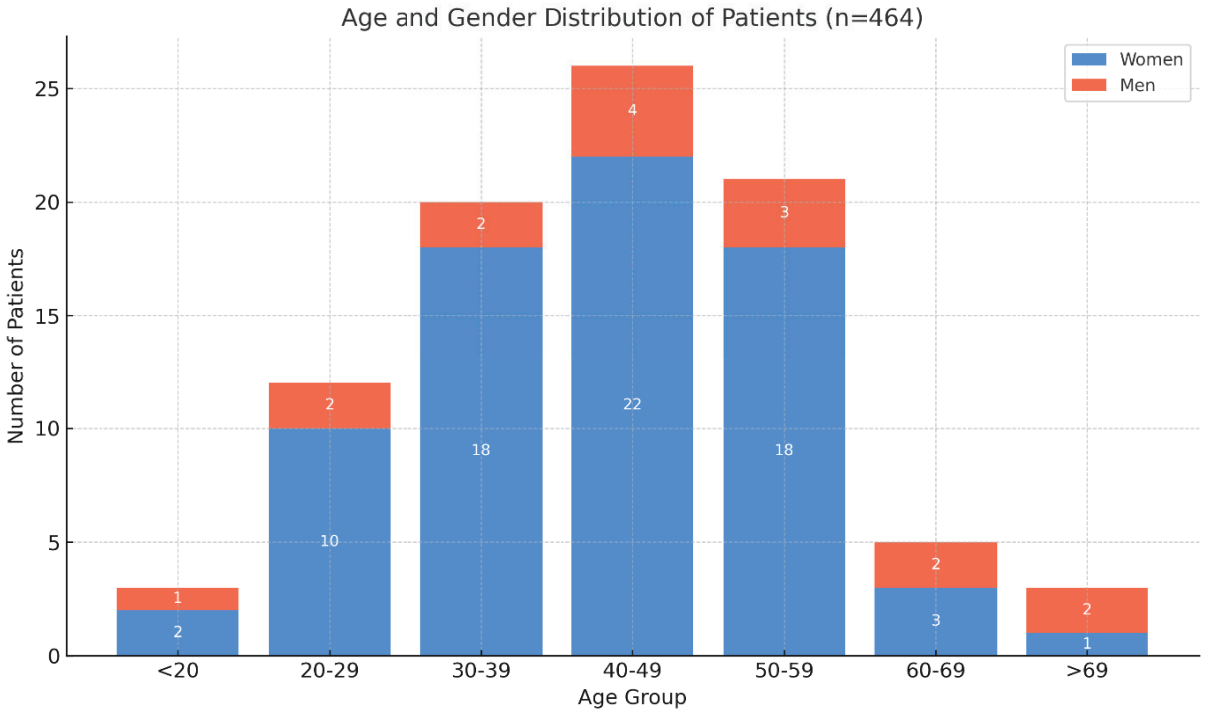

Figure 1: *Distribution of patients by age group and gender in a retrospective analysis of 464 migraine cases*

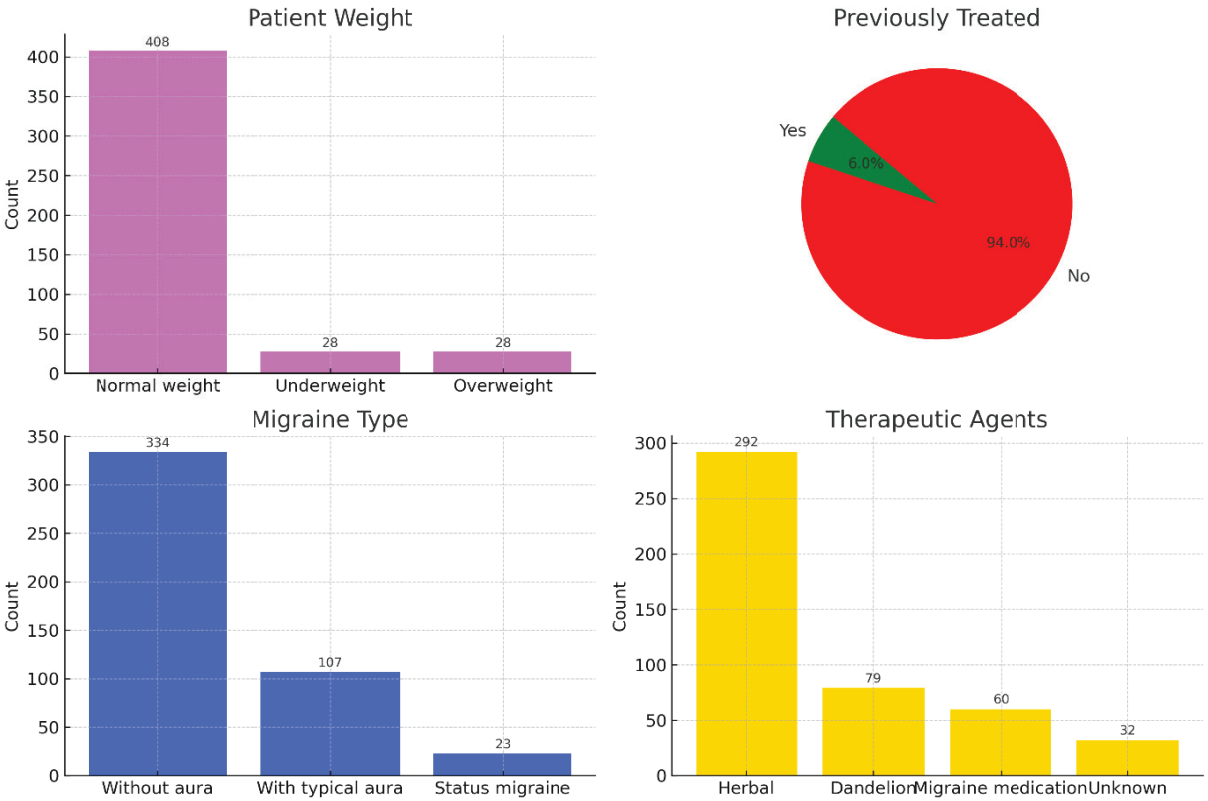

Figure 2: *Overview of patient characteristics, including weight distribution, prior treatment status, migraine types, and types of therapeutic agents used*

- Most individuals received neural therapy for the first time, allowing for a clear assessment of the method's immediate effects.
- Migraine without aura was by far the most common type, highlighting this subgroup as particularly well-suited for regulatory therapies such as neural therapy, as the presence of aura typically indicates greater neurological complexity.
- Most patients were treated with herbal or regulatory remedies, underlining the consistent application of gentle, low-risk therapeutic modalities.
- Overall Evaluation of Effectiveness and Treatment Quality
- A high proportion of previously untreated patients (94%)enables a more accurate evaluation of therapeutic effectiveness.
- Marked clinical efficacy was observed in uncomplicated migraine without aura (72%).
- The frequent use of plant-based and regulatory treatments reflects a strong preference for gentle medicine.
- A few heavily pretreated cases and a moderate BMI profile were associated with favorable clinical outcomes.

Statistical Effectiveness

The data support neural therapy as an effective, safe, and holistically oriented treatment option.Withover 90% of patients showing clinical improvement (based on prior evaluations) and predominantly short treatment durations, neural therapy proved highly effective in this patient cohort.

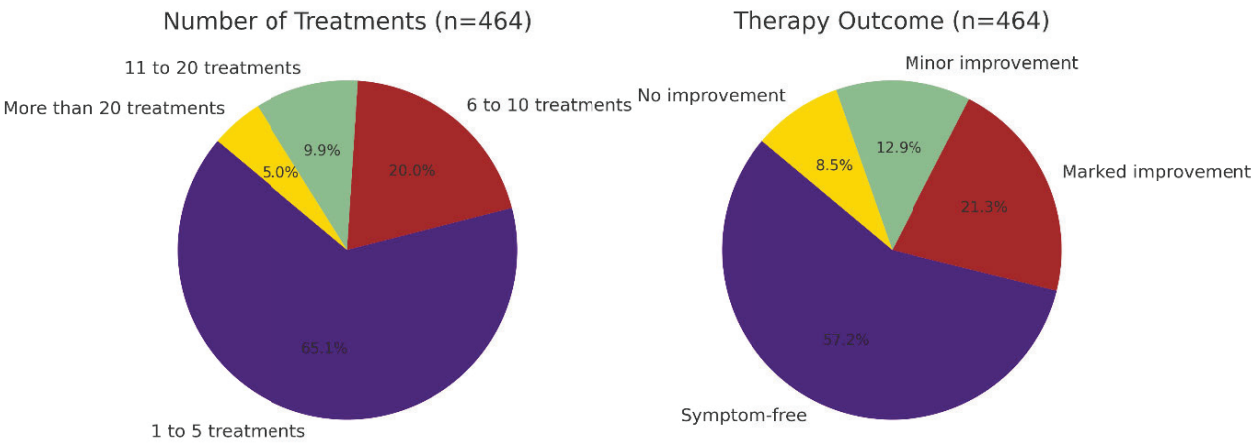

Figure 3: *Distribution of the number of neural therapy sessions and corresponding treatment outcomes among 464 migraine patients*

Graph on the left: Number of treatments

Number of Treatments	Estimated Count	Percentage
1 to 5 treatments	302	65.10%
6 to 10 treatments	93	20.00%
11 to 20 treatments	46	9.90%
More than 20 treatments	23	5.00%
Total	**464**	**100%**

Most patients (65%) were successfully treated with just 1-5 sessions. Only 5% required more than 20 sessions, which speaks to the method's high efficiency.

Graphic right: Therapy result

Therapy Outcome	Estimated Count	Percentage
Symptom-free	278	59.90%
Marked improvement	93	20.00%
Minor improvement	56	12.10%
No improvement	37	8.00%
Total	**464**	**100%**

Statistical Evaluation of Effectiveness

- Overall improvement rate (complete or partial): 427 out of 464 patients → 91.9%
- Non-response rate: 37 out of 464 patients → 8.0%
- Interpretation:
- Neural therapy demonstrated high clinical efficacy, with nearly 60% of patients achieving complete symptom relief.
- Only 8% of patients did not respond to the therapy.
- The low number of treatment sessions required in most cases (1–5 sessions) highlights the method's economical and efficient nature.
- The combination of short treatment duration and high therapeutic impact strongly supports neural therapy's value as a holistic and regulatory intervention.

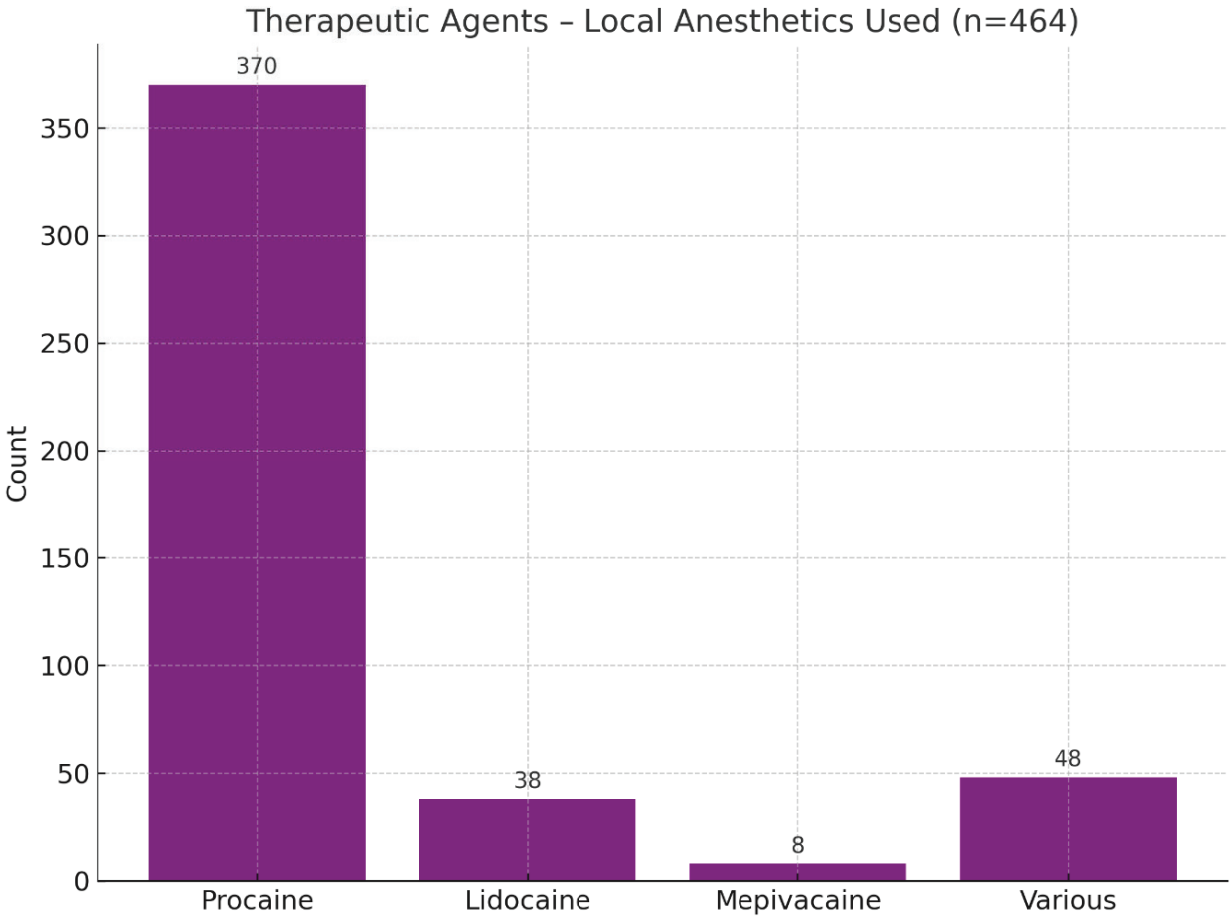

Figure 4: *This bar chart illustrates the distribution of local anesthetics used in neural therapy among 464 migraine patients. Procaine was by far the most frequently administered agent, followed by lidocaine, various combinations, and mepivacaine.*

RESULTS

The most frequently observed associated findings among the patients were:

- Disturbed gut microbiota (dysbiosis): 89.2% (n = 414)
- History of dental interventions: 75.0% (n = 348)
- Temporomandibular dysfunction (CMD): 42.8% (n = 199)
- Hormonal dysregulation: 71.4% (n = 331), particularly in association with menstrual cycles and gut dysbiosis in female patients
- C2 vertebral blockade (upper cervical spine): 42.8% (n = 199), with potential irritation of the nervusoccipitalis central and indirect involvement of the trigeminal system through dural connections

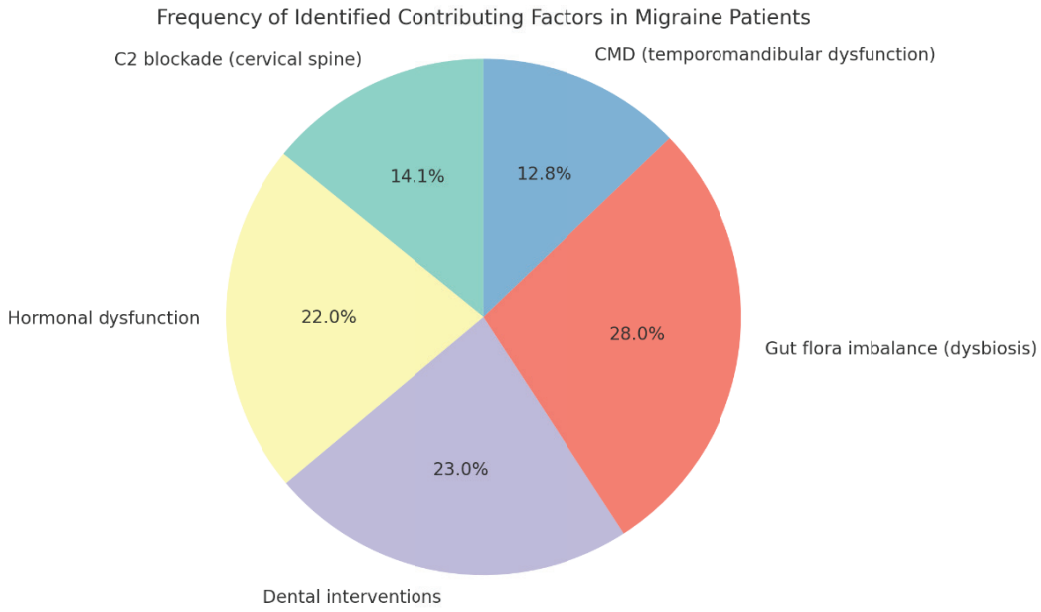

Figure 5: *Common contributing factors identified in migraine patients, including dysbiosis, dental issues, and hormonal imbalance*

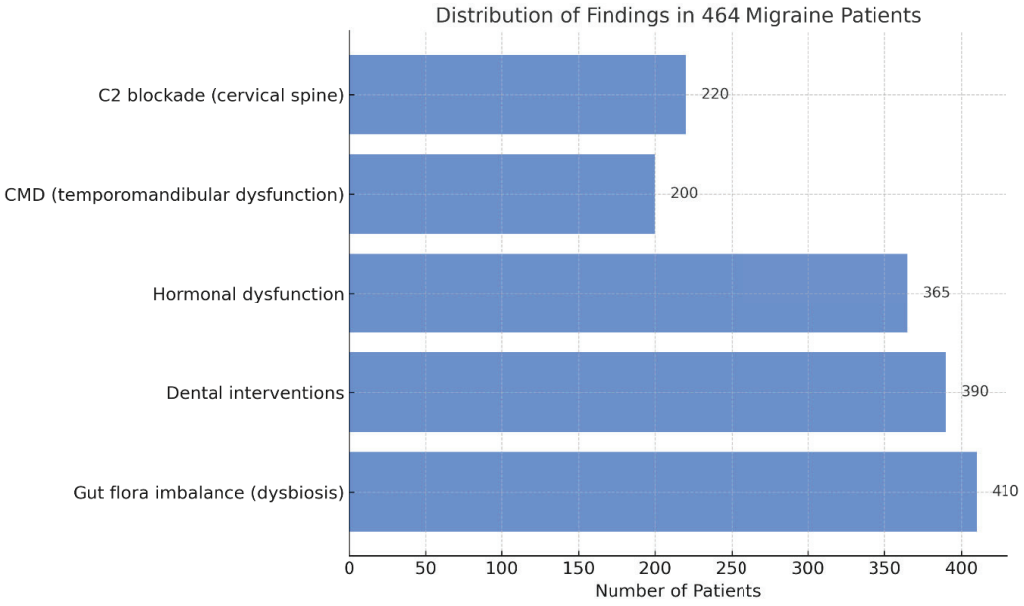

Figure 6: *Distribution of frequently identified clinical findings in 464 migraine patients.*

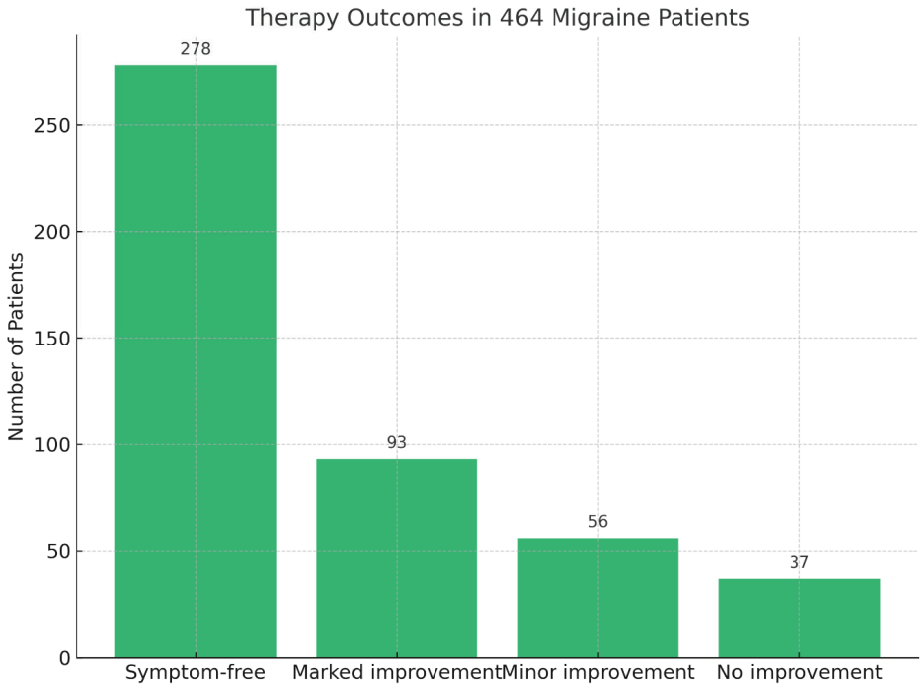

Figure 7: *Therapy outcomes in 464 migraine patients.*
The high rate of symptom relief highlights the clinical effectiveness of neural therapy.

Statistical Assessment of Effectiveness

- The overall clinical effectiveness (complete remission + partial improvement) exceeded 90%, indicating a very high therapeutic efficacy.
- The proportion of patients achieving complete symptom resolution (59.9%) suggests that the method is not only palliative but frequently exerts a causally regulatory effect.
- The non-responder group (8%) was relatively small and may be associated with therapy-resistant forms or comorbid factors such as hormonal imbalances, severe dysbiosis, or psychological stress.
- Summary (Key Points)
- High success rate with nearly 60% complete remission
- Over 90% of patients experienced some degree of improvement
- Only 8% showed no response
- The method can be effective and clinically recommendable based on this sample.

DISCUSSION

The findings of this study demonstrate that migraine is a multifactorial condition, with structural, hormonal, neurovegetative, and metabolic factors playing key roles in its pathogenesis.

While conventional medicine primarily focuses on symptomatic relief, neural therapy aims to restore regulatory balance at the source of dysfunction. Through targeted injections with local anesthetics such as procaine or lidocaine, disrupted control loops can be reactivated, autonomic imbalances corrected, and underlying causes of pain effectively addressed.

A genuinely holistic diagnostic approach should always consider the following systems:

- The musculoskeletal system (especially the upper cervical spine)
- The dentoalveolar and temporomandibular system
- The intestinal microbiota
- Hormonal regulation
- Toxic load and latent acidosis
- Hydration status

The results presented here compellingly support the clinical relevance of neural therapy as an integral component of a holistic, regulation-oriented treatment approach for migraine. With an overall improvement rate of over 90% and complete symptom remission in nearly 60% of patients, this method offers symptomatic relief and deep regulatory intervention

into pathophysiological mechanisms. The modulatory influence on the autonomic nervous system emerges as a key mechanism often overlooked in conventional treatment strategies.

Patients with functional comorbidities such as intestinal dysbiosis, hormonal imbalance, temporomandibular dysfunction (CMD), or C2 vertebral blockades benefit particularly from targeted neural therapeutic interventions. These injections do not merely address the pain itself but intervene causally in dysfunctional regulatory circuits. The therapeutic outcome is further enhanced when neural therapy is combined with other regulatory measures such as nutritional optimization, micronutrient supplementation, manual therapy, and psychosomatic support.[11,71–74,86]

Another important result of this study is the economic efficiency of neural therapy. Approximately two-thirds of patients require no more than five sessions to achieve significant clinical improvement. This makes neural therapy particularly attractive for primary care and general medical practice, especially considering the often-high cost and adverse effects of pharmacological treatments. Its ease of application and precise controllability also support its integration into diverse healthcare models.[67]

Despite these promising results, further controlled multicenter studies are needed to validate the effectiveness and sustainability of neural therapy in migraine management. Future research should emphasize standardized documentation, long-term follow-up, and the inclusion of psychosocial and functional parameters. Incorporating individual stress profiles may significantly improve therapeutic outcomes, especially in the context of chronic pain syndromes.[66,67]

Recent randomized controlled trials have investigated alternative non-invasive approaches for acute migraine treatment, including the intranasal application of local anesthetics. In the study by Avcu et al. (2016), the efficacy of intranasal 10% lidocaine was evaluated in comparison with normal saline in 162 emergency patients. Although the immediate pain reduction at 15 and 30 minutes was not significantly superior to placebo, a higher proportion of patients in the lidocaine group reported being pain-free after 24 to 72 hours. This delayed therapeutic effect may indicate a mechanism beyond rapid peripheral action (113).

The study's conceptual foundation lies in the modulation of the sphenopalatine ganglion (SPG), which is believed to mediate cranial parasympathetic outflow and contribute to neurogenic inflammation and pain. Blocking the SPG early in the course of migraine, before central sensitization becomes established, may be crucial. This supports the broader therapeutic concept that early autonomic modulation—whether by intranasal lidocaine or neural therapy—might offer meaningful benefits in selected patients (113).

Despite the lack of significant short-term results, the study demonstrates the safety of intranasal lidocaine and highlights the relevance of treatment timing. It also aligns with previous literature suggesting variable outcomes depending on administration protocols. These findings contribute to the growing body of research emphasizing the role of parasympathetic interventions in migraine, which could complement regulatory and neural therapeutic strategies (113).

CONCLUSION

Neural therapy emerges from this retrospective analysis as a highly effective, low-risk, and economically viable treatment modality that can be successfully applied both in acute migraine management and for long-term stabilization of complex symptomatology.

Neural therapy's ability to regulate autonomic imbalances, modulate myofascial trigger points, and restore functional coherence across multiple physiological systems makes it a valuable and versatile tool in integrative pain management. Nearly 60% of patients achieved complete symptom remission, and the majority required only a few sessions, underscoring its efficiency, tolerability, and therapeutic potential.

In clinical practice, neural therapy offers a causally oriented alternative to conventional pharmacological strategies, which often focus solely on symptom suppression. Particularly for patients with multifactorial migraine etiologies—including hormonal dysregulation, intestinal dysbiosis, temporomandibular dysfunction, and upper cervical impairments—neural therapy provides a targeted, mechanism-based intervention with holistic benefits.

Given these compelling findings, neural therapy should be considered not only as a complementary measure but also as a primary therapeutic option for selected migraine patients, especially those with functional, regulatory, or autonomic involvement.

Prospective multicenter studies with standardized protocols, long-term follow-up, and the integration of psychosocial and lifestyle-related variables are strongly

recommended to further substantiate these promising results and enhance access to this approach. Such studies could significantly contribute to the scientific validation and broader implementation of neural therapy in routine care.

Ultimately, this study supports the view that migraine is not an unchangeable or incurable condition but rather a treatable disorder when addressed through a comprehensive, individualized, and cause-oriented framework. As part of such a paradigm, Neural therapy holds great promise in reshaping how we understand and manage migraines—not just as a neurological disease but as a systemic dysregulation requiring systemic solutions.

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- We believe that both publications, once released, will make a substantial contribution to the broader international recognition and understanding of neural therapy.

DECLARATION OF CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest related to this publication and no competing interests to disclose.

The authors confirm that no competing financial interests are associated with this work.

No human or animal studies were conducted by the authors for this contribution. The studies mentioned in this publication adhere to the ethical guidelines specified in their respective sources.

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Authors' Contributions

The contributions of H. Nazlikul and F.G. Ural Nazlikul were central throughout the entire article and played a decisive role in the conceptual organization and content development.

Both authors contributed their extensive experience in physiotherapy, manual medicine, neural therapy, and pain management, significantly advancing the understanding of neural therapy's mechanisms of action in the context of migraine.

As academics with specialization in pain physiology and integrative pain management, they provided in-depth insights into the pathophysiological role of increased sympathetic tone, which is a key factor in many migraine patients.

In addition, their solid expertise in the diagnosis and treatment of mechanical and functional dysfunctions—particularly through the lens of manual medicine—was integrated into the analysis of clinical correlations. Their comprehensive perspective greatly enriched the article by addressing both neurovegetative and structural dimensions of migraine within a holistic, regulation-based therapeutic framework.

H. Nazlikul, F.G. Ural Nazlikul, N. Özkan and TijenAcarkan played a central role in the development of this case series by conducting a comprehensive retrospective analysis of clinical data from migraine patients treated with neural therapy at the Naturel Nazlikul Clinic between 2019 and 2024. Their meticulous work in patient selection, documentation review, and data interpretation laid the foundation for the clinical relevance of this article.

Y. Tamam supported the project by compiling and critically evaluating an extensive body of literature on non-vascular and multifactorial mechanisms of migraine, with a particular focus on the role of muscular, ligamentous, and myofascial dysfunctions as both initiating and perpetuating factors in chronic migraine syndromes.

As a board-certified neurologist and university lecturer, he not only contributed his expertise in the classical diagnosis and treatment of migraine, but also offered valuable insight into the broader, integrative neural therapy approach explored in this study. His academic perspective significantly enriched the interpretation of clinical findings and greatly supported the structured evaluation of relevant literature within the framework of this work.

M.D. Bilgin contributed his expertise in neuroanatomy, segmental innervation, and the regulation of the autonomic nervous system in the context of migraine. He played a key role in the conceptual structuring, scientific writing, and critical revision of the manuscript, ensuring both clinical accuracy and scientific rigor throughout the article's development.

Notably, his contribution enriched the manuscript by introducing a biophysical perspective on migraine, helping to deepen the understanding of the condition in relation to autonomic dysfunction and neurogenic inflammation. His interdisciplinary insights added a valuable dimension to the overall scientific depth of the work.

Ethical Approval

Not applicable.

Data Availability Statement

Not applicable.

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