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The Role of Plastic Surgery in Management of Migraine Headaches: A Literature Review

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ABSTRACT

Introduction: Migraine headache is a neurovascular disorder characterized by primary chronic headache pain, with attacks lasting between 4 to 72 hours. Plastic surgery interventions such as targeted nerve decompression and Botox injections can provide better results.

Methods: We created this literature review by compiling and analysing data and information from publicly accessible scientific journals available on the web.

Results and Discussions: Many studies show that surgical treatment for chronic migraines can produce substantial results. However, such surgery is only effective for those who have not benefited from other treatments. Botox injections can block NO and CGRP activation in the central nervous system, whereas nerve decompression can lessen the activity of pain-signaling nerves associated with chronic migraine headaches. Overall, 68.3% to 93.3% of patients reported satisfactory results. Complete elimination of migraine headaches was achieved in 28.3% to 59% of cases, while significant improvement was observed in 26.5% to 60% of patients.

Conclusion: Botulinum injections for migraine headache treatment are more effective when combined with nerve decompression procedures. Giving preoperative Botox injections can reduce NO activation in the CNS, thereby limiting the activity of decompressed nerves and alleviating migraine headache symptoms.

KEYWORD: Migraine headache, surgical treatment, abortive and prophylactic treatment

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INTRODUCTION

Migraine headache, a neurovascular disorder, presents as chronic headache pain lasting 4 to 72 hours. The pain is typically throbbing, unilateral, moderate to severe, and worsens with physical activity, often accompanied by nausea, vomiting, photophobia, and phonophobia.[1] Migraine headache affects 12% of the population, with 17% of women and 6% of men experiencing it annually.[2] About 70% of sufferers experience migraine headaches without aura, a tendency that increases with age. Most migraine headaches (90%) occur before age 40, peaking between 35 and 45 years.[3,4]

The International Classification of Headache Disorders (ICHD-3) divides migraine headaches into six categories, including those with and without aura. Migraine headache without aura involves recurrent, throbbing headaches, while migraine headache with aura includes neurological symptoms like sensory and visual disturbances. Chronic migraine headaches occur on 15 or more days per month for over 3 months. Other categories include migraine complications, probable migraines, and episodic syndromes linked to migraine, like GI disorders and paroxysmal vertigo.[5]

The World Health Organization ranks migraine headache as the sixth leading cause of disability worldwide.3 Although not life-threatening, migraine headaches significantly impair quality of life, affecting work, school, home, and social interactions.[3,4] In Indonesia, 1.3 million people experience disability due to migraine headaches.[4] Current treatments, including pharmacological, non-pharmacological, and surgical interventions, have only achieved partial success, necessitating comprehensive management strategies.[5]

Recent studies identify extracranial nerve compression as a migraine headache trigger, with surgical decompression showing symptom improvement. Common trigger sites include the frontal, temporal, and occipital regions, where nerve compression causes pain.[6] Surgery improves symptoms

in 70%-95% of patients, often resolving migraine headaches entirely.[7] This surgical approach, aimed at reducing nerve compression, benefits those with severe migraine headaches.[8] Extracranial nerve decompression has shown promise in improving migraine headache outcomes. Botulinum toxin (Botox) is also effective in preventing chronic migraine headaches, serving as a diagnostic tool to identify trigger points by reducing nerve pressure.[2,6] Botox involves 31 subcutaneous injections in the head and neck every 3 months.[9]

Guyuron and colleagues[9] found significant improvement in 84% of patients following nerve decompression therapy, with total symptom resolution in over 95% of patients who also received preoperative Botox. A research by Dirnberger and Becker supports these findings, with 68% of patients reporting improvement after surgery.[10] Janis an colleagues reported significant improvement in 79% of patients undergoing surgery guided by Botox.[11]

METHODS

We conducted this literature review by analyzing data from publicly accessible scientific journals, primarily using Google Scholar and PubMed. We focused on keywords such as migraine, surgical treatment of migraine, and abortive and prophylactic treatment of migraine. We selected relevant open-access journals with full text available in English or Indonesian, ensuring they addressed our research questions.

PATHOPHYSIOLOGY OF MIGRAINE HEADACHES

Migraine aura likely stems from cortical spreading depression (CSD), where a wave of depolarization spreads through the brain's cortical layers. This phenomenon, triggered by elevated extracellular K+ and N-methyl-D-aspartic acid (NMDA) receptor stimulation, causes visual and sensory disturbances associated with aura.[12]

CSD also triggers neurogenic inflammation by activating trigeminal nociceptors in the meninges. This leads to vasodilation of the middle meningeal artery, plasma protein leakage, and disruption of the blood-brain barrier via matrix metalloproteinase-9 (MMP-9). The result is plasma protein extravasation, releasing mediators like glutamate and ATP, which activate nociceptors, causing inflammation and prolonged cerebrovascular dysfunction, leading to migraine symptoms with aura.[4,12]

CSD activates both peripheral and central trigeminal pathways. Studies show increased c-Fos expression in the trigeminal caudal nucleus (TNC) due to CSD, which is linked to trigeminal pathway activation in the thalamus and somatosensory cortex. These neurons project to areas like the primary and secondary somatosensory cortex, primary visual cortex, and primary auditory cortex.[12]

Peripheral trigeminal activation occurs when neurons innervating the meninges and blood vessels are stimulated, leading to localized headache. Additionally, parasympathetic neurons in the superior salivatory nucleus may induce inflammation in the meninges, releasing pro-inflammatory mediators.[12]

Genetic and environmental factors also contribute to migraine headaches. Patients with a family history are three times more likely to experience migraine headaches. Multiple genes, interacting with environmental triggers like stress (80%), hormonal changes (65%), and sensory stimuli (40%), play roles in onset of migraine headaches.[2]

CONSERVATIVE TREATMENT OF MIGRAINE HEADACHES

Migraine management encompasses acute and preventive treatments using pharmacological and non-pharmacological approaches. The primary goals include preventing disease progression, reducing attack frequency and severity, decreasing disability, and enhancing life quality.[3]

Abortive management

Non-Steroid Anti-Inflammatory Drugs **(**NSAIDs) are the first-line treatment for mild to severe migraines. They work by inhibiting prostaglandin-E2 (PGE2) synthesis through cyclooxygenase-1 (COX-1) and cyclooxygenase-1 (COX-2) inhibition, reducing inflammation triggered by vasoactive peptides. Common side effects include gastrointestinal issues, hypersensitivity reactions, and renal or liver disorders.[13,14] Patients with hypersensitivity, renal failure, or gastrointestinal bleeding should avoid NSAIDs. Dosages include acetylsalicylic acid (up to 1000 mg), ibuprofen (200-800 mg), and others. To minimize side effects, clinicians normally limit NSAID use to 15 days per month and combination analgesics to fewer than 10 days monthly. Anti-emetics like domperidone, metoclopramide, and prochlorperazine can be used alongside. Combining aspirin, acetaminophen, and caffeine proves more effective than separate administration.[14,15]

Triptans are serotonin receptor agonists effective when NSAIDs and acetaminophen fail.[13,14] They act by vasoconstricting intracranial vessels and inhibiting nociceptive neurotransmitter release via 5-hydroxytryptamine receptor 1B/1D (5-HT1B/1D) receptors. Triptans reduce plasma vasodilation and inflammation.[14,15] Side effects include chest pressure, limb heaviness, myalgia, and less commonly, dizziness and cognitive issues. Contraindications include high blood pressure, ischemic heart disease, and recent ergotamine use.[1,13] In term of daseges for treating migraines, Sumatriptan can be taken orally at 25-100 mg, with the option to repeat the dose every 2 hours as needed, up to a maximum of 200 mg per day. Alternatively, it can be administered intranasally at 5-20 mg, with a maximum daily dose of 40 mg, or subcutaneously at 4-6 mg, with a maximum of 12 mg per day. Almotriptan is available at 6.25–12.5 mg orally, with doses repeated every 2 hours if necessary, up to 25 mg per day. Eletriptan is taken orally at 20–40 mg, with repetitions every 2 hours if required, up to 80 mg per day. Frovatriptan is administered orally at 2.5 mg, with a maximum of 7.5 mg per day. Naratriptan, taken orally

at 1–2.5 mg, allows for a maximum daily dose of 5 mg, and Rizatriptan is given at 5–10 mg orally, with up to 30 mg per day.

Prophylactic management

Clinicians often use prophylactic therapy to prevent migraine recurrences in patients who experience more than two headaches per month, with each lasting over 24 hours, causing significant disability, or when abortive therapy fails. This approach is also used for menstrual migraines, migraines with aura, hemiplegic migraines, and migraine-related infarction.[1]

Medications for prophylaxis include antihypertensives, antidepressants, anticonvulsants, and monoclonal antibodies. Beta-blockers, such as propranolol (80-240 mg/day), timolol (10-15 mg twice daily), and metoprolol (50-200 mg/day), are particularly effective by inhibiting norepinephrine release and reducing thalamic activity. Common side effects include fatigue, sleep disturbances, and nausea, with contraindications in conditions like congestive heart failure and asthma.[13-15]

Antiepileptics, especially topiramate and valproic acid, also prove effective. Topiramate (25-200 mg/day) works by blocking sodium and calcium channels, reducing glutamate activity, and enhancing γ-aminobutyric acid (GABA) inhibition. Side effects include nausea, dizziness, and weight loss. Valproic acid, although effective, is not recommended during pregnancy due to teratogenic risks and is less effective in adolescents.[1,13,14]

Non-pharmacological behavioral therapy

Non-pharmacological migraine treatments focus on psychological therapies and lifestyle changes. Behavioral therapy, including biofeedback and cognitive behavioral therapy (CBT), plays a key role. Biofeedback uses electronic devices to help patients monitor and control pain-related processes like muscle tension, blood pressure, and heart rate. CBT, on the other hand, focuses on stress management.[3]

Lifestyle modification

Lifestyle modifications for managing migraines can be summarized as SEEDS: sleep, exercise, eat, diary, and stress management. Ensuring quality sleep through good sleep hygiene and managing sleep disorders can reduce chronic migraines to episodic ones. Regular exercise, such as walking or cycling, 3-5 times a week for 30-50 minutes, is recommended. A balanced diet without specific restrictions, along with sufficient hydration, helps maintain an ideal body weight, while caffeine intake should be limited to less than 200 mg daily. Keeping a diary to track migraine frequency, triggers, and medication use aids in diagnosis and therapy adjustments. Finally, managing stress through techniques like CBT, mindfulness, and relaxation is crucial as stress and anxiety are known migraine triggers.[16]

ROLE OF INVASIVE/SURGICAL INTERVENTIONS

We are now familiar with plastic surgery, a branch of medicine aimed at correcting and restoring anatomical form, physical function as well as refining appearance. It addresses bodily defects caused by burns, lacerations, or illnesses through surgical intervention.[17] Facial reconstructive surgery, a common area of focus, involves various subunits such as the glabella, eyebrows, central and lateral forehead, and temple, with key surgical points include preserving motor function, particularly the temporalis branch of the facial nerve, and ensuring optimal scar concealment near hairlines and eyebrows while minimizing skin tension. Tension lines typically run horizontally in the forehead and curvilinear in the temple, with age-related changes making wrinkles more prominent.[18,19]

Facial plastic surgeons have long utilized Botulinum neurotoxin (BoNT) injection, popularized since 1989, for its ability to reduce muscle tension, improving the appearance of scars and treating pathological scarring, such as hypertrophic scars and keloids.[20] Later, Botulinum neurotoxin A (BoNT-A) slowly became one of the most used invasive interventions for chronic migraine. However, these injections require precise anatomical knowledge of target muscles and careful handling of the toxin.

Surgical decompression is increasingly popular for treating migraines, especially when pharmaceutical treatments fail. This approach targets peripheral sensory nerve branches associated with migraine trigger points, primarily in the frontal region. Surgical decompression typically associates with irritation of the supraorbital nerve (SON), the supratrochlear nerve (STN), and the terminal branches of the frontal nerve.[21] According to one study, 28 out of 50 patients experienced permanent pain reduction after one post-operative year and reported that they had benefited from the operation. Another study also demonstrated a reduction in headaches following 72% of surgeries, with a mean follow-up period of 28 months.[8]

BOTULINUM NEUROTOXIN IN MIGRAINE MANAGEMENT

Botulinum neurotoxin (BoNT), produced by Clostridium botulinum, is a polypeptide with a 100-kd heavy chain and a 50-kd light chain linked by a disulfide bond. It inhibits Synaptosomal-Associated Protein Receptor (SNARE) protein fusion at the neuromuscular junction, blocking acetylcholine release and causing temporary muscle paralysis. Of the seven BoNT types, only Botulinum neurotoxin A (BoNT-A) and Botulinum neurotoxin A (BoNT-B) are available commercially; BoNT-A cleaves SNAP-25, while BoNT-B targets synaptobrevin. Muscle function recovers as new axonal sprouts and neuromuscular junctions develop. In the U.S., three BoNT-A products are used: OnabotulinumtoxinA (OnaBT-A; Botox®), AbobotulinumtoxinA (AboBT-A; Dysport®), and IncobotulinumtoxinA (IncoBT-A; Xeomin®). Although precise conversion factors between these formulations are not

fully established, studies suggest 1 : 2 ratio of OnaBT-A to (AboBT-A, and a 1 : 2.5 ratio can lead to diffusion and different areas of effects. IncoBT-A shows similar efficacy and adverse effects to OnaBT-A when using a 1 : 1 ratio.[20]

OnabotulinumtoxinA (OnaBT-A) is frequently used for chronic migraine, and the protocol from Phase III Research Evaluating Migraine Prophylaxis Therapy (PREEMPT) describes it has 31 specific sites of injections in various muscles, including the frontalis, corrugator, and trapezius. Injection into specific muscles can contains up to 40 IU of OnaBT-A. This treatment targets peripheral nerve terminals, affecting neurotransmitter release by disrupting the SNARE complex and vesicle fusion. Vesicles containing neurotransmitters and neuropeptides undergo docking, priming, and fusion, processes mediated by SNARE proteins.[22] OnaBT-A primarily targets Synaptosomal-Associated Protein 25 kDa (SNAP-25), a SNARE protein crucial for vesicular fusion. After injection, the toxin binds to polysialogangliosides (PSG) such as GT1b and synaptic vesicle proteins. Additionally, it binds with higher affinity to synaptic vesicle protein 2 (SV2), a protein present on vesicles exposed during fusion. Once botulinum toxin A binds to its receptors, endocytosis takes in and inside the endosome, the light chain separates from the heavy chain and moves into the cytosol, where it specifically cleaves SNAP-25. This proteolytic action of the light chain disrupts the SNARE protein complex, blocking the fusion of synaptic vesicles with the inner membrane of the cell. Consequently, affected vesicles are unable to release their contents into the synaptic cleft or insert receptors and ion channels, such as the transient receptor potential cation channel subfamily V member 1 (TRPV1) and purinergic receptor 2 subclass X 3 (P2X3), into the neuronal membranes. This disruption is evident in how OnaBT-A impairs the trafficking of thermo-transient receptor potential (TRP) channels. The subsequent inhibitory effects vary depending on the target organ, including nociceptors, motor nerves, autonomic nerves in skeletal or smooth muscles, or glands. However, in clinical practice, the effects of OnaBT-A typically persist for about 3 to 4 months in motor nerves and 6 to 9 months in autonomic nerves.[23]

Migraine pain starts with the activation of nociceptive neurons in the duramater, releasing neuropeptides and neurotransmitters like calcitonin gene-related peptide (CGRP), pituitary adenylate cyclase activating polypeptide 38 (PACAP-38) along with nitric oxide (NO) triggering vasodilation. OnaBT-A reduces the release of these factors and affects both peripheral and central pathways. Preclinical studies showed that part of its mechanism in preventing migraines may involves decreasing CGRP release from peripheral nerve terminals in the meningeal and trigeminal nociceptors. Functional evidence showed that extracranial administration of OnaBT- A also affecting intracranial nerves by mechanism of inhibiting the responses of C-fibers, not Aδ-fibers, to stimulation of their intracranial meningeal receptive fields with the transient receptor potential cation channel subfamily V member 1 (TRPV1) and the transient receptor potential cation channel subfamily A member 1 (TRPA1) channel ligands, and thus proved the existence of intracranial-to-extracranial and extracranial-to-intracranial pathways. OnaBT-A injections work on both peripheral and central pathways to prevent migraine headaches by increasing the threshold for nociceptive activation, decreasing TRPV1-immunoreactive neurons, and lowering the activation of central neurons.[23]

Previously published studies confirmed the effectiveness of Botulinum neurotoxin A (BT-A) for migraine treatment. A study by Khalil and colleagues involving 254 adults showed that a single BT-A injection in accordance to PREEMPT protocol reduced headache frequency and migraine days, with decreased use of analgesics and triptans.[24] Aicua-Rapun and colleagues supported that findings and further confirmed long-term effectiveness and safety of BT-A.[25] Furthermore, Ilgas-Aydinlar and colleagues found decreased Migraine Disability Scores (MIDAS) with repeated BT-A injections, though sleep quality did not improve.[26] These findings highlight BT-A's benefits in treating chronic migraine, regardless of gender or age, and its suitability for elderly patients where other treatments might be unsuitable.

RECONSTRUCTIVE SURGICAL DECOMPRESSION

Initially, clinicians recognized the supraorbital and supratrochlear nerves as primary triggers for chronic migraines due to compression from the corrugator supercilii muscle (see **Figure 1**).[27] Research later re-identified that as site I and expanded this to include the temporal nerve (zygomatic-temporal branch of the trigeminal nerve) as site I (see **Figure 2**), nasal nerve (terminal branches of the trigeminal nerve) as site III, and the occipital nerve (including the greater occipital nerve/GON, lesser occipital nerve/LON, and third occipital nerves/TON) as site IV.

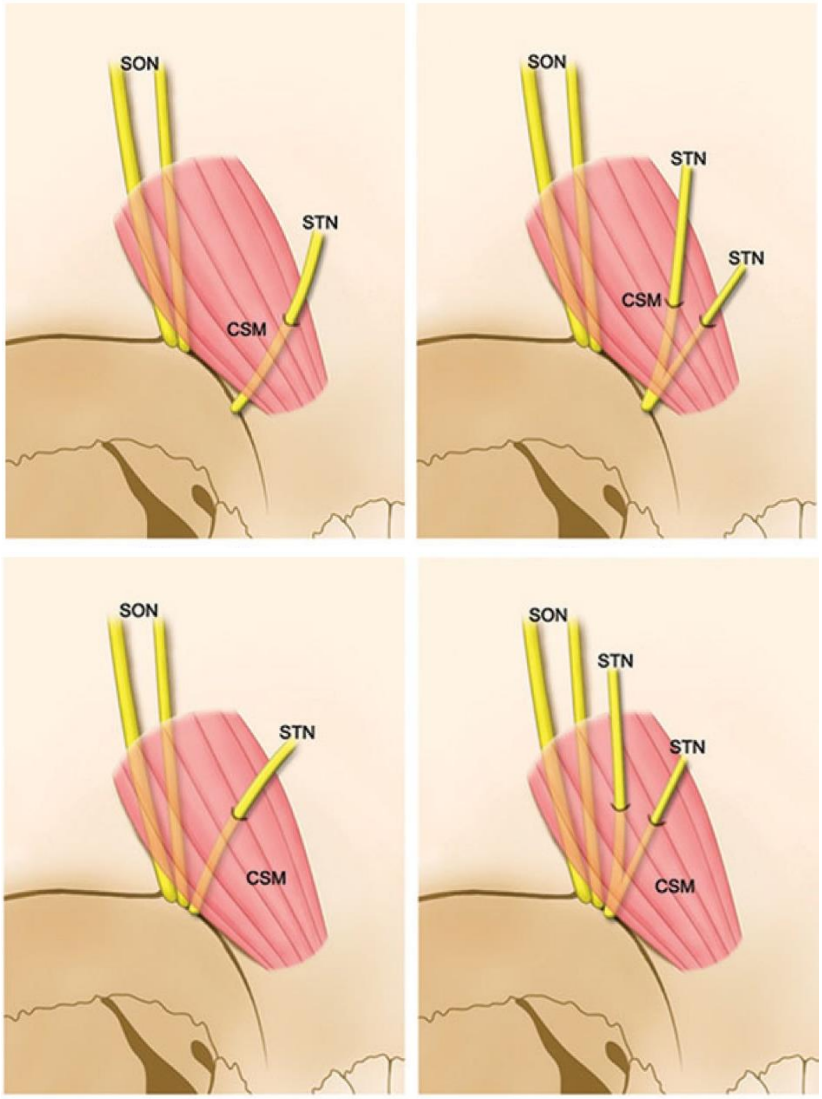
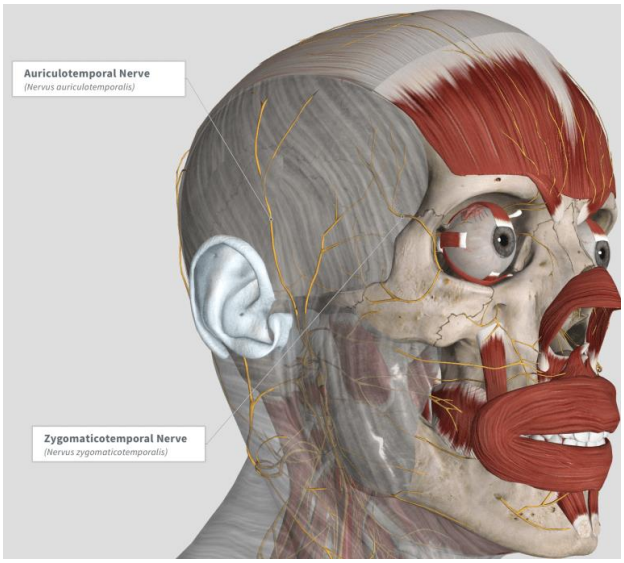


Figure 1. Illustrations showing the patterns of the supraorbital nerve (SON), the supratrochlear nerve (STN) in relation to the corrugator supercilii muscle (CSM). [27]



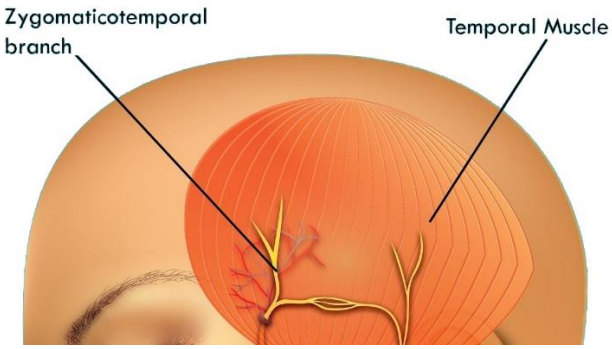


Figure 2. Illustrations showing the patterns of the zygomaticotemporal branch of trigeminal nerve in relation to the temporal muscle.

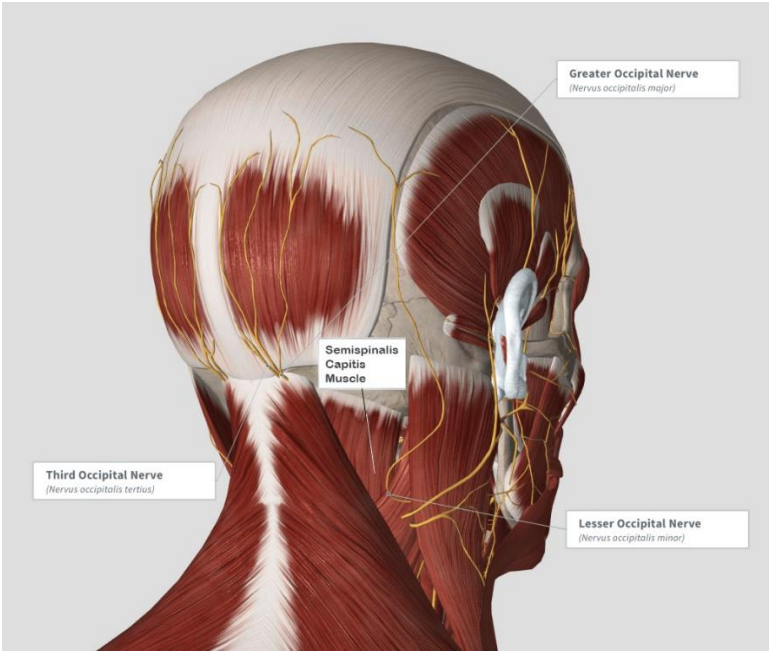


Figure 3. Illustrations showing the distribution patterns of the occipital nerves.

Surgical treatment of migraine headaches has gained popularity, especially for patients unresponsive to standard treatments. In 2018, the American Society of Plastic Surgeons (ASPS) endorsed peripheral nerve/trigger site surgery for chronic migraine, citing two decades of supportive research.[28] Peer-reviewed evidence strongly suggests that nerve compression or irritation near head and neck muscles, fascia, arteries, and bony canals are significant migraine triggers. Traditionally, occipital migraine treatment focused on GON compression by the semispinalis capitis muscle and obliquus capitis during neck flexion-extension or trauma (see **Figure 3**). However, the pulsating pain linked to occipital migraines suggests a neurovascular origin, where the terminal branches of the external carotid artery may exert pressure on the occipital nerve, leading to pulsating headaches and subsequent chronic muscle contraction around the head and neck.[29,30]

Peripheral nerve decompression, also known as trigger point deactivation or headache surgery, is a recognized treatment for chronic migraines and conditions like occipital and supraorbital neuralgia. Plastic surgeons use the Migraine Headache Index (MHI) to assess outcomes, while neurologists focus on reducing migraine days per month.[31] Although deemed safe and effective, nerve decompression surgery is suitable only for patients unresponsive to conservative treatments, with migraines originating outside the skull.

Candidate selection include a thorough history, physical examination, and diagnostic methods like nerve blocks using an injection of a local anesthetic substance and triamcinolone or Botulinum neurotoxin at the suspected trigger site can predict symptom improvement post-surgery.[32] It is important for clinicians to identify nerve compression symptoms and exclude other causes.[8]

Surgical approach to migraine headaches so far provides promising results. Lucia Mangialardi and colleagues reported long term results of decompression surgeries for frontal migraine headache, with follow-up periods ranged from 6 to 126 months, and their results showed 68.3% to 93.3% of patients reporting satisfactory outcomes in which complete migraine elimination varied from 28.3% to 59%, significant improvement rates between 26.5% and 60%, depending on the surgical technique used.[21] Li and colleagues reported 90% of patients experiencing complete headache relief after

microsurgical decompression of the greater occipital nerve, with a 20-month follow-up.[33] Blake and colleagues in 2019 reported on a 41-year-old woman with a 7-year history of chronic daily migraines and three previous occipital nerve blocks providing only temporary relief. After nerve decompression surgery, her suboccipital pain decreased by 90% during the following 3 years, although occipital and temple migraines continued about 15 days per month. The previously ineffective OnaBT-A injections became highly effective after the nerve decompression surgery, reducing migraines to 2.5 days per month. At 5.5 years post-surgery, her headaches and allodynia were significantly reduced, and she also had successful treatment of frontal headaches using OnaBT-A injections.[34]

COMBINED APPROACHES

Migraine surgery has emerged as a viable treatment for chronic migraines since clinicians started to report conventional therapies fail in relieving the symptoms significantly.[35,36] Nerve decompression surgery aims to relieve pressure on key migraine triggers such as trigeminal and occipital nerves, by removing removing muscle, blood vessels, or other tissues compressing these nerves, thus significantly reducing migraine frequency in many patients by over 50%.[29,37] Successful treatment requires plastic surgeons to collaborate closely with neurologists to pinpoint trigger points and choose the best procedure, ensuring comprehensive patient care.[37]

Some approaches combine cosmetic procedures with nerve decompression, such as eyelid surgery paired with supraorbital nerve decompression, offering both aesthetic and therapeutic benefits. Additionally, using Botox preoperatively to identify trigger points enhances surgical outcomes.[38] Integrating conventional medical therapies like medications and lifestyle changes with surgery proves more effective, significantly reducing migraine frequency and intensity while improving quality of life.[16,39]. A meta-analysis by Nagori and colleagues confirms that combining nerve decompression with medical therapy yields better long-term outcomes than either treatment alone.[40]

CHALLENGES AND CONSIDERATIONS

Many patients improve after nerve decompression surgery, but challenges persist. Variability in patient response means not all achieve the same results; some see no improvement, highlighting the need for careful patient selection to ensure optimal outcomes.[41] Surgical risks, including infection, nerve damage, and scarring, require patients to have realistic expectations, managed through thorough preoperative and postoperative counseling.[42] The procedure is also expensive, often inaccessible to those without sufficient insurance, raising ethical concerns when cosmetic motivations overlap with medical needs.[43] Additionally, the lack of long-term data on the procedure's efficacy and safety underscores the need for more extensive research.[35]

FUTURE DIRECTION AND RESEARCH

Future research in plastic surgery for migraine should target several key areas. Identifying the most effective surgical techniques and factors predicting success is crucial. Studies must compare various nerve decompression methods to find the best approaches for different patient groups.[44] Advanced imaging technologies like functional MRI and neuroimaging can enhance patient selection and surgical planning, allowing for precise identification of trigger points and reducing complications.[41] Long-term benefits of nerve decompression surgery should be assessed through longitudinal studies, which will offer insights into the durability of benefits and potential risks, aiding in clinical decision-making.[42] Additionally, understanding the psychological impact and quality of life improvements post-surgery is essential.40 Interdisciplinary collaboration between plastic surgeons, neurologists, and researchers will ensure the development of evidence-based treatment protocols, improving patient care.[42] Ethical and social aspects of using plastic surgery for migraine management also need exploration, focusing on the psychological and social effects and how to make this treatment accessible to a broader population.[43]

CONCLUSION

Migraines, a neurovascular disorder, cause chronic headaches accompanied by nausea, vomiting, photophobia, and phonophobia, severely impacting sufferers' quality of life. The WHO ranks migraines as the sixth leading cause of global disability. Types of migraines include those with aura, without aura, and chronic migraines. Management combines pharmacological methods, like NSAIDs, triptans, antihypertensive drugs, antidepressants, anticonvulsants, and botulinum toxin (BoNT) injections, with non-pharmacological approaches. OnabotulinumtoxinA injections in the head and neck areas reduce migraine frequency and intensity. Studies indicate that decompression surgery, which relieves nerve compression, also improves migraine symptoms. This type of surgery, supported by the American Society of Plastic Surgeons in 2018, is especially effective in patients unresponsive to conventional treatments. Diagnostic nerve blocks can predict success, making evaluation by a headache specialist essential to confirm chronic migraines and determine surgical eligibility. Combining conventional therapy with nerve decompression surgery significantly reduces migraine frequency and intensity, enhancing the quality of life. Collaboration between plastic surgeons and neurologists is vital for optimal results, with future research needed on the best surgical techniques and long-term effects.

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