Journal of Biosciences and Medicines, 2025, 13(12), 36-48
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

Exploring the Efficacy of Repetitive Transcranial Magnetic Stimulation in Treating Vestibular Migraine and Its Clinical Prospects

Junlong Wang, Lan Zhang*, Tao Yang*, Xia Liu, Yue Wang

Department of Neurology, The First Affiliated Hospital of Yangtze University, Jingzhou, China
Email: *lanzhang_516@163.com, *yangtao9819@163.com

How to cite this paper: Wang, J.L., Zhang, L., Yang, T., Liu, X. and Wang, Y. (2025) Exploring the Efficacy of Repetitive Transcranial Magnetic Stimulation in Treating Vestibular Migraine and Its Clinical Prospects. *Journal of Biosciences and Medicines*, **13**, 36-48.
https://doi.org/10.4236/jbm.2025.1312004

Received: November 4, 2025
Accepted: November 29, 2025
Published: December 2, 2025

Abstract

Vestibular Migraine (VM) is a common clinical neurological disorder characterized by recurrent episodes of dizziness and headaches. It has a significant genetic predisposition and a high morbidity and disability rate, posing a serious burden on patients' quality of life and social functioning. The clinical manifestations of VM are diverse and may cause notable symptoms such as nausea and vomiting, phonophobia and photophobia, blurred vision, tinnitus, and a feeling of fullness in the ears. It can also lead to atypical comorbid symptoms such as anxiety, depression, loss of interest, cognitive decline, and insomnia, which are easily overlooked but profoundly affect the psychological health and long-term prognosis of patients. Currently, its pathological mechanisms are not completely understood, and the effectiveness of clinical drug treatments varies, highlighting the urgent need for safer and more effective treatment methods. Repetitive Transcranial Magnetic Stimulation (rTMS), as a non-invasive neuromodulation technique, can precisely regulate neural circuit activity, improve brain metabolism, and promote the integration of multisensory information. It has been widely applied in the modulation of the nervous system. Several neurophysiological and clinical studies have shown that rTMS has potential application value in the adjunctive treatment of VM. This article systematically reviews the pathological mechanisms of VM, the effects and therapeutic potential of rTMS, analyzes the shortcomings and limitations present in the current research phase, and looks forward to future development directions, aiming to provide a theoretical basis and practical reference for the clinical diagnosis and treatment of VM.

Keywords

Vestibular Migraine, Repetitive Transcranial Magnetic Stimulation,

Pathological Mechanism, Treatment Progress

1. Introduction

Vestibular Migraine (VM) is a common type of disorder in neurology and is an important subtype of migraine [1]. Its etiology is complex, and its clinical manifestations are diverse. The main symptoms include long-term recurrent episodes of dizziness and migraine. The dizziness symptoms are particularly prominent, characterized by frequent occurrences and longer duration [2]. It has become the second most common cause of recurrent dizziness after Benign Paroxysmal Positional Vertigo (BPPV) [3]. Migraine, as another core symptom, varies in severity, presenting as moderate to severe pulsating pain or merely as episodic mild headaches [4]. In addition to the core symptoms mentioned above, VM may also be accompanied by phonophobia, photophobia, blurred vision, cognitive dysfunction, anxiety, depression, and sleep disturbances [5] [6]. According to epidemiological survey data, the lifetime prevalence of VM is approximately 1%, with an annual incidence rate of 0.9% [2]. The frequent episodes of VM severely affect patients' daily living abilities and quality of life, exacerbating their economic burden. Currently, the treatment of VM still primarily relies on medication. Commonly used medications include non-steroidal anti-inflammatory drugs, triptans, and ergotamine for acute pain relief, as well as beta-blockers, antiepileptic drugs, and calcium channel blockers for preventive treatment. However, long-term medication use can easily lead to drug resistance, and excessive use of acute pain relief medications may trigger medication overuse headache, thus increasing the frequency and intensity of attacks [7]. Some patients may develop a certain resistance to medication treatment, leading to poor compliance, which severely affects disease control effectiveness. Due to the various limitations of pharmacological treatment, non-pharmacological intervention strategies have gradually gained attention in recent years and have become an important research direction in the management of VM.

Transcranial Magnetic Stimulation (TMS) is a non-invasive neuromodulation technique that was first proposed by Barker *et al.* in 1985 [8] [9]. This technique regulates the excitability and synaptic plasticity of cortical and subcortical neurons by applying magnetic field pulses to specific areas of the brain, thereby further influencing the functional connectivity of neural circuits at the whole-brain level [10] [11]. Depending on the stimulation pattern, TMS can be classified into single-pulse TMS (sTMS), paired-pulse TMS (dTMS), and repetitive TMS (rTMS) [12]. Currently, rTMS is relatively widely used in treatment, and it can be divided into low-frequency and high-frequency types based on the differences in stimulation frequency. Low-frequency rTMS reduces the excitability of the stimulated area through Long-Term Depression (LTD) [13] [14]; high-frequency rTMS directly excites the cortical center through Long-Term Potentiation (LTP), enhancing the

efficiency of neural signal transmission [15] [16]. The rTMS technique has advantages such as being non-invasive, well-tolerated, and having lower treatment costs, demonstrating good safety and patient acceptance in clinical applications [10]. Nevertheless, as a clinical tool, rTMS still requires attention to its overall safety. Common side effects include mild headaches, scalp discomfort, or brief dizziness, which are mostly temporary and self-relieving; serious adverse events such as seizures are relatively rare. Major contraindications involve intracranial metal implants, uncontrolled epilepsy history, implanted electronic medical devices, and special conditions such as pregnancy, necessitating strict evaluation of individual patient risks before clinical application. In addition to rTMS, other non-invasive neuromodulation techniques, such as transcranial Direct Current Stimulation (tDCS) and non-invasive Vagus Nerve Stimulation (nVNS), have also gained attention. tDCS modulates cortical excitability through weak currents, is easy to operate but has lower spatial resolution; nVNS affects brainstem and cortical networks by stimulating the vagus nerve and is often used for acute intervention in migraines. In contrast, rTMS, with its higher spatial localization accuracy and ability to directly induce neural plasticity, shows unique potential in mechanism research and long-term treatment. In recent years, with the deepening of neuromodulation concepts, the potential of rTMS in the treatment of VM has gradually gained attention. This article aims to clarify the potential mechanisms of rTMS intervention in VM, hoping to provide theoretical references and treatment ideas for research and clinical work in related fields.

2. The Mechanism of Repetitive Transcranial Magnetic Stimulation in the Treatment of Vestibular Migraine

Patients with VM exhibit multidimensional abnormalities in brain function and metabolism, and rTMS has been shown to have significant efficacy in alleviating VM symptoms. However, there is currently no consensus on the specific mechanisms by which rTMS treats VM. The prevailing theories include the trigeminal theory, cortical inhibition diffusion theory, ion channel theory, neurotransmitter theory, and central signal integration abnormality theory, among others [17]. The pathogenesis of VM is a complex multi-stage process involving pain coding, conduction, central processing and regulation, as well as vestibular and sensory information integration dysfunction [18]. Below, based on different theories and combined with experimental and clinical evidence from scholars around the world in recent years, the potential mechanistic pathways of rTMS in the intervention of VM will be discussed one by one.

2.1. rTMS Modulates Trigeminal Nerve Function

The trigeminal nerve, as one of the 12 pairs of cranial nerves, primarily regulates sensation in the oral, nasal, and facial regions, and innervates the masticatory muscles [19]. When the branches of the trigeminal nerve are abnormally activated, electric shock-like, knife-like, or tearing pain often occurs, a characteristic also

commonly seen during vestibular migraine attacks. During VM attacks, the afferent pathways of the trigeminal nerve are abnormally activated and sensitized, prompting depolarization of neurons and subsequently causing abnormal excitation [20]. It is noteworthy that there is a close bidirectional connection between the trigeminal nerve and the vestibular system: its fibers project to the vestibular nuclei, participating in the integration and processing of balance information [21]. On one hand, excessive excitation of the trigeminal nerve can trigger a neurogenic inflammatory response, leading to intense dilation and increased permeability of the meninges and inner ear microvessels, causing local tissue edema and microcirculation disorders. This pathological change not only impairs local microcirculation but also disrupts the balance of endolymph production and absorption, inducing endolymphatic hydrops (membranous labyrinth edema). At the same time, the cochlea and vestibular organs fall into an ischemic state, resulting in abnormal discharges. Ultimately, erroneous vestibular balance signals are transmitted to the central nervous system, leading to symptoms of vestibular dysfunction such as vertigo and balance disorders. On the other hand, the ascending projection pathways of the trigeminal nerve include pain control centers such as the medullary ventromedial nucleus, periaqueductal gray matter in the midbrain, locus coeruleus, and raphe nuclei [22]. Once these structures are activated, they may disrupt central inhibitory functions, causing changes in the concentration of key neurotransmitters such as norepinephrine and serotonin (5-HT), which in turn lead to abnormal transmission of pain signals, resulting in pain sensitization and excessive amplification of pain signals [21]. Multiple studies have confirmed that trigeminal nerve-mediated vestibular symptoms and migraine attacks are closely related to fluctuations in cortical excitability.

In recent years, rTMS has shown significant potential in the treatment of VM neural regulation. The pathological process of VM is closely related to the dysfunction of the trigeminal-vestibular system. rTMS can exert multi-level intervention effects on this pathway by modulating cortical excitability. This mechanism is believed to be closely related to the overall modulatory effect of rTMS on neural networks [23]. Scholars like Jayantee Kalita *et al.* applied high-frequency rTMS to the left dorsolateral prefrontal cortex as the target area. The study explored the effects of this intervention on sound phobia symptoms and Brainstem Auditory Evoked Potentials (BAEP) in patients with migraines. It was found that rTMS significantly reduced the frequency of headache attacks, Visual Analog Scale (VAS) scores, and the degree of sound fear by indirectly inhibiting the abnormal activation of the trigeminal vascular system through modulating cortical excitability. This result suggests that high-frequency rTMS may improve various clinical symptoms caused by the over-excitation of the trigeminal nerve by regulating the motor cortex and brainstem neural pathways [24]. Another case study by Sascha Freigang *et al.*, which used neuro-navigation technology to accurately locate the left primary Motor Cortex (M1) hand and facial representation areas, showed that a patient with refractory trigeminal neuralgia who had recurrent pain after microvas-

cular decompression achieved significant and lasting pain relief after long-term rTMS treatment. With symptom improvement, the patient gradually reduced and eventually discontinued the use of antiepileptic medications such as oxcarbazepine and pregabalin, demonstrating that rTMS treatment not only alleviated symptoms but also potentially reduced reliance on traditional medications [25]. In summary, rTMS, by modulating the function of the trigeminal-vestibular pathway, is expected to become a new strategy for treating VM. Particularly in regulating cortical excitability and improving central sensitization, rTMS shows unique advantages, but further research with larger samples is still needed to validate its extensive application value.

2.2. Application of rTMS in Cortical Inhibition Diffusion

Cortical Spreading Depression (CSD) theory was first proposed by Leao in the 1940s through extensive experimental research. This mechanism is now widely recognized as the core initiating link of the neurological symptoms of VM, particularly the aura phenomena. The characteristic of CSD is a slowly propagating depolarization wave that spreads over seconds to minutes among neurons and glial cells in the cerebral cortex [26]. This depolarization process disrupts the ionic homeostasis of the cell membrane, leading to a sharp increase in intracellular calcium ion concentration, which activates multiple intracellular signaling pathways, ultimately causing neurons to enter a state of sustained inhibition [27]. If this inhibition process spreads to the visual and somatosensory cortical areas such as the occipital and parietal lobes, it may manifest as typical visual auras; when it further affects vestibular-related cortical areas or brainstem vestibular nuclei such as the posterior insula and temporoparietal junction, it may induce dizziness and other vestibular symptoms. When the extracellular local potassium ion concentration increases, it promotes calcium ion influx, forming a positive feedback loop that exacerbates vestibular dysfunction. Meanwhile, the inhibition of cortical synaptic activity that accompanies depolarization can cause significant changes in cerebral blood flow, leading to a protective increase in blood flow in the short term, but sustained inhibition may result in insufficient perfusion and ischemia of local brain tissue [26]. This rapid change in blood flow participates in the onset process of migraines by triggering rapid vascular dilation and constriction responses.

Research has found that high-frequency rTMS can reduce the occurrence of CSD by increasing the threshold of the cerebral cortex to CSD. Low-frequency rTMS, on the other hand, reduces the propagation of CSD by inhibiting the abnormally excited neuronal network. Babak Khodaie and others found that in a potassium chloride-induced CSD rat model, after rTMS intervention, the volume of normal neurons in the hippocampal dentate gyrus, somatosensory cortex, and entorhinal cortex significantly increased, while the number of apoptotic neurons and dark neurons significantly decreased. This indicates that rTMS not only alleviates cell damage caused by depolarization by regulating ionic homeostasis but also enhances neural plasticity, improving the brain's tolerance and repair capac-

ity against CSD. During the propagation of CSD, a large amount of ion transmembrane flow occurs, requiring significant ATP consumption to restore membrane potential; this energy supply-demand imbalance may lead to transient energy exhaustion and abnormal glucose metabolism. rTMS regulates neuronal excitability through multiple pathways, improves cerebral blood flow and energy metabolism status, and effectively inhibits the initiation and propagation of CSD [28]. Another clinical study applied high-frequency rTMS to the left dorsolateral prefrontal cortex (DLPFC) of chronic migraine patients and found that DLPFC activation not only negatively regulates the central spinal pain pathways, inhibiting pain perception, but also promotes changes in neural plasticity, enhancing functional connectivity between motor-related brain regions. Its regulatory effect spreads to broader cortical areas through complex neural networks such as the default mode network and the salience network, ultimately achieving an overall optimization of cortical inhibitory functions [29]. Based on existing evidence, although there is no direct research confirming the clear efficacy of rTMS on VM, its multiple mechanisms, such as balancing cortical excitatory-inhibitory levels, optimizing brain energy metabolism, and enhancing neural plasticity, have demonstrated significant effects in CSD regulation. Considering that CSD is an important pathological and physiological basis for VM, rTMS is expected to become a new neuromodulation strategy for the treatment of VM by regulating vestibular-cortical interactions.

2.3. rTMS Regulates the Function of Ion Channels

Transient Receptor Potential (TRP) channels are a class of ligand-gated cation channels extensively expressed on cell membranes. They can respond to various chemical and physical stimuli, playing a crucial role in pain signal transduction and neural regulation. Research indicates that abnormal activation of these channels is closely related to neuropathic pain associated with various neurological disorders. Upon activation, TRP channels can mediate the influx of calcium and sodium ions, as well as the efflux of potassium ions, and this ionic dynamic change constitutes an important pathological basis for VM [30]. Intracellular calcium overload can trigger the release of neuropeptides and disrupt vasodilatory functions, thereby participating in the onset of migraines [31]. Continuous influx of sodium ions leads to abnormal increases in neuronal excitability. This not only activates the trigeminal ganglion and its terminals but also promotes the release of inflammatory mediators such as Substance P (SP) and Calcitonin Gene-Related Peptide (CGRP), triggering neurogenic inflammation and further exacerbating headache. Meanwhile, the imbalance between sodium and calcium ions results in elevated extracellular potassium ion concentrations, which are crucial for maintaining inner ear vestibular function. The increase in extracellular potassium concentration caused by ionic imbalance can lead to abnormal depolarization of hair cells, resulting in vestibular symptoms such as dizziness and balance disorders. At the same time, the sharp changes in sodium ion concentration can lower the action potential threshold of neurons, causing excessive excitation of the trigeminal

nucleus and vestibular nucleus, thereby worsening vestibular dysfunction.

rTMS stimulation demonstrates a multi-layered regulatory capacity. The induced electric field can influence and alter the cell membrane potential, thereby regulating the opening state and activity of ion channels. This gradually restores the physiological balance of ion concentrations inside and outside the cells, ultimately affecting a series of neural activities in the brain. In 2023, Zhu Haijun *et al.* applied high-frequency rTMS to the dentate gyrus granule cells of mouse hippocampal slices and recorded using whole-cell patch clamp techniques. They found that rTMS significantly altered the current dynamics and voltage dependence of Voltage-Gated Sodium Channels (VGSCs) and potassium channels (Kv): on one hand, it enhanced VGSC activity, causing them to open at lower membrane potentials, delay closing, and accelerate the recovery from inactivation, thus promoting sodium ion influx; on the other hand, it inhibited the peak Kv current, delaying potassium ion efflux and increasing intracellular potassium ion concentration. This bidirectional synergistic regulation of sodium and potassium ion channels comprehensively enhances neuronal excitability, revealing the intrinsic mechanism by which rTMS regulates neural function at the ion channel level [32]. Wentao Hou's research on mice also confirmed that rTMS can enhance sodium currents and inhibit potassium efflux, significantly improving neuronal stability. VM patients exhibit imbalances in ionic homeostasis and abnormal neuronal excitability. Based on this pathological feature, rTMS, through precise regulation of sodium and potassium ion channels, is expected to restore normal electrical activity in neurons [33]. Overall, these findings provide important experimental evidence for rTMS treatment of VM, and its clinical application value can be further explored in the future.

2.4. rTMS Regulation of Neurotransmitters

Neurotransmitters are a class of key chemical messengers in the nervous system. They are released by neurons and act on specific receptors on the postsynaptic membrane, completing the process of information transmission by activating or inhibiting subsequent neuronal activity [34]. During the pathogenesis of VM, various neurotransmitters are involved in the regulatory process, including Substance P (SP), Neuropeptide Y (NPY), Norepinephrine (NE), Gamma-Aminobutyric Acid (GABA), Glutamate (Glu), Dopamine (DA), Calcitonin Gene-Related Peptide (CGRP), and serotonin (5-HT). During VM attacks, abnormal neuronal excitability leads to the massive release of multiple neurotransmitters, which in turn causes significant vasodilation, increased vascular permeability, and plasma protein extravasation, triggering neurogenic inflammation. This inflammatory response can lead to the sensitization of peripheral nociceptors, resulting in severe headaches. Additionally, the continuous release of neuropeptides such as CGRP and SP promotes mast cell degranulation, releasing histamine, proteases, and other inflammatory mediators, further exacerbating local inflammation and vasodilation, leading to recurrent and progressively worsening pain. On the other

hand, the frequent and irregular vasoconstriction and vasodilation of blood vessels can affect the microcirculation of the inner ear, causing blood flow disturbances and inadequate tissue perfusion, resulting in local ischemia and hypoxia, which disrupts the homeostasis of the internal environment. This series of pathological changes ultimately impairs the function of the vestibular end organs, inducing vestibular symptoms.

rTMS induces the generation of induced currents by applying varying magnetic fields to the cerebral cortex. When the intensity of this current exceeds the activation threshold of neurons, it triggers axonal depolarization and action potentials, regulating the release of neurotransmitters at synapses, thus intervening in the pathological processes of VM. Tetsurou Ikeda *et al.* found that by stimulating the cerebellum and brainstem areas of mice and combining gene expression microarray analysis, rTMS can significantly regulate the mRNA expression of various neurotransmitter-related transport proteins, including upregulating the glutamate transporter gene Slc17a6, inhibiting the GABA transporter Slc32a1, and selectively regulating members of the glycine transporter family, indicating that rTMS has the ability to regulate neurotransmitter metabolism across multiple systems [35]. Research by Wang Xiaonan *et al.* on depressive behavior in mice shows that rTMS can upregulate the expression of the glutamate transporter GLT-1 in astrocytes, reducing extracellular glutamate concentrations, thereby improving cell apoptosis and synaptic plasticity damage caused by it. This effect not only exerts an antidepressant effect but also enhances pain tolerance [36]. Furthermore, after applying rTMS to the contralateral parietal cortex, Raffaele Nardone *et al.* found a significant increase in serum β-endorphin levels in patients, suggesting that the endogenous analgesic system is activated [37]. These results indicate that rTMS can regulate neurochemical balance through a multi-neurotransmitter, multi-target mechanism, providing an experimental and theoretical basis for clinical treatment of VM and related symptoms. Based on the neurotransmitter disorders involved in VM, rTMS is expected to restore network stability by regulating the aforementioned neurotransmitter systems, thus alleviating the occurrence of dizziness and headaches.

2.5. Regulation of rTMS on Abnormal Central Signal Integration

Abnormal central signal integration refers to functional disturbances in the central nervous system, particularly in the cerebral cortex and related neural networks. This abnormality affects the processing and integration of multisource sensory information, manifesting as signal transmission distortion, processing delays, or regulatory imbalances. These signals include somatic pain, balance from the inner ear, auditory signals from the cochlea, and visual signals from the retina. It is currently believed that the key neural structures involved in the abnormal central signal integration in VM patients include the trigeminal nucleus, vestibular nuclei, and brainstem modulation networks (such as the raphe nuclei and locus coeruleus). Additionally, the thalamic multisensory integration areas, occipital

visual cortex, and vestibular cortical areas such as the insula and parietal operculum are also involved. The generation of vestibular symptoms relies both on the conversion of mechanical stimuli by peripheral vestibular receptors and on the stepwise processing of signals by the central system. The vestibular nuclei initially perform preliminary integration of bilateral afferent signals, which are then projected upward to the cerebellum, brainstem, and thalamus, eventually transmitting to multiple brain regions including the vestibular cortex located in the parietal operculum [38]. We found significant anatomical overlap between the vestibular system and the migraine-associated neural circuits, which jointly involve critical nodes such as the trigeminal nucleus, brainstem modulation system, and thalamocortical pathways. This high correlation between structure and function allows the onset of VM to be viewed as a result of abnormal interactions and integration dysfunction between vestibular signaling pathways and migraine pathways at multiple central nodes.

rTMS achieves precise intervention on the neural activity of specific brain regions through the accurate adjustment of stimulation frequency, intensity, and target areas. Its potential in treating VM primarily stems from its regulatory effects on key nodes such as the vestibular-cortex pathway and thalamocortical loop. In a study on patients with fibromyalgia syndrome, Laura Ackermann *et al.* applied rTMS to the left dorsolateral prefrontal cortex and found a significant increase in gray matter volume in that area, improved white matter microstructural integrity, and enhanced transmission of inhibitory neurotransmitters. Diffusion tensor imaging further demonstrated that the integrity of white matter fiber bundles connecting the right cingulate gyrus, pontine-cerebellar fibers (a component of the cerebellar middle peduncle), as well as the frontal, temporal, and occipital lobes was also enhanced [39]. Another study conducted by Can Luo *et al.* on rats indicated that rTMS could effectively improve white matter damage after cerebral ischemia, promote myelin repair, and enhance trans-cortical network connectivity. These results indirectly reflect the potential regulatory effects of rTMS on abnormal central signal integration [40]. Although the direct mechanisms by which rTMS modulates central signal integration in VM patients are not yet fully understood, the aforementioned findings support its therapeutic effects. This can be realized through multiple mechanisms such as regulating neuroplasticity, restoring network balance, and improving white matter integrity, showcasing a promising application prospect. Although the direct mechanisms by which rTMS regulates central signal integration in VM patients remain unclear, existing evidence supports its role through multiple mechanisms, possibly by modulating neural plasticity, restoring network balance, and improving white matter integrity, especially targeting key nodes such as the left DLPFC and the insular cortex. rTMS is expected to correct the sensory information processing disorder caused by abnormal central signal integration, thereby providing new directions for the treatment of VM and showing promising application prospects.

In summary, rTMS acts synergistically through multiple mechanisms in the

treatment of VM, with these mechanisms closely intertwined and mutually reinforcing in the pathophysiology of VM. Abnormal trigeminal nerve function can trigger CSD, and the imbalance of ion homeostasis during CSD exacerbates the disordered release of neurotransmitters, such as the abnormality of CGRP, leading to dysregulation of central signal integration. Overall, rTMS intervenes in the vicious cycle of VM at multiple levels, from peripheral to central, by regulating cortical excitability, stabilizing ion channels, balancing neurotransmitters, and enhancing neural plasticity, effectively alleviating dizziness and pain symptoms, and demonstrating its potential as an innovative therapeutic strategy. Future research needs to further explore the dynamic interactions of these mechanisms to optimize clinical applications.

3. Summary and Outlook

Despite extensive research by scholars worldwide on the neurobiological mechanisms of VM, there is still a lack of a unified treatment plan in clinical practice. Although conventional pharmacological treatments are effective for some patients, there is significant individual variability in efficacy. Adverse reactions, often accompanied by drowsiness, fatigue, and cognitive decline, severely affect patients' long-term adherence to treatment and quality of life. Moreover, for special populations such as children and pregnant women, the choice of suitable medications is even more limited. The rTMS technology, with its ability to precisely modulate specific neural circuits, shows promising potential in alleviating VM-related dizziness, vertigo, and accompanying mood disorders. Existing evidence indicates that rTMS can exert therapeutic effects by modulating brain network connectivity and neuroplasticity, particularly for patients who are ineffective or intolerant to traditional pharmacological treatments. However, research on rTMS in VM is still limited. Currently, rTMS treatment for VM faces challenges such as borrowing stimulation parameters from other diseases, lacking specific target localization based on VM neuropathological characteristics, small sample sizes, short follow-up periods, and the absence of multicenter randomized controlled trial support. Future research needs to investigate the optimal treatment parameters for rTMS in VM patients and conduct multicenter, large-sample randomized controlled trials. Additionally, it is essential to gain a deeper understanding of how individual differences, such as age, gender, neurochemical indicators, and brain network characteristics, affect the efficacy of rTMS, to promote the development of personalized treatment strategies. At the mechanistic level, further studies should explore the impact of rTMS on the reorganization of the vestibular system neural networks and neurotransmitter balance, providing a solid theoretical foundation for its clinical application. In summary, rTMS has demonstrated significant potential as an adjunctive treatment method for VM. With the continuous accumulation of clinical research, there is hope for utilizing rTMS to adjust stimulation frequency and sites based on the different symptoms of VM patients, achieving personalized precision treatment and offering patients safer and more effective non-pharmacological therapy options.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Kovalchick, J. (2022) Recognizing Vestibular Migraine. *JAAPA*, **35**, 21-26. https://doi.org/10.1097/01.jaa.0000830172.67790.85

[2] Dieterich, M., Obermann, M. and Celebisoy, N. (2016) Vestibular Migraine: The Most Frequent Entity of Episodic Vertigo. *Journal of Neurology*, **263**, 82-89. https://doi.org/10.1007/s00415-015-7905-2

[3] Koc, A. and Cevizci Akkılıc, E. (2022) Effects of Vestibular Rehabilitation in the Management of Patients with and without Vestibular Migraine. *Brazilian Journal of Otorhinolaryngology*, **88**, S25-S33. https://doi.org/10.1016/j.bjorl.2021.07.011

[4] Arnold, M. (2018) Headache Classification Committee of the International Headache Society (IHS) the International Classification of Headache Disorders. *Cephalalgia*, **38**, 1-211.

[5] Lempert, T. and von Brevern, M. (2019) Vestibular Migraine. *Neurologic Clinics*, **37**, 695-706. https://doi.org/10.1016/j.ncl.2019.06.003

[6] Ashina, M., Terwindt, G.M., Al-Karagholi, M.A., de Boer, I., Lee, M.J., Hay, D.L., *et al.* (2021) Migraine: Disease Characterisation, Biomarkers, and Precision Medicine. *The Lancet*, **397**, 1496-1504. https://doi.org/10.1016/s0140-6736(20)32162-0

[7] Hird, M.A. and Sandoe, C.H. (2023) Medication Overuse Headache: An Updated Review and Clinical Recommendations on Management. *Current Neurology and Neuroscience Reports*, **23**, 389-398. https://doi.org/10.1007/s11910-023-01278-y

[8] Jayathilake, N.J., Phan, T.T., Kim, J., Lee, K.P. and Park, J.M. (2025) Modulating Neuroplasticity for Chronic Pain Relief: Noninvasive Neuromodulation as a Promising Approach. *Experimental & Molecular Medicine*, **57**, 501-514. https://doi.org/10.1038/s12276-025-01409-0

[9] Reeves, S.D., Deng, Z.D. and Young, J.R. (2025) A History of Transcranial Magnetic Stimulation. In: *Advances in Neuroethics*, Springer, 11-27. https://doi.org/10.1007/978-3-031-92401-9_2

[10] Lefaucheur, J.P., Aleman, A., Baeken, C., *et al.* (2020) Evidence-Based Guidelines on the Therapeutic Use of Repetitive Transcranial Magnetic Stimulation (rTMS): An Update (2014-2018). *Clinical Neurophysiology*, **131**, 1168-1169. https://doi.org/10.1016/j.clinph.2020.02.003

[11] Pei, Q., Zhuo, Z., Jing, B., Meng, Q., Ma, X., Mo, X., *et al.* (2019) The Effects of Repetitive Transcranial Magnetic Stimulation on the Whole-Brain Functional Network of Postherpetic Neuralgia Patients. *Medicine*, **98**, e16105. https://doi.org/10.1097/md.0000000000016105

[12] Szymoniuk, M., Chin, J.H., Domagalski, Ł., *et al.* (2023) Brain Stimulation for Chronic Pain Management: A Narrative Review of Analgesic Mechanisms and Clinical Evidence. *Neurosurgical Review*, **46**, Article No. 127. https://doi.org/10.1007/s10143-023-02032-1

[13] Zheng, J., Zhang, W., Liu, L. and Hung Yap, M.K. (2023) Low Frequency Repetitive Transcranial Magnetic Stimulation Promotes Plasticity of the Visual Cortex in Adult Amblyopic Rats. *Frontiers in Neuroscience*, **17**, Article 1109735. https://doi.org/10.3389/fnins.2023.1109735

[14] Moretti, J., Terstege, D.J., Poh, E.Z., Epp, J.R. and Rodger, J. (2022) Low Intensity

Repetitive Transcranial Magnetic Stimulation Modulates Brain-Wide Functional Connectivity to Promote Anti-Correlated C-Fos Expression. *Scientific Reports*, **12**, Article No. 20571. https://doi.org/10.1038/s41598-022-24934-8

[15] Fu, R., Xu, G.Z., Zhu, H.J., *et al.* (2021) Research Progress on the Effects of Transcranial Magnetic Stimulation on Learning and Memory and Neurosynaptic Plasticity of the Brain. *Journal of Biomedical Engineering*, **38**, 783-789.

[16] Noda, Y. (2020) Toward the Establishment of Neurophysiological Indicators for Neuropsychiatric Disorders Using Transcranial Magnetic Stimulation-Evoked Potentials: A Systematic Review. *Psychiatry and Clinical Neurosciences*, **74**, 12-34. https://doi.org/10.1111/pcn.12936

[17] Dodick, D.W. (2018) A Phase-by-Phase Review of Migraine Pathophysiology. *Headache: The Journal of Head and Face Pain*, **58**, 4-16. https://doi.org/10.1111/head.13300

[18] Ceriani, C.E.J. (2024) Vestibular Migraine Pathophysiology and Treatment: A Narrative Review. *Current Pain and Headache Reports*, **28**, 47-54. https://doi.org/10.1007/s11916-023-01182-7

[19] Chen, Q.L., Yi, D.I., Perez, J.N.J., *et al.* (2022) The Molecular Basis and Pathophysiology of Trigeminal Neuralgia. *International Journal of Molecular Sciences*, **23**, Article 3604. https://doi.org/10.3390/ijms23073604

[20] Ashina, M., Hansen, J.M., Do, T.P., Melo-Carrillo, A., Burstein, R. and Moskowitz, M.A. (2019) Migraine and the Trigeminovascular System—40 Years and Counting. *The Lancet Neurology*, **18**, 795-804. https://doi.org/10.1016/s1474-4422(19)30185-1

[21] Suzuki, K., Suzuki, S., Shiina, T., Kobayashi, S. and Hirata, K. (2022) Central Sensitization in Migraine: A Narrative Review. *Journal of Pain Research*, **15**, 2673-2682. https://doi.org/10.2147/jpr.s329280

[22] Edvinsson, J.C.A., Viganò, A., Alekseeva, A., Alieva, E., Arruda, R., De Luca, C., *et al.* (2020) The Fifth Cranial Nerve in Headaches. *The Journal of Headache and Pain*, **21**, Article No. 65. https://doi.org/10.1186/s10194-020-01134-1

[23] Somaa, F.A., de Graaf, T.A. and Sack, A.T. (2022) Transcranial Magnetic Stimulation in the Treatment of Neurological Diseases. *Frontiers in Neurology*, **13**, Article 793253. https://doi.org/10.3389/fneur.2022.793253

[24] Kalita, J., Bansal, R., Mahajan, R. and Jha, V. (2025) Effect of High-Rate Repetitive Transcranial Magnetic Stimulation on Phonophobia and Brainstem Auditory Evoked Potential in Migraine. *Molecular Neurobiology*, **62**, 14079-14088. https://doi.org/10.1007/s12035-025-05190-z

[25] Freigang, S., Fresnoza, S., Lehner, C., Jasinskaitė, D., Ali, K.M., Zaar, K., *et al.* (2023) Twenty-Three Months Repetitive Transcranial Magnetic Stimulation of the Primary Motor Cortex for Refractory Trigeminal Neuralgia: A Single-Case Study. *Life*, **13**, Article 126. https://doi.org/10.3390/life13010126

[26] Yang, M., Ren, D., Pan, X., Li, C. and Xu, S. (2024) The Role of Astrocytes in Migraine with Cortical Spreading Depression: Protagonists or Bystanders? A Narrative Review. *Pain and Therapy*, **13**, 679-690. https://doi.org/10.1007/s40122-024-00610-9

[27] Jiang, L.W., Ma, D.Q., Grubb, B.D., *et al.* (2019) ROS/TRPA1/CGRP Signaling Mediates Cortical Spreading Depression. *The Journal of Headache and Pain*, **20**, Article No. 25.

[28] Khodaie, B. and Saba, V. (2018) The Neuroprotective Effects of Long-Term Repetitive Transcranial Magnetic Stimulation on the Cortical Spreading Depression-Induced Damages in Rat's Brain. *Basic and Clinical Neuroscience Journal*, **9**, 87-100.

https://doi.org/10.29252/nirp.bcn.9.2.87

[29] Abd-Elkader, A.A., Fahmy, M.E., Ahmad, F.A.A, *et al.* (2021) The Efficacy of Repetitive Transcranial Magnetic Stimulation in Treating Patients with Chronic Daily Headache. *The Egyptian Journal of Neurology, Psychiatry and Neurosurgery*, **57**, Article No. 21. https://doi.org/10.1186/s41983-021-00278-4

[30] Souza Monteiro de Araujo, D., Nassini, R., Geppetti, P. and De Logu, F. (2020) TRPA1 as a Therapeutic Target for Nociceptive Pain. *Expert Opinion on Therapeutic Targets*, **24**, 997-1008. https://doi.org/10.1080/14728222.2020.1815191

[31] Guo, S., Christensen, S.L., Al‐Karagholi, M.A. and Olesen, J. (2024) Molecular Nociceptive Mechanisms in Migraine: The Migraine Cascade. *European Journal of Neurology*, **31**, e16333. https://doi.org/10.1111/ene.16333

[32] Zhu, H.J., Yin, X.N., Yang, H.L., *et al.* (2023) Repetitive Transcranial Magnetic Stimulation Enhances the Neuronal Excitability of Mice by Regulating Dynamic Characteristics of Granule Cells' Ion Channels. *Cognitive Neurodynamics*, **17**, 431-443. https://doi.org/10.1007/s11571-022-09837-8

[33] Hou, W., Fu, R., Zhu, M., *et al.* (2023) Effects of Repetitive Transcranial Magnetic Stimulation on Neuronal Excitability and Ion Channels in Hindlimb Unloading Mice. *Journal of Biomedical Engineering*, **40**, 8-19.

[34] Südhof, T.C. (2021) The Cell Biology of Synapse Formation. *Journal of Cell Biology*, **220**, e202103052. https://doi.org/10.1083/jcb.202103052

[35] Ikeda, T., Kobayashi, S. and Morimoto, C. (2018) Gene Expression Microarray Data from Mouse CBS Treated with rTMS for 30 Days, Mouse Cerebrum and CBS Treated with rTMS for 40 Days. *Data in Brief*, **17**, 1078-1081. https://doi.org/10.1016/j.dib.2018.01.079

[36] Wang, X.N., Zhang, F.P., Niu, L., *et al.* (2023) High-Frequency Repetitive Transcranial Magnetic Stimulation Improves Depressive-Like Behaviors in CUMS-Induced Rats by Modulating Astrocyte GLT-1 to Reduce Glutamate Toxicity. *Journal of Affective Disorders*, **348**, 265-274.

[37] Nardone, R., Versace, V., Sebastianelli, L., Brigo, F., Christova, M., Scarano, G.I., *et al.* (2019) Transcranial Magnetic Stimulation in Subjects with Phantom Pain and Non-Painful Phantom Sensations: A Systematic Review. *Brain Research Bulletin*, **148**, 1-9. https://doi.org/10.1016/j.brainresbull.2019.03.001

[38] Cullen, K.E. (2019) Vestibular Processing during Natural Self-Motion: Implications for Perception and Action. *Nature Reviews Neuroscience*, **20**, 346-363. https://doi.org/10.1038/s41583-019-0153-1

[39] Ackermann, L., Zeller, D., Odorfer, T., Homola, G.A., Kampf, T., Pham, M., *et al.* (2025) Effect of Repetitive Transcranial Magnetic Stimulation on the Symptoms and Brain Imaging in Patients with Fibromyalgia Syndrome: A Randomized Controlled Pilot Trial. *Pain and Therapy*, **14**, 1547-1572. https://doi.org/10.1007/s40122-025-00770-2

[40] Luo, C., Tang, X., Shao, H. and Guo, F. (2025) High-Frequency Repetitive Transcranial Magnetic Stimulation Attenuates White Matter Damage and Improves Functional Recovery in Rats with Ischemic Stroke. *Neuroscience*, **575**, 48-56. https://doi.org/10.1016/j.neuroscience.2025.04.024