SHORT COMMUNICATION

Therapeutic application of rtms in atypical parkinsonian disorders

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ABSTRACT

Progressive Supranuclear Palsy (PSP), Corticobasal Degeneration (CBD), Multiple System Atrophy (MSA), and dementia with Lewy bodies are the four principal forms of sporadic neuronal multisystem degeneration that are referred to as Atypical Parkinsonian Disorders (APDs), parkinson plus syndromes and Lewy Body Dementia (LBD). APDs are characterised by a wide range of symptoms and a paucity of disease-modifying medications; as a result, symptomatic therapy is the norm. Repetitive Transcranial Magnetic Stimulation (RTMS) is a noninvasive and safe brain stimulation technique that uses a magnetic coil and is used to treat a variety of neuropsychiatric disorders. In this work, we evaluate the research on the efficacy of Repetitive Transcranial Magnetic Stimulation (RTMS) in the treatment of various APDs and Parkinson+syndromes. The majority of research has found that it helps with both motor and nonmotor symptoms.

Rehabilitation programme in Namibia if tailored to the contextualized needs for the cardiac patients in Namibia.

Key Words: *Parkinsonian disorders; Atypical parkinsonian disorders; Transcranial magnetic stimulation*

INTRODUCTION

Atypical Parkinsonian Disorders/Parkinson plus Syndromes are a type of Parkinson's disease. Atypical Parkinsonian Disorders (APDs), also known as "Parkinson Plus" syndromes, include Progressive Supranuclear Palsy (PSP), Multiple System Atrophy (MSA), Corticobasal Degeneration (CBD), and Lewy Body Dementia (LBD). As a result of the various underlying pathophysiological mechanisms, the clinical presentations of these syndromes are extremely diverse. An atypical parkinsonian syndrome with symmetric distribution, rapid deterioration, and poor response to medications characterises these disorders. Other atypical clinical symptoms include supranuclear gaze palsy, asymmetrical apraxia, early postural instability, early dementia, and autonomic system symptoms, in addition to Parkinsonism. Based on the abnormally accumulating protein that contributes to neurodegenerative damage, APD is split into "synucleinopathies" and "tauopathies." PSP and CBD are tauopathies, while MSA and LBD are synucleinopathies. Although APDs are less common than PD, differential diagnosis is critical since disease progression and functional losses frequently occur earlier in APDs than in PD, and traditional PD medications are only partially effective. PSP is the most frequent of the atypical parkinsonian syndromes, and it can be difficult to identify from Parkinson's disease. The most common and typical manifestations of this condition include early postural instability and falls, which are often accompanied by an akinetic stiff syndrome and ocular dysfunction. Richardson's syndrome was named after the phenotypic of PSP that had these symptoms (PSP-RS). The criteria for the clinical differential diagnosis of PSP had remained unchanged from 1990 until the 2017 update. PSP comprises a variety of different clinical phenotypes, and the 2017 update identified 10 of them, with Richardson's syndrome (PSP-RS) being only one of them. PSP is characterised by an overexpression of a specific tau protein isoform, 4R-tau, which has four microtubule-binding repeat domains. The most prevalent pathological anomaly in PSP is the tufted astrocyte, however Neurofibrillary Tangles (NFTs) and coiled bodies also play a role in the disease. The diverse clinical manifestations are caused by the different locations where tau protein accumulates. Pure akinesia is a symptom of brainstem illness, whereas a focused cortical syndrome is a symptom of cortical pathology. Despite the fact that cerebellar symptoms in PSP are uncommon, studies have demonstrated a significant role for cerebellar structures, particularly the dentate nucleus, in disease. MSA is a neurodegenerative disease that causes parkinsonism, cerebellar ataxia, and autonomic dysfunction, among other symptoms. Two main MSA phenotypes are identified based on the prevailing symptoms: MSA-C with significant cerebellar symptoms and MSA-P with prominent parkinsonian features. Both kinds have sleep abnormalities (especially RM sleep behaviour disturbances), autonomic failure, and respiratory dysfunction, which can occur years before motor symptoms appear [1]. MSA is a synucleinopathy characterised by glial cytoplasmatic inclusions created by fibrillated -synuclein proteins in the striato-nigral and olivo-ponto-cerebellar regions, as previously described. CBD is a rare degenerative neurological illness marked by asymmetrical cortical brain shrinkage, which is usually more severe in the frontoparietal regions, as well as deteriorated basal ganglia. The term CBD refers to the pathology of a condition that frequently, but not always, coexists with the corticobasal syndrome's Clinical Symptoms (CBS). Asymmetric hand dysfunction, bradykinesia, dysphagia, tremor, stiffness, dystonia, and gait and postural instability are common motor symptoms in the CBS phenotype, while cognitive impairment, visuospatial impairments, and apraxia are common nonmotor symptoms. Finally, LBD, which includes Dementia with Lewy Bodies (DLB) and Parkinson's disease dementia, is the second most common neurodegenerative dementia after Alzheimer's Disease (AD). The aggregation of a-synuclein, which results in so-called Lewy bodies, is a pathogenic feature of this illness. The most common and usual symptoms are Parkinsonism, cognitive impairment, significant behavioural abnormalities, vivid and repeated hallucinations, and antipsychotic treatment sensitivity. Another feature of these entities is the lack of effective Disease-Modifying Medications (DMDs) or other therapy options in this area. The therapy of APDs is mostly symptomatic, such as when dystonia develops with botulinum injections, and levodopa is either useless or useful for a short period of time, therefore there is no easy way to alleviate Parkinsonism symptoms. As a result, it is clear that safe and effective treatment choices are critical. The application of Transcranial Magnetic Stimulation is a new study topic that has been gaining traction in this approach (TMS) [2].

Principles of TMS

TMS was the first noninvasive brain stimulation technology to be introduced. TMS activates neurons by using a magnetic coil to target the scalp and produce a high-intensity pulse. The stimulation of the neurons can vary depending on the exact protocol and the various coil parameters, resulting in a wide range of intervention potentials. Single and paired stimuli are typically used in pathophysiological investigations, whereas studies evaluating the therapeutic application of TMS use a sequence of repeating stimuli repetitive TMS (rTMS). Cortical excitability can be altered by repetitive transcranial magnetic stimulation (rTMS) at specific frequencies or patterns, and the effects can continue for a long time. Through its effect on blood circulation within the CNS, neuronal metabolism, and excitability of the cortex directly receiving stimulation as well as areas related to the stimuli's goal, rTMS can cause long-term alterations. The plasticity of the cortex is modulated by brain stimulation in general. Long-Term Potentiation

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(LTP) and long-term depression are used to cause these changes. The essential factors that characterise a stimulation regimen are frequency, duration, and intensity, and its effects can be either excitatory or inhibitory. Low-Frequency Stimulation (LF-rTMS) (less than 1Hz) generates LTD and decreases cortical excitability, whereas high-frequency stimulation (HF-rTMS) (>1Hz) induces LTP and raises cortical excitability. TMS has been linked to a number of negative side effects. The most common symptoms are transient headaches and scalp discomfort, which are caused by the activation of scalp pericranial muscles [3]. Furthermore, the most serious adverse effects include mood swings (induced mania), scalp burns, and seizures. However, because these side effects are so rare, rTMS is often regarded as a safe therapy option.

A growing body of evidence suggests that the cerebellum has a role in PSP pathogenesis. Tau isoforms have been observed to build up in the cerebellum, resulting in a reduction in cerebellar volume. TMS studies have also revealed a loss of functional connection between the contralateral primary motor cortex (M1) and the cerebellar hemispheres Cerebellar Brain Inhibitory circuit (CBI). Some PSP symptoms, such as akinesia and rigidity, are only partially and temporarily alleviated by levodopa, with postural instability remaining a significant issue. A series of studies has looked into the effectiveness of cerebellar rTMS in PSP based on these considerations. The PSP-RSc was given after a resting state functional magnetic resonance (rs-fMRI) scan. All PSP patients reported a significant improvement in dysarthria after receiving iTBS treatment, and two out of ten patients reported a significant improvement in gait. Only the CBI measurements improved as a result of the stimulation. According to the findings, PSP patients who had cerebellar iTBS demonstrated some clinical improvement as well as an increase in functional connectivity between the cerebellum's hemispheres, the caudate nucleus, and the cerebral cortex. Due to the open-label trial design, a placebo effect could not be ruled out. In a sham-controlled case study, the efficacy of rTMS over the cerebellum in PSP was also studied [4]. They used neuronavigation to examine CBI in two patients with PSP before and after cerebellar HF-rTMS or sham TMS, collecting posturography data and speech samples before and after the intervention. Reading a standard text was used to assess speech quality, and the tempo of speech, articulatory difficulty, and article and phonemic errors were all observed. 4,000 pulses were administered over the cerebellum (10 Hz, 90%-110% of Resting Motor Threshold (RMT) intensity in the precise rTMS procedure.

Stimulation of the motor area

Disinhibition of the motor cortex has been found to be a common feature of PSP pathology. RTMS has already been considered as a potential therapy for parkinsonism in Parkinson's disease, and its therapeutic contribution to other similar disorders like PSP is being investigated, particularly in terms of axial rigidity and falls, which are cardinal symptoms of PSP [5].

Stimulation of the dorsolateral prefrontal cortex

In PSP patients, anomalies in the prefrontal cortex are hypothesised to be the pathophysiological basis of depression. Following this line of reasoning, and in light of the fact that rTMS over this area is strongly recommended for the treatment of major depression in the most recent guidelines, LF-rTMS (1 Hz, 120% RMT intensity) was applied to the right Dorsolateral Prefrontal Cortex (DLPFC) of a 62-year-old PSP male patient with treatment-resistant major depression. Depressive symptoms and apathy improved after rTMS application; more specifically, the MADS and STAI scores decreased, while the LARS and GAF scale scores increased. This case study demonstrates that repetitive Transcranial Magnetic Stimulation (rTMS) over the right DLPFC can help PSP patients feel better and live longer [6].

Multiple System Atrophy (MSA) is a disease that affects (MSA)

cerebellar stimulation is a method of stimulating the cerebellum

Patients with MSA typically have poor movement control due to cerebellar dysfunction and damage to cerebellar neural pathways. Purkinje cells suppress the cerebellar dentate nuclei, which typically exert excitatory effects on the M1 area via the ventral thalamus, in the cerebellum-M1 circuit. Purkinje cell loss in MSA indicates a disinhibition of the dentate nucleus and its excitatory function, making it a target in rTMS investigations. It was discovered that cognitive problems in MSA patients were linked to Short-Latency Afferent Inhibition (SAI). SAI is a neurophysiological tool that measures motor cortex excitability modulation and also corresponds to cholinergic system-mediated inhibition of the brain cortex [7].

DISCUSSION AND CONCLUSION

The majority of rTMS studies on parkinsonism are focused on Parkinson's Disease (PD). Given the high prevalence of this degenerative disease, this is reasonable; however, the small number of studies on Atypical Parkinsonian Disorders (APDs) highlights the need for more research into these diseases, as they affect a large number of people and may ultimately be more debilitating than PD due to the lack of effective treatments. CBI is a cerebellar Purkinje cell-mediated physiological cortical inhibition that is necessary for optimal motor control. TMS studies have indicated that stimulating the cerebellum activates the cerebello-thalamo-cortical circuit and restores CBI, which could explain the improvement in kinetic metrics shown in PSP cerebellar rTMS investigations. In addition, rTMS over the motor region and the DLPFC had positive effects on motor and depressed symptoms, respectively. Nonetheless, a number of questions arise that remain unaddressed. PSP patient groups in practically all of the aforementioned investigations almost entirely contained the Richardson's syndrome subgroup of PSP. Another topic that has to be considered is the use of rTMS in the early stages of disease. For example, in the case of Alzheimer's Disease (AD), a prevalent degenerative ailment accompanied by dementia, the research showed that patients in earlier stages of the disease responded better to rTMS treatment. These phenomena could be explained by a lower level of brain atrophy, which contributes to enhanced rTMS response.

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