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Perspective

Correlation of Cognitive Impairment with Motor Speech Disorder (MSD) in Parkinson's Diseases

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ABOUT THE STUDY

Worldwide, more than 1% of people suffer from Parkinson's Disease (PD), a widespread movement illness that is more prevalent in people over sixty. Abnormal motor symptoms of many different types are caused by PD. Due to its sluggish progression and involvement of non-dopaminergic neurons in apoptosis, Parkinson's Disease (PD) causes varying degrees of Non-Motor Manifestations (NMM) load in both young and old people, making it difficult for them to carry out everyday tasks and get social support. New research has revealed that NMM can happen in both early and late stages of PD and at any time of life. The course of motor symptoms can also be influenced by some NMM, such as autonomic symptoms, cognitive decline, mood disorders, and sensory dysfunction.

Nearly 90% of PD patients also experience Motor Speech Disorder (MSD), which is characterised by impaired motor control of the trunk and limbs. MSD is seen as a major barrier to both family care and individual psychological health because it often manifests as decreased vocal frequency and volume as well as aberrant motor component of speech control. MSD thus plays a significant unfavourable role in PD patients' quality of life. The two main therapeutic modalities for PD linked MSD at this time are routine speech therapy and medication therapies employing anti-PD medicines. In the past, ordinary speech treatment, comprising respiratory, voice, and tuning training, as well as rhythmic training, was customarily carried out for PD-associated MSD.

We hypothesised that patients with PD-associated MSD could face a larger NMM burden, specifically more severe cognitive impairment, because it has recently been demonstrated that cognitive impairment is a risk factor for different types of Speech Disorders (SD). There is currently insufficient data to determine if the MSD group has a heavier NMM load than the non-MSD group. We evaluated potential independent risk factors, such as demographic and clinical traits and Non-motor Symptom Scale (NMSS) scores, in order to fill in the current gaps in the clinical data. We wanted to advance the body of knowledge on PD-associated MSD and support upcoming investigations into its pathophysiological mechanism. Additionally, we aimed to offer data that would support the creation of fresh rehabilitation regimens and the advancement of existing ones.

The occurrence of frontal lesions in the MSD group may also be linked to a deficiency in plastic healing after PD, particularly in the frontal lobe, which is a well-known critical region for central modulation during speech motor processing. On the other hand, subsequent injury in the frontal lobe, a vital fibre network and subcortical linking structure, could happen independently of the basal ganglia. Future prospective clinical and neuroimaging research are therefore required to validate these connections and offer fresh perspectives on the neuroplastic repair in cortical regions. The specific neurophysiologic and neuropathological mechanisms underlying the aberrant speech motor modulation could be identified by analysing the cause-and-effect relationship between MSD and attention/memory impairment in PD.

It was challenging to distinguish between the two distinct cognitive categories in the MSD and non MSD subgroups, despite the fact that attention and memory are two distinct high-level cognitive functions. This might be explained by the fact that the majority of the patients displayed combined impairment of the two cognitive subtypes or by the fact that speech modulation includes both cognitive subtypes.

For the first time, it was demonstrated that MSD in PD patients had a high correlation with cognitive impairment. Early implementation of cognitive therapy may help individuals with PD-associated MSD better manage their rehabilitation and their quality of life. Future research may also evaluate cognitive impairment to identify the precise mechanisms driving improper speech motor modulation in people with Parkinson's disease.

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