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ORIGINAL ARTICLE

Cognitive Behavioural Interventions for Freezing of Gait in Parkinson's Disease: A Case Study

R.B. Uppin[1], Sana S. Momin[2], Ravi B. Patil[3]

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ABSTRACT

This case study investigates the transformative impact of cognitive behavioural interventions on freezing of gait (FOG) in an 80-year-old female patient, Sharadha, diagnosed with Parkinson's disease. Despite conventional treatments, Sharadha's FOG persisted, hindering her mobility and diminishing her quality of life.[1] Through a tailored treatment plan encompassing psycho education, cognitive restructuring, behavioural techniques, and stress management, Sharadha experienced remarkable improvements. She learned to challenge negative thought patterns, visualise fluid movement, and utilise external cues to overcome FOG episodes.[2] As a result, Sharadha regained confidence, independence, and mobility, demonstrating the profound potential of non-pharmacological interventions in managing FOG in Parkinson's disease. This study underscores the importance of holistic approaches in improving outcomes for individuals living with Parkinson's disease and offers hope for enhanced quality of life through cognitive behavioural interventions.

The implementation of the cognitive-behavioural intervention led to significant improvements in Sharadha's mobility and overall quality of life. Within several weeks of beginning treatment, Sharadha reported a marked reduction in the frequency and severity of her FOG episodes. She became more confident in her ability to walk independently, even in challenging environments. The use of external cues, combined with her cognitive restructuring efforts, allowed Sharadha to break through the "freeze" moments, significantly reducing her risk of falls.

Aim and Objectives

Aim:

- The aim of this study is to evaluate the effectiveness of cognitive behavioural interventions in reducing freezing of gait (FOG) episodes and improving mobility in individuals with Parkinson's disease (PD).

AUTHOR'S AFFILIATION:

[1] Consultant, Department of Orthopedics, Vijaya Hospital and Trauma Centre, Belgavi, Karnataka, India.

[2] Bachelor's of Physiotherapy, Department of Orthopedics, Vijaya Hospital and Trauma Centre, Belgavi, Karnataka, India.

[3] Consultant, Department of Orthopedics, Vijaya Hospital and Trauma Centre, Belgavi, Karnataka, India.

CORRESPONDING AUTHOR:

Sana S. Momin, Bachelor's of Physiotherapy, Department of Orthopedics, Vijaya Hospital and Trauma Centre, Belgavi 590001, Karnataka, India.

E-mail: sanamomin625@gmail.com

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Objective:

- To investigate the efficacy of a four-week program of repetitive exercise with cueing and movement strategies upon freezing of gait in people with Parkinson's disease.[6]

Materials

- Informed consent forms
- Participant information sheets
- Video recording equipment for capturing gait and mobility assessments

Assessment Tools:

- Freezing of Gait Questionnaire (FOG-Q)
- Unified Parkinson's Disease Rating Scale (UPDRS)
- Timed Up and Go (TUG) test
- 10-Meter Walk Test

Parkinson's Disease Questionnaire (PDQ-39) or Parkinson's Disease Quality of Life Questionnaire (PDQL)

Result: By targeting both cognitive and behavioural aspects of the condition, individuals like Mrs. Sharadha can experience meaningful improvements in symptom management and overall well-being.

Conclusion: This case study underscores the potential efficacy of cognitive behavioural interventions in addressing FOG in Parkinson's disease patients. By targeting both cognitive and behavioural aspects of the condition, individuals like Mrs. Sharadha can experience meaningful improvements in symptom management and overall well-being. Further research and clinical trials are warranted to validate the effectiveness of CBT and other non-pharmacological interventions in the treatment of FOG in Parkinson's disease[(9)]

KEYWORDS

• Parkinson's disease • Cognitive behavioural • Freezing of gait

INTRODUCTION

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterised by a range of motor and non-motor symptoms, significantly impacting the quality of life of affected individuals.[3] Motor symptoms include tremors, rigidity, bradykinesia, and postural instability, while non-motor symptoms encompass cognitive impairment, mood disorders, and autonomic dysfunction.[1] One particularly challenging aspect of PD is the occurrence of freezing of gait (FOG), an episodic phenomenon characterised by a sudden and temporary inability to initiate or continue walking. FOG often leads to falls, injuries, and a loss of independence, presenting a significant burden for patients and their caregivers.

In this case study, we explore the application of cognitive behavioural interventions in managing FOG in a patient diagnosed with Parkinson's disease.[2] Through a comprehensive treatment plan incorporating psycho education, cognitive restructuring, behavioural techniques, and stress management, we aim to mitigate the impact of FOG on the patient's mobility and overall well-being. This case study highlights the potential efficacy of non-pharmacological approaches in addressing the complex manifestations of Parkinson's disease and improving the quality of life for affected individuals.[1]

AIM AND OBJECTIVES

AIM:

- The aim of this study is to evaluate the effectiveness of cognitive behavioural interventions in reducing freezing of gait (FOG) episodes and improving mobility in individuals with Parkinson's disease (PD).

Objective:

- To investigate the efficacy of a four-week program of repetitive exercise with

cueing and movement strategies upon freezing of gait in people with Parkinson's disease.[6]

HYPOTHESIS

Alternative Hypothesis (Ha):

There is a significant reduction in the frequency and severity of freezing of gait (FOG) episodes among individuals with Parkinson's disease (PD) following participation in a cognitive behavioural intervention program.

Null Hypothesis (Ho):

There is no significant reduction in the frequency and severity of freezing of gait (FOG) episodes among individuals with Parkinson's disease (PD) following participation in a cognitive behavioural intervention program.

MATERIALS AND METHODOLOGY

Source of data: Tertiary care hospital

Methods of data collection:

- study design: experimental study. Prospective single arm intervention study
- study type:
- Target population: patient with Parkinson's disease and freezing of gait
- Duration of the study: Four weeks
- Sampling design: sample of convenience
- Sampling technique:

Inclusion Criteria: Diagnosis of idiopathic PD, presence of FOG as confirmed by clinical assessment, ability to participate in a cognitive behavioural intervention program.[8]

Exclusion Criteria: Presence of other neurological or psychiatric disorders affecting mobility, inability to participate in the intervention program due to cognitive impairment or severe physical limitations.

RESULT

By targeting both cognitive and behavioural aspects of the condition, individuals like Mrs. Sharadha can experience meaningful improvements in symptom management and overall well-being.

ETIOLOGY AND PATHOPHYSIOLOGY

1. Genetic Factors:

- Genetic predisposition plays a role in some cases of PD, with mutations in certain genes implicated in the disease's pathogenesis.[5]
- Mutations in genes such as SNCA (encoding alpha-synuclein), LRRK2 (leucine-rich repeat kinase 2), Parkin (PRKN), PINK1 (PTEN-induced kinase 1), and DJ-1 (PARK7) have been associated with familial forms of PD.[2]
- Genome-wide association studies (GWAS) have identified common genetic variants associated with an increased risk of developing sporadic PD, further highlighting the genetic component of the disease.[6]

2. Environmental Factors:

- Environmental toxins and exposures may contribute to the development of PD, although their precise role remains incompletely understood.
- Pesticides, herbicides, heavy metals (such as lead and manganese), and industrial chemicals have been implicated as potential environmental risk factors for PD.
- Additionally, factors such as rural living, well water consumption, and exposure to certain viruses have been associated with an increased risk of PD.

3. Neurobiological Mechanisms:

- PD is characterized by the degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc) region of the brain.
- The accumulation of misfolded alpha-synuclein protein aggregates, known as Lewy bodies, within neurons is a hallmark pathological feature of PD.[7]
- Dysfunction in mitochondrial bioenergetics, oxidative stress, impaired protein clearance mechanisms (such as autophagy and the ubiquitin-proteasome system), and neuroinflammation contribute to neuronal dysfunction and cell death in PD.
- Disruption of neural circuits, including the nigrostriatal pathway involved in

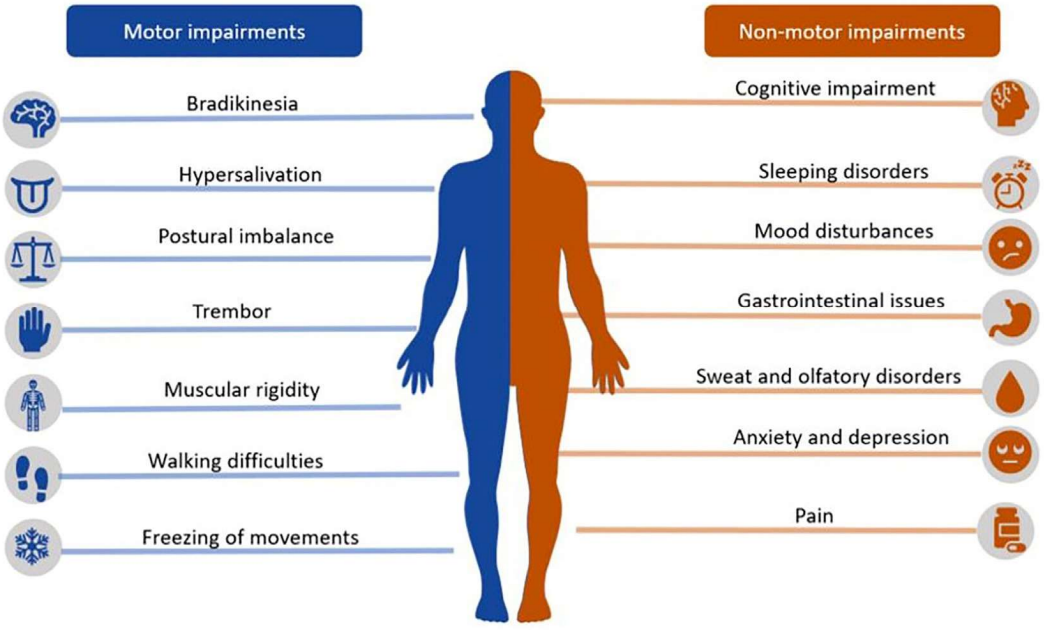
motor control, contributes to the motor symptoms observed in PD.

4. Neurotransmitter Imbalance:

- Dopamine depletion in the basal ganglia, particularly the striatum, leads to motor symptoms such as tremor, rigidity, bradykinesia, and postural instability.
- Dysfunction in other neurotransmitter systems, including acetylcholine, glutamate, and serotonin, also contributes to non-motor symptoms such as cognitive impairment, autonomic dysfunction, and mood disturbances in PD.[8]

Overall, the etiology and pathophysiology of PD are multifactorial and complex, involving a combination of genetic susceptibility, environmental exposures, and neurobiological mechanisms. Understanding these underlying factors is crucial for developing targeted therapies and interventions aimed at slowing disease progression and improving outcomes for individuals affected by PD.

CLINICAL MANIFESTATIONS:



The clinical features of Parkinson's disease (PD) encompass a wide range of motor and non-motor symptoms that can vary in severity and presentation among affected individuals.

1. Motor Symptoms:

- **Tremor**: Typically, a resting tremor characterized by rhythmic shaking of the hands, fingers, or other body parts, which often subsides during voluntary movement.
- **Bradykinesia**: Slowness of movement, including reduced amplitude and speed of voluntary movements, as well as difficulty initiating and executing movements.
- **Rigidity**: Increased muscle tone leading to stiffness and resistance to passive movement, commonly affecting the limbs, neck, and trunk.
- **Postural Instability:** Impaired balance and coordination, leading to difficulty maintaining an upright posture and an increased risk of falls, particularly in later stages of the disease.
- **Freezing of Gait (FOG):** Sudden and temporary episodes of inability to initiate or continue walking, often described as feeling "stuck to the ground.[9]"

2. Non-MotorSymptoms:

- **Cognitive Impairment:** A spectrum of cognitive deficits ranging from mild cognitive impairment to dementia, including difficulties with memory, attention, executive function, and visuospatial skills.

- **Autonomic Dysfunction:** Dysregulation of autonomic functions, leading to symptoms such as orthostatic hypotension (low blood pressure upon standing), urinary urgency, constipation, and sexual dysfunction.
- **Mood Disorders:** Depression, anxiety, apathy, and irritability are common psychiatric symptoms in PD, which can significantly impact the individual's quality of life.
- **Sleep Disturbances:** Sleep disturbances are prevalent in PD, including insomnia, excessive daytime sleepiness, restless legs syndrome, REM sleep behavior disorder (RBD), and nocturia.[10]
- **Sensory Symptoms:** Hyposmia (reduced sense of smell), visual disturbances, and pain (e.g., dystonia, musculoskeletal pain) may occur in PD.
- **Speech and Swallowing Difficulties:** Dysarthria (slurred speech) and dysphagia (difficulty swallowing) can occur due to impaired motor control of the orofacial muscles.[3]

3. Other Features:

- **Dystonia**: Involuntary muscle contractions causing abnormal postures or repetitive movements, commonly affecting the hands, feet, or neck.
- **Micrographia**: Small, cramped handwriting due to bradykinesia and rigidity.
- **Mask-like Facies**: Reduced facial expression and infrequent blinking, giving a fixed or "masked" appearance.[3]

Overall, PD is a heterogeneous disorder with a diverse array of motor and non-motor symptoms that can significantly impact the individual's functional abilities and quality of life. Effective management requires a comprehensive approach addressing both motor and non-motor aspects of the disease, tailored to the individual's specific needs and preferences.

TREATMENT STRATEGIES[4]

Pharmacological interventions targeting dopaminergic pathways remain the cornerstone of PD management, with levodopa being the most effective symptomatic therapy.[4]

- Additionally, non-pharmacological approaches, including physical therapy, occupational therapy, speech therapy, and deep brain stimulation (DBS), play a crucial role in optimizing motor function and enhancing quality of life for PD patients.
- Emerging therapeutic modalities, such as gene therapy, stem cell transplantation, and neuroprotective agents, hold promise for modifying disease progression and improving long-term outcomes in PD.

DISEASE PROGRESSION AND MANAGEMENT

- PD is a progressive neurodegenerative disorder, with symptoms worsening over time and often leading to significant disability and functional impairment.
- Multidisciplinary care involving neurologists, movement disorder specialists, rehabilitation therapists, and social support services is essential for addressing the complex needs of PD patients throughout the course of their illness.[7]

NEED OF THE STUDY

- Freezing of gait (FOG) is a distressing symptom in Parkinson's disease (PD), leading to falls, injuries, and reduced quality of life.
- Freezing of gait (FOG) in Parkinson's disease (PD) is a debilitating symptom that significantly impairs mobility and quality of life for patients.
- Cognitive behavioural interventions offer a promising avenue for managing FOG by targeting both the physical manifestations and the underlying psychological factors contributing to the symptom.[8]
- However, empirical evidence supporting the efficacy of these interventions in the context of FOG is limited. Therefore, there is an urgent need for comprehensive research to evaluate the effectiveness, feasibility, and long-term outcomes of cognitive behavioural interventions for FOG in PD.

OUTCOME MEASURES

After several weeks of cognitive behavioural therapy sessions, Sharadha experiences significant improvements in managing her FOG episodes. She becomes adept at recognizing early signs of FOG and implementing coping strategies to mitigate its impact on her mobility. With newfound confidence and resilience, Sharadha regains a sense of control over her condition, leading to enhanced mobility and an improved quality of life.[4]

PROCEDURE

1. Recruitment of eligible participants from neurology clinics or support groups for PD patients.
2. Informed consent obtained from all participants prior to enrollment in the study.
3. Baseline assessments conducted to measure baseline frequency and severity of FOG, mobility and gait parameters, and quality of life.
4. Participants undergo the cognitive behavioural intervention program, consisting of multiple sessions delivered by trained therapists over a specified intervention period (e.g., 8-12 weeks).[8]
5. Post-intervention assessments conducted to evaluate changes in FOG frequency and severity, mobility and gait parameters, and quality of life.
6. Participant satisfaction and perceptions regarding the intervention program collected through qualitative interviews or structured questionnaires.[2]

INTERVENTIONS

Recognising the limitations of conventional treatments, Mrs. Shraddha's healthcare team proposes the integration of cognitive behavioural interventions to address her FOG.

Treatment Plan:

1. **Psycho education:** Sharadha receives comprehensive education about FOG, its triggers, and its impact on daily functioning. Understanding the nature of her condition empowers Sharadha to actively participate in managing her symptoms.
2. **Cognitive Restructuring:** Guided by a therapist, Sharadha identifies and challenges negative thought patterns associated with FOG. She learns to reframe her perceptions of FOG episodes and develop adaptive coping strategies to minimise their impact on her mobility and emotional well-being.[3]
3. **Behavioural Techniques:** Sharadha undergoes training in various behavioural techniques aimed at managing FOG episodes, such as:
   - *Visual Imagery:* Sharadha practices visualizing herself walking smoothly and confidently, focusing on the sensation of fluid movement to counteract the hesitation and uncertainty that often accompany FOG episodes.
   - *External Cues:* Sharadha learns to utilize external cues, such as rhythmic auditory cues or visual markers, to facilitate gait initiation and continuation. These cues serve as prompts to bypass neurological blockages that lead to FOG.[4]
   - *Dual-Task Training:* Through dual-task training exercises, Sharadha improves her ability to perform motor tasks while engaging in cognitive activities, reducing the occurrence of FOG during complex movements.[6]
4. **Stress Management:** Sharadha adopts stress management techniques, including deep breathing exercises and progressive muscle relaxation, to alleviate anxiety and muscle tension that can exacerbate FOG episodes.

DISCUSSION

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by a range of motor and non-motor symptoms, significantly impacting the quality of life of affected individuals. Motor symptoms include tremors, rigidity, bradykinesia, and postural instability, while non-motor symptoms encompass cognitive impairment, mood disorders, and autonomic dysfunction. Treatment Strategies.[4]

Pharmacological interventions targeting dopaminergic pathways remain the

cornerstone of PD management, with levodopa being the most effective symptomatic therapy.

CONCLUSION

This case study underscores the potential efficacy of cognitive behavioural interventions in addressing FOG in Parkinson's disease patients. By targeting both cognitive and behavioural aspects of the condition, individuals like Mrs. Sharadha can experience meaningful improvements in symptom management and overall well-being. Further research and clinical trials are warranted to validate the effectiveness of CBT and other non-pharmacological interventions in the treatment of FOG in Parkinson's disease.[9]

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