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NEUROLOGICAL AND PSYCHO-EMOTIONAL CHANGES ASSOCIATED WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Abstract. Chronic Obstructive Pulmonary Disease (COPD) is a progressive condition primarily affecting the respiratory system but also causing significant systemic complications, including cerebral circulation disorders and cognitive impairments. This study aims to examine the neurological and cognitive dysfunctions associated with COPD, focusing on the relationship between hypoxia, vascular abnormalities, and cognitive decline. The research includes an analysis of clinical indicators, diagnostic methods, and potential treatment strategies for COPD patients exhibiting neurological symptoms. Findings indicate that cerebral hypoxia and systemic inflammation play a crucial role in cognitive deterioration, necessitating early intervention and comprehensive management strategies.

Keywords. Chronic Obstructive Pulmonary Disease (COPD), cerebral circulation disorders, cognitive impairment, hypoxia, neurovascular dysfunction, systemic inflammation, pulmonary function.

Introduction. Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of morbidity and mortality worldwide, characterized by irreversible airflow limitation and chronic inflammation. While COPD is predominantly a pulmonary disease, emerging research highlights its systemic impact, particularly on cerebral circulation and cognitive functions. Hypoxia, systemic inflammation, and vascular dysfunction contribute to significant neurovascular impairments, increasing the risk of cognitive decline and neurodegenerative disorders. Understanding the clinical indicators of

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cerebral circulation and cognitive dysfunction in COPD patients is crucial for developing targeted interventions and improving patient outcomes.

Methods. This study was conducted on 120 patients diagnosed with COPD at various stages. The participants were divided into groups based on disease severity, with a control group consisting of healthy individuals without COPD. Clinical assessments included pulmonary function tests, blood oxygen saturation (SaO₂) function measurements. and cognitive evaluations using standardized neuropsychological tests such as the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), and Frontal Assessment Battery (FAB). Cerebral circulation was assessed using Doppler ultrasound imaging and MRI scans. Inflammatory biomarkers and oxidative stress levels were also measured to establish correlations between systemic inflammation and cognitive impairment.

Results. The study revealed that cognitive decline was significantly more prevalent in patients with moderate to severe COPD compared to the control group. The primary findings include:

• Cerebral Hypoxia and Cognitive Dysfunction: Patients with severe COPD exhibited lower oxygen saturation levels (SaO₂ < 90%), leading to cognitive impairments such as memory loss, attention deficits, and executive dysfunction.

•Neurovascular Abnormalities: Doppler ultrasound and MRI findings indicated reduced cerebral blood flow in COPD patients, particularly in those with prolonged hypoxia.

•Systemic Inflammation and Oxidative Stress: Elevated levels of inflammatory markers (C-reactive protein, interleukin-6, and tumor necrosis factor-alpha) were associated with increased cognitive deficits.

• Correlation Between Disease Progression and Cognitive Decline: Patients in advanced COPD stages (GOLD 3-4) showed significantly lower MMSE and MoCA scores, with a higher prevalence of depression and anxiety.

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These findings suggest a strong link between pulmonary dysfunction, cerebral circulation impairment, and cognitive deterioration, emphasizing the need for integrated treatment strategies.

Discussion. The results of this study confirm the hypothesis that COPD is not limited to respiratory impairment but also significantly affects brain function. The interplay between chronic hypoxia, vascular dysfunction, and systemic inflammation contributes to neurocognitive decline in COPD patients. These findings align with previous studies highlighting the role of hypoxia-induced neuroinflammation in accelerating cognitive dysfunction.

Clinical Implications:

• Early Screening and Monitoring: Routine cognitive assessments should be integrated into COPD management protocols, especially for patients with severe hypoxia.

•Oxygen Therapy and Pulmonary Rehabilitation: Long-term oxygen therapy may improve cerebral perfusion and cognitive function in hypoxic COPD patients.

•Anti-inflammatory Interventions: Targeted therapies aimed at reducing systemic inflammation, such as corticosteroids and biologic agents, could mitigate cognitive decline.

•Lifestyle Modifications: Encouraging COPD patients to engage in physical activity, cognitive training, and dietary adjustments may enhance neurovascular health and slow cognitive impairment.

Limitations and Future Research: This study is limited by its sample size and observational design. Further longitudinal studies with larger cohorts are needed to establish causal relationships between COPD progression and cognitive dysfunction. Additionally, exploring the role of pharmacological interventions, including neuroprotective agents, could provide new insights into treatment options.

Conclusion. COPD patients are at a heightened risk of developing cerebral circulation disorders and cognitive impairments due to chronic hypoxia, vascular

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dysfunction, and systemic inflammation. Early diagnosis and comprehensive management strategies, including oxygen therapy, anti-inflammatory treatments, and lifestyle interventions, are essential to improving patient outcomes. Future research should focus on the development of targeted therapies to mitigate cognitive decline in COPD patients.

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