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Endocrine Factors and Their Impact on the Pathogenesis of Parkinson's Disease

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Abstract: *Disruption of thyroid homeostasis can play a significant role in the pathogenesis and progression of neurodegenerative disorders such as Parkinson's disease (PD). Although hyperthyroidism by itself does not directly cause Parkinson's disease, accumulating evidence from recent studies suggests that an excess of thyroid hormones may contribute to the onset or exacerbate the progression of neurodegenerative processes characteristic of PD. Elevated levels of thyroid hormones can influence dopaminergic neuronal function, potentially accelerating neurodegeneration, increasing oxidative stress, and impairing motor and cognitive regulation. Consequently, imbalances in thyroid function may serve as an important modulating factor in the severity and course of Parkinson's disease. Understanding this relationship highlights the necessity of monitoring and managing thyroid function in patients at risk for or diagnosed with PD, thereby offering a potential avenue for mitigating disease progression and optimizing therapeutic strategies.*
Key words: *thyroid homeostasis, neurodegenerative diseases, EMG, MMSE test.*

Introduction

Parkinson's disease (PD) and hyperthyroidism are two distinct clinical conditions that can both result in tremor, yet they arise from different etiologies and underlying pathophysiological mechanisms [4,8]. Parkinson's disease is a neurodegenerative disorder primarily affecting the central nervous system [10,12]. Tremor in PD is predominantly caused by apoptosis of dopaminergic neurons in the substantia nigra region of the brain, leading to dopamine deficiency. This deficiency disrupts motor control, resulting in characteristic clinical manifestations such as tremor, rigidity, and bradykinesia [11,2]. While studies directly linking Parkinson's disease and thyrotoxicosis are limited, both conditions influence the nervous system and can induce tremor. The precise mechanisms by which excess thyroid hormones impact neural function and potentially exacerbate neurodegenerative processes specific to Parkinson's disease remain an important area of investigation [14,17]. The effect of thyrotoxicosis on the dopaminergic system, as well as on the striatum and basal ganglia, has not been fully elucidated. This influence may contribute to the development and progression of Parkinsonian symptoms [3,18]. Thyrotoxicosis may act either as a precursor or a risk factor in the pathogenesis of Parkinson's disease. Currently, public health measures and healthcare systems emphasize early detection, diagnosis, and treatment of motor and non-motor impairments in Parkinson's disease, aiming to prevent disability and improve quality of life [19,20]. Achieving these objectives requires optimizing PD management strategies, including accurate diagnosis, comprehensive treatment, and disability prevention [1,22].

Rationale and Objective

Motor and non-motor impairments in Parkinson's disease can continue to progress even under levodopa therapy, presenting significant clinical challenges. Therefore, implementing a comprehensive management strategy for PD—particularly under levodopa therapy—can help mitigate long-term disability, improve patients' quality of life, and modify disease progression. Recent studies have highlighted that thyroid hormones (T3 and T4), and particularly subclinical hypothyroidism manifested as TSH deficiency, constitute risk factors for accelerated cognitive decline. Consequently, assessing motor and cognitive deficits dynamically at various stages of Parkinson's disease, and applying appropriate interventions under the guidance of endocrinologists and neurologists, may slow disease progression, stabilize clinical symptoms, and support holistic treatment approaches. Addressing thyroid homeostasis is therefore a potentially valuable adjunctive strategy, particularly in alleviating tremor, one of the hallmark symptoms of PD.

Aim

The aim of this study was to investigate the impact of disrupted thyroid homeostasis on the development and progression of Parkinson's disease.

Materials and Methods

A total of 60 patients diagnosed with Parkinson's disease were included in this study. They were divided into two groups: the main group (n=35) with coexisting hypothyroidism and Parkinson's disease, and a comparison group (n=30) with Parkinson's disease alone. Clinical-neurological assessments, laboratory evaluations of TSH, T3, T4, and thyroglobulin (TG), neuroimaging studies, and specialized neurological scales were employed, including the Hoehn-Yahr scale, UPDRS, and MMSE (Mini-Mental State Examination). Multivariate statistical analysis was applied for comprehensive evaluation.

Patients were selected based on a diagnosis of hypothyroidism and Parkinson's disease corresponding to stages II–III on the Hoehn-Yahr scale. The control group comprised 30 patients (16 males, 14 females), while the comparison group included 30 patients (17 males, 13 females). The mean age of participants was 56.82 ± 0.91 years, with a disease duration of 3.22 ± 0.15 years. Both groups received stable and adequate doses of anti-Parkinsonian medications. UPDRS assessment in the main group yielded an average score of 47.2 ± 13.6. PD diagnosis followed the clinical diagnostic criteria established by the UK Parkinson's Disease Society Brain Bank (Gibb W.R.G., Lees AJ., 1994).

Results

Clinical evaluation indicated that among PD patients, the akinetic-rigid and mixed forms were more prevalent compared to the tremor-dominant form. Motor impairments were assessed using the Hoehn-Yahr scale, which revealed an average severity score of 69.2 ± 0.7 in the main group.

Statistical Analysis of Tremor Dynamics and the Interrelation with Thyroid Dysfunction in Parkinson's Disease

Based on the statistical analysis of tremor dynamics, it was observed that in the second group (Group II, n=30), patients exhibited tremor predominantly as a result of Parkinson's disease (PD), whereas in the first control group (Group I, n=30), tremor was more dominant in a different pattern. A significant correlation was found between anti-thyroid peroxidase (TPO) antibodies and thyroglobulin (TG) concentrations, which are indicators of autoimmune thyroid damage.

In patients with Parkinson's disease presenting with tremor and mixed types, TG concentrations were markedly higher compared to akinetic-rigid patients. Similarly, in patients with tremor-dominant PD, thyroid-stimulating hormone (TSH) levels were lower relative to those with akinetic-rigid type PD. Notably, elevated triiodothyronine (T3) levels were associated with a reduction in disease severity.

The degradation of dopaminergic neurons in patients with Parkinson's disease may contribute to dysregulation of TG levels, leading to impaired motor function and an intensification of tremor. Furthermore, both hypothyroidism and hyperthyroidism may exacerbate oxidative stress, thereby disrupting dopaminergic pathways and promoting neuronal apoptosis.

Conclusion:

Thus, in the control group (Group I), patients suffering from both Parkinson's disease and hypothyroidism displayed distinct characteristics compared to Group II, which included patients affected solely by Parkinson's disease. Specifically, PD-induced symptoms manifested with more pronounced tremor and progressive cognitive impairment in Group II. Evaluating motor and cognitive dysfunction dynamically at various stages of the disease allows for timely interventions by endocrinologists and neurologists, facilitating stabilization of disease progression.

Patients diagnosed with Parkinson's disease should be screened for concomitant hypothyroidism, while patients with hypothyroidism should be assessed for Parkinson's disease. Integrating such evaluations into comprehensive treatment strategies can support optimal disease management and maintain the stability of core symptoms arising from Parkinson's disease. The coexistence of Parkinson's disease and hypothyroidism appears to accelerate neurodegeneration, leading to an increased progression of dementia and other characteristic PD symptoms.

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