Etiology of Depressive Symptoms in Parkinson's Disease: A Result from Neuroanatomical Deficiencies or a Consequence of the Psychosocial Stress of PD Diagnosis

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1. Introduction

Parkinson's Disease (PD) is a common neurological illness and various degrees of depression frequently complicate its course.[1] While the high frequency of depression in PD seems established, the nature of the relationship between the two remains uncertain. Attempting to distinguish the origin of depression in PD patients is complex due to the shared functional impairments and neuroanatomical changes of both disorders. PD is a progressive disorder that results in complete dependency of the patient at its most severe state; therefore, some research suggests depression as secondary to the reaction and consequence of the diagnosis of this persistent disease. However, the neurodegeneration that occurs in PD, which is also implicated in the neurobiological basis of depression, supports the idea of a biological origin of both PD and depression[2]. Hence, the purpose of this study is to evaluate the onset of depression in Parkinson's disease patients and establish grounds for this co-morbidity with depression by examining the psychosocial stress of the PD diagnosis triggering depressive symptoms, as well as the collective biological foundation of both PD and depression.

2. Methodology

To evaluate the link between the co-morbidity of Parkinson's disease and depression a systematic review of the literature was undertaken. The investigation utilized the Medline database and included articles from 1970 to the present. Twenty articles were reviewed using evidence-based methods. Articles included for review incorporated Parkinson's disease and depression. Variables examined consisted of neurodegeneration that occurs in PD patients, neurotransmitters involved in PD as well as depression, psychosocial stressors of a chronic illness diagnosis, and the onset of depression relative to the diagnosis of PD. Articles were chosen for analysis based on the journal's reputation, the study type (randomized controlled trials and literary reviews) and the included relevant material considered necessary to address the premise being examined.

3. Discussion

The findings in this literature review demonstrate that the diagnosis of PD, a progressive incurable disorder, is a powerful cause of despair for the patient. Activities of daily living such as walking, eating, and talking are all, in time, negatively affected and many, in the diseases later stages, are eventually unfeasible for the PD patient. The course of this disease is a long one, with pharmacological agents less able to control the physical manifestations of the disease in its later stages. Initial treatment demonstrates optimistic outcomes for the patient physically, however as the disease progresses, the characteristic traits of PD; tremor of the hands, limbs, jaw, and face, rigidity or stiffness of the limbs and trunk, bradykinesia or slowness of movement, and postural instability or impaired balance and coordination, become a battle that the patient is unable to overcome. Research on the occurrence of depression during the course of PD was seen to be greatest at three stages; the period of initial diagnosis, the period of decrease in independence, and the rapid decline and worsening of symptoms[3]. The beginnings of the disease (the initial diagnosis) and its later advancement (total dependence) have a substantial impact with respect to depressive episodes.

Alternatively, a biological basis linking PD and depression implicated common areas of neurological involvement. Depression in PD is the result of the neurodegenerative process characterized by deterioration of the subcortical nuclei, some of which have also been implicated in depression.[4] PD patients with co-morbid depression have smaller subcortical nuclei which is similar to non-PD patients with depression.[5] Changes in the brain chemical messengers are thought to be the key neurochemical mechanism behind depression and PD. Changes in both the serotonin and dopamine systems have been implicated in depression. Decreased numbers of serotonin neurons in the dorsal raphe nucleus and dopamine neurons in the ventral tegmental area are found postmortem in PD patients with a history of depression.[6] Evidence from transcranial sonography studies of depression suggests a pathophysiological role of a variation of ascending and descending pathways in the brainstem with a consecutive imbalance of neurotransmission. These similar findings in unipolar depression and depression in Parkinson's disease indicate that alteration in the basal limbic system may be a common pathway in the pathogenesis of depressive illness.[7]

4. Conclusion

Twenty articles were examined and incorporated in this literary review. Twelve articles in this review meet the criteria for Level I randomized controlled trials. The supplementary articles meet the criteria for Level II and Level III verification. In accordance with the Level I criteria reviewed, a Grade A recommendation for a dual etiology behind the co-morbidity involving the disease states of Parkinson's and depression is recommended. The Parkinson's disease patient population is biologically affected by an underlying physiological degeneration. This, paired with the fact that PD is a physically impeding illness, thus adding stress and despair to the diagnosed individual, readily explain the frequent occurrence of depression as a concurrent state. [8, Fig.1]

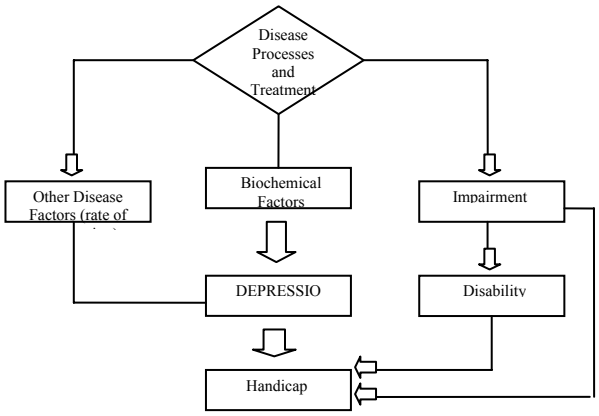

Fig. 1. Suggested Model for the multi-factorial causes of depression in patients with Parkinson's disease

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