Review Article

iMedPub Journals
www.imedpub.com

Journal of Neuropsychiatry
ISSN 2471-8548

2023
Vol.7 No.1:001

A Review on Neuropsychiatry of Parkinson's Disease

Marcos Altable[1*], Juan Moises de la Serna[2], Emilio Diaz-Moreno[3] and Muhammad Akram[4]

[1]Department of Neurology, Virgin of Africa Clinic, Ceuta, Spain

[2]Department of Neurology, Norbert Wiener Private University, Lima, Peru

[3]Department of Neuropsychology, Virgin of Africa Clinic, Ceuta, Spain

[4]Department of Medicine, Government College University, Faisalabad, Pakistan

[*]**Corresponding author:** Marcos Altable, Department of Neurology, Virgin of Africa Clinic, Ceuta, Spain; Email: maraltable@gmail.com

Received: June 08, 2023, Manuscript No. IPJNP-23-17055; **Editor assigned:** June 12, 2023, PreQC No. IPJNP-23-17055 (PQ); **Reviewed:** June 26, 2023, QC No.IPJNP-23-17055; **Revised:** June 30, 2023, Manuscript No. IPJNP-23-17055 (R); **Published:** July 28, 2023, DOI: 10.36648/2471-8548.23.07.001

Citation: Altable M, de la Serna JM, Diaz-Moreno E, Akram M (2023) A Review on Neuropsychiatry of Parkinson's Disease. J Neuropsychiatry Vol.7 No.1: 001.

Abstract

Parkinson's Disease (PD) can cause a variety of non-motor symptoms, including sadness, anxiety, sleep difficulties, psychosis, behavioural and cognitive abnormalities. Currently, research on Parkinson's disease has shifted away from focusing solely on motor symptoms, instead focusing on features that are commonly affected along the course of the disease and are extremely restricting for the patient, such as cognitive, behavioural and functional components. The neurological grounds for the development of neuropsychiatric problems in Parkinson's disease are becoming more understood. These neuropsychiatric conflicts are frequently more challenging and distressing for patients and families than the motor features of Parkinson's disease. This symptom frequently precedes typical motor symptoms, emphasising the significance of early diagnosis and treatment.

Keywords: Neuropsychiatric conflicts; Parkinson's disease; D iagnosis; Treatment declaration

Introduction

Parkinson's Disease (PD) is the second most common neurological illness, affecting about 1.7% of persons over the age of 651. Parkinson's Disease (PD) is a complicated, severe disease characterised by motor symptoms such as tremor, stiffness, bradykinesia and postural instability, as well as non-motor symptoms such as neuropsychiatric symptoms, autonomic and sensory dysfunctions and sleep problems. PD typically manifests itself in the second half of life, with a little masculine predisposition. The cause is still unknown, but Parkinson's syndromes are known, which can have a variety of causes (degenerative, viral, toxic and hereditary). Parkinson's disease is distinguished by the loss of pigmented dopaminergic neurons in the compact section of the substantia nigra as well as the appearance of Lewy bodies.

It is a biochemical collapse of the nigrostriatal dopaminergic system, with a clinical threshold of more than 80% of the system's potential loss, unbalancing numerous brain circuits, not just motors. Other neurotransmitters are also changed in Parkinson's disease, including cholinergic neurotransmission. As a result, a lack of cholinergic pathways explains some of the memory decline (Meynert's basal nucleus). On the other hand, inadequacy of the serotonergic and noradrenergic systems (locus coeruleus) may lead to akinesia and freezing phenomena, but it may also be involved in the emergence of a depressive syndrome, which occurs in 20% to 40% of patients [1]. Psychiatric issues affect up to 90% of people with Parkinson's disease.

Throughout their PD, these patients endure neuropsychiatric abnormalities such as sadness, anxiety, sleep difficulties, psychosis, behavioural and cognitive changes. These neuropsychiatric problems are frequently more challenging and stressful for patients and families than the motor features of Parkinson's disease [2].

Literature Review

In recent years, research on Parkinson's disease has shifted away from focusing solely on motor symptoms, instead focusing on features that are typically impacted along the course of the disease and are extremely restricting for the patient, such as cognitive, behavioural and functional components. We now know that many of the patients experience cognitive abnormalities ranging from Moderate Cognitive Impairment (MCI) to dementia (Parkinson's dementia) throughout the disease. What is currently known regarding the prevalence of MCI in Parkinson's disease, as well as its various cognitive characteristics, has been influenced by a number of factors [3]. On the one hand, because of the numerous definitions of "cognitive impairment" based on the use of various neuropsychological tests, some of which are not valid in this population.

On the other hand, the question of what DCL applied to this neurodegenerative disease must be understood. The

neuropsychological evaluation is not given much weight until the movement disorders society task force publishes criteria 8 for DCL-EP in 2012. Initial cognitive abnormalities in Parkinson's disease may not be obvious, but they can be diagnosed with the correct neuropsychological examination. This method allows us to detect cognitive abnormalities in people who appear to be unaffected. These deficiencies are primarily dysexecutive diseases. In this regard, the neuropsychological profile found in individuals with PD is comparable to that described in patients with frontal lobe impairment, thus strengthening the hypothesis of striatal-frontal network dysfunction secondary to dopamine deficit.

On the other hand, we can encounter patients who display clinical symptoms from the beginning, such as difficulties keeping concentration while reading, when making prolonged mental exertion, or when doing simultaneous mental processes. The difficulty in "finding the word" (the tip of the tongue phenomenon) is evident, and it is linked to deficiencies in semantic verbal fluency from the beginning. Problems remembering recent episodic occurrences are also common. Difficulties in planning activities and organising daily life can be reported by patients very early on, and they have been linked to executive dysfunction [4]. Memory and executive symptoms become increasingly noticeable as cognitive impairment advances.

In the transition to dementia, language impairments occur, and patients with PD discover obstacles for comprehending and generating language, and there is a propensity to lose the thread of the discussion arises. The standard definition of subcortical dementia in Parkinson's disease states that it mostly affects executive functioning, attention, visual perception and domains that do not influence memory. However, as previously said, memory and language impairment can be detected in these patients. It has recently been proposed that executive frontal function (frontostriatal circuits) and posterior cortical involvement (temporal and parieto-occipital) in Parkinson's disease are two distinct notions with distinct hereditary bases and predispositions.

A number of longitudinal studies have discovered that frontal-subcortical abnormalities, which have been linked to dopaminergic dysfunction in the nigrostriatal circuit, can persist for many years. They did, however, find that impairment of the most posterior cortical processes associated to the cholinergic system would raise the risk of dementia. Thus, neuropsychiatric illnesses such as cognitive impairment, which is characterised by cognitive and motor slowdown, executive dysfunction and memory loss, could be related to an alteration of the frontal-subcortical circuits.

Neuropsychiatric disorders

Depression: It is widely acknowledged that clinically significant depression symptoms occur in 40%-50% of people with Parkinson's disease. A systematic review found that 17%, 22% and 13% of patients present with major depression, moderate depression and dysthymia, respectively [5]. Depression affects 23%-40% of PD patients and depressive symptoms frequently precede typical motor symptoms, with no linear link with PD duration or severity. Some authors observed that depression was widespread in both advanced and early phases. However, it appears to connect positively with the patient's age 4. Only 25% of depressive symptoms are treated adequately. The fundamental processes of depression in Parkinson's disease are unknown. Psychosocial variables and disability are relevant, although they are not the primary determinants of depressive disorders in Parkinson's disease. Rather, neurobiological features associated with the underlying neurodegenerative disease and its somatic therapies provide a backdrop for increased incidence of depressive symptoms in Parkinson's disease patients matched for disability.

Discussion

Depression is more common in people with Parkinson's disease than in other patients with comparable disability (for example, paraplegic patients), ruling out the notion of solely reactive depression. Furthermore, the depressed syndrome's great response to tricyclic antidepressants and serotonin reuptake inhibitors, as well as its poor response to levodopa, point to an underlying biochemical change [6]. A noradrenergic deficit in the coeruleus nucleus 2 has been noted in particular. Postmortem examinations of depressed Parkinson's disease patients revealed lower densities of serotonin and dopamine neurons in the dorsal raphe and ventral tegmental areas, respectively.

The three serotonin-norepinephrine-dopamine systems co-evolve in depression. The majority of antidepressant compounds currently in use operate on the neurotransmission of one or more of these chemicals. The findings on dopamine are contentious, as they are dependent not just on dopaminergic neurotransmission but also on other brain circuits affected by its dysregulation. Depression, like Parkinson's disease, affects the basal nuclei and frontal brain. Because of these parallels in the damaged neurological circuits, depression symptoms may accompany or even precede motor symptoms in the condition. Although these signals are frequently overlooked, owing to the inherent overlap between parkinsonian and depressed symptoms (hypopmimia, apathy, etc.), they are critical for the diagnosis and treatment of depression.

Depression appears to be five times more common in individuals with left hemilateral parkinsonism and people with atypical parkinsonian syndrome experience depression more frequently than those with typical parkinsonian syndrome [7]. It should be noted that certain depressed symptoms, such as slowing or a lack of mental flexibility, are added to the symptoms of the generally associated dysexecutive condition and, as a result, may contribute to the cognitive impairment of Parkinson's disease.

Anxiety disorders

The frequency of anxiety disorders in Parkinson's disease ranges from 25% to 45%, and anxiety disorders are frequently associated with depression. Patients with Parkinson's disease who also have depression and anxiety have more severe PD symptoms, a worse response to depression treatment, and

higher functional impairment. They are distinguished by discomfort, inappropriate or exaggerated fear and the sense that something terrible will occur. They are commonly accompanied with vegetative diseases including as palpitations, excessive sweating, tremors, chest or throat tightness, shortness of breath, dry mouth and abdominal pain.

These anxiety paroxysms can occur during the motor fluctuations associated with levodopa therapy, especially during the off periods and are accompanied by intense melancholy and irritation. Non-motor symptoms can be linked to motor fluctuations [8]. When compared to non-depressed PD patients, PD patients with depression experience faster cognitive and motor decline, as well as lower quality of life and greater mortality.

Emotional lability

It is characterised by a sudden and severe shift in mood that can arrive and dissipate fast (mood swings). Generally associated with a deterioration of the frontal cortico-subcortical systems that underpin voluntary control of emotional reactions. Increased sensitivity and even "sentimentality" are terms used to indicate emotional lability. They are the type of persons who cry over stimuli that would be insignificant in another circumstance. Emotional lability in Parkinson's disease can appear as a bipolar disorder in phases, linked with on-off motor oscillations. However, bipolar disorder has been linked to Parkinson's disease, with patients feeling euphoric at times and suicidal at others.

Anhedonia

Anhedonia is characterised by a decrease of pleasure during pleasurable activity. It is usually caused by bilateral basal nuclei lesions [9]. Affective dullness is associated with dysfunction of the anterior cingulate regions, nucleus accumbens, substantia nigra and ventral tegment, as well as the basal telencephalon and is frequently bilateral.

Impulse control disorders

In PD, obsessive-compulsive symptoms such as mannerism, moral rigidity and organised routines have been described. These obsessive-compulsive characteristics may even constitute a premorbid personality several years before the beginning of motor symptoms, allowing EP to be diagnosed. Later, they are linked to levodopa-induced motor fluctuations or "dopaminergic dysregulation" syndrome. There is a behavioural change associated with dopaminergic replacement therapy that can lead to impulse control disorder (TCI) (during abuse or when tolerance is low). They include a loss of critical sense, social skills, indifference to the consequences of their activities, emotion during compulsive acts, loss of self-esteem, guilt, humiliation, social withdrawal, depressive symptoms and paranoid features [10]. This illness is typically characterised by obsessive shopping or hypersexuality.

Emotional communication disorders

The modification of the spontaneous emotional expression of the face is a component of Parkinsonian symptomatology. Similar changes in verbal communication, with a loss of prosody 10 in the foreground, and gestural communication, with akinesia of gestures and postures, have been described. Furthermore, there is little literature on communication fragmentation (comprehension, semantics, syntactics, alexithymia), memory and emotional mental representations, as well as interpreting others' emotions. The perception of emotional prosody in PD is characterized by:

- Ability to explicitly recognize emotional speech tone is diminished.
- Already preattentive processing of emotional prosody is altered.
- Deficits reduce life quality of the patients.
- Depression has to be considered as a possible confound.

Psychosis

Psychotic symptoms are highly related to the necessity for nursing home placement as well as mortality. Visual hallucinations, which are usually benign, affect up to 40% of patients, whereas more serious symptoms, including as delusions, paranoid ideation and delirium, become more common as the disease advances. Psychotic profile disorders have been reported often in Parkinson's disease, primarily after taking dopaminergic or anticholinergic drugs [11]. Hallucinations, illusions, paranoia, capgras syndrome, delusions, but also sexual disorders, melancholy, anxiety, mania, obsessive-compulsive disorders, and sleep disorders (insomnia and nightmares) have been described as psychotic features.

There have been reports of diplopia (visual or mild hallucinations) and illusions in the periphery visual field. These illusions, as well as distinct periods of bewilderment, are illnesses that can be detected in their early stages [12-14]. It is a critical step in adjusting the medication and avoiding a worsening that can occasionally result in hospitalisations in a safe setting. Subjects with isolated minor hallucinations are more likely to acquire more severe hallucinations and are more likely to develop dementia or be admitted to a nursing institution. Atypical antipsychotics, particularly clozapine and quetiapine, are often effective in treating psychotic symptoms while having the least impact on motor symptoms [15].

Conclusion

The neurological grounds for the development of neuropsychiatric problems in Parkinson's disease are becoming more understood. We must not, however, overlook the disease's psychosocial and emotional elements. It should be highlighted that symptoms frequently precede typical motor signs, emphasising the necessity of early detection and treatment. The patient will have to acclimatise to the rhythms imposed on him, particularly the on-off dopaminergic variations, which will be largely dependent on his own administration of an adjusted and personalised medicine combination. Despite the ongoing

insecurity in which he lives, the patient's day must be precisely arranged according to the areas where he knows he is more functional. Furthermore, the oscillations of behaviour, mood and merely motor symptoms exhaust the patients' adaptive resources in the long run. More research is needed to understand the pathogenesis of depression and depressed symptoms in Parkinson's disease patients should be assessed in clinical practise.

References

1. Zhang ZX, Roman GC, Hong Z, Wu CB, Qu QM, et al. (2005) Parkinson's disease in China: Prevalence in Beijing, Xian, and Shanghai. Lancet 365: 595-597.
2. Chaudhuri KR, Healy DG, Schapira AH (2006) Non-motor symptoms of Parkinson's disease: Diagnosis and management. Lancet Neurol 5: 235-245.
3. Chaudhuri KR, Odin P, Antonini A, Martinez-Martin P (2011) Parkinson's disease: The non-motor issues. Parkinsonism Relat Disord 17: 717-723.
4. Zhu J, Lu L, Pan Y, Shen B, Xu S, et al. Depression and associated factors in nondemented Chinese patients with Parkinson's disease. Clin Neurol Neurosurg 163: 142-14 8.
5. Aarsland D, Marsh L, Schrag A (2009) Neuropsychiatric symptoms in Parkinson's disease. Mov Disord 24: 2175-2186.
6. Hely MA, Morris JG, Reid WG, Trafficante R (2005) Sydney multicenter study of Parkinson's disease: Non-L-dopa-responsive problems dominate at 15 years. Mov Disord 20: 190-199.
7. Aarsland D, Zaccai J, Brayne C (2005) A systematic review of prevalence studies of dementia in Parkinson's disease. Mov Disord 20: 1255-1263.
8. Williams-Gray CH, Evans JR, Goris A, Foltynie T, Ban M, et al. (2009) The distinct cognitive syndromes of Parkinson's disease: 5 year follow-up of the CamPaIGN cohort. Brain 132: 2958-2969.
9. Broeders M, de Bie RM, Velseboer DC, Speelman JD, Muslimovic D, et al. (2013) Evolution of mild cognitive impairment in Parkinson disease. Neurology 81: 346-352.
10. Weintraub D, Moberg PJ, Culbertson WC, Duda JE, Stern MB (2004) Evidence for impaired encoding and retrieval memory profiles in Parkinson disease. Cogn Behav Neurol 17: 195-200.
11. Dubois B, Pillon B (1996) Cognitive deficits in Parkinson's disease. J Neurol 244: 2-8.
12. Lewis SJ, Cools R, Robbins TW, Dove A, Barker RA, et al. Using executive heterogeneity to explore the nature of working memory deficits in Parkinson's disease. Neuropsychologia 41: 645-654.
13. Yarnall AJ, Breen DP, Duncan GW, Khoo TK, Coleman SY, et al. (2014) Characterizing mild cognitive impairment in incident Parkinson disease: The ICICLE-PD study. Neurol J 82: 308-316.
14. Litvan I, Aarsland D, Adler CH, Goldman JG, Kulisevsky J (2011) MDS Task Force on mild cognitive impairment in Parkinson's disease: Critical review of PD-MCI. Mov Disord 26: 1814-1824.
15. Goldman JG, Holden S, Bernard B, Ouyang B, Goetz CG, et al. (2013) Defining optimal cutoff scores for cognitive impairment using movement disorder society task force criteria for mild cognitive impairment in Parkinson's disease. Mov Disord 28: 1972-1979.