Original Article

Neuropsychiatric Disorders and Sleep Disturbances in Patients with Parkinson's Disease

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ABSTRACT

Background: Patients with Parkinson's disease (PD) have wide range of neuro-psychiatric symptoms and sleep disturbances as well as the motor manifestation of the disease. Several non-motor features of PD have attracted attention of physicians as these features have been correlated with the quality of life of the patients. **Objective:** is to evaluate the extent of neuropsychiatric disorders and sleep disturbances in patients suffering from PD. **Methods:** This study include 30 patients suffering from PD (13 males and 17 females), compared with 20 healthy subjects age and sex matched as a control group. Patients were diagnosed as having PD using Parkinson's Disease Society Brain Bank Diagnostic Criteria (PDSBBDC). All patients were subjected to full history taking and neurological examination, MMSE (Mini-mental state examination), Semi-Structured clinical interview (SCID-1) based on DSM-IV, and sleep questionnaire of Asaad and Kahla. **Results:** The most common psychiatric disorders in PD patients were depression (60%), anxiety 43.3%, and psychosis in 20%. The difference between patients and control groups was significant as regarding depression but not significant for others. The most common sleep disorders were nocturia (40%), sleep maintenance problem (26.7%), and bad quality of sleep (23.3%). All sleep disorders were higher in patients than in control group. Different types of psychiatric disorders sleep disorders were higher in L- Dopa receivers than in L- Dopa non-receivers. **Conclusion:** There were significant neuropsychiatric and sleep disturbances in patients with Parkinson's disease which were higher in L-Dopa receivers than in L- Dopa non-receivers. **(*Egypt J Neurol Psychiat Neurosurg*. 2010; 47(2): 317-324)**

Key Words: Parkinson's, neuropsychiatric, sleep.

INTRODUCTION

There are several non-motor aspects of the Parkinson's disease (PD) that are of at least equal importance in the management of patients with Parkinson's disease. They include depression, cognitive impairment, anxiety, psychosis, autonomic disturbance and sleep disturbances among others. In addition to the disease duration and severity, other factors including drugs may contribute to their occurrence. Pathogenesis of these aspects is not fully understood, though there has been a significant increase in the knowledge in recent years[1].

Being the most frequent psychiatric disorder in patients with PD, the average prevalence of depression in the patients of PD was estimated to be as high as 40% to 50%[2].

Up to 40% of patients with PD suffer from clinically significant anxiety; this is higher than expected for this age group of patients. Anxiety may cause a significant deterioration of parkinsonian symptom[3]. Delusions and hallucinations occur in patients with PD with reported prevalence ranging from 6% to 40%. Such symptoms are associated with major behavioral and functional problems. For patients and their families, delusions and hallucinations can decrease the quality of life and increase the burden of the illness[4].

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Sleep disturbances are a common complaint among patients with idiopathic PD. People with Parkinson's disease suffer from insomnia, excessive daytime sleepiness, sleep attacks, nightmares, REM sleep behavior disorders, periodic limb movement in sleep, restless leg syndrome and sleep apnea syndrome[5].

Depression, dementia, and physiologic changes contribute to the high prevalence of sleep disturbances in patients with PD. Antiparkinsonian drugs also play a role in insomnia by increasing daytime sleepiness[6].

The aim of the present work is to explore the range of neuron-psychiatric disorders and various patterns of sleep disturbances in patients with PD without dementia and to evaluate the relation between these disorders and the demographic and clinical variables.

SUBJECTS AND METHODS

Subjects:

This study included 30 consented patients suffering from PD selected from patients attending outpatient clinic and in the neurology department at Minoufiya University Hospital, Egypt between April and December 2007. Patients were 13 males and 17 females with age range between 50 - 80 years with a mean age of 61.7±7.1. They were compared with 20 healthy consented subjects (8 males and 12 females)

with age range between 52-80 years with a mean age of 63.45±7.83.

Exclusion criteria:

1. Patients with atypical features e.g.: pyramidal manifestations, cerebellar signs, prominent autonomic dysfunction.
2. Patients with dementia using Mini Mental State Examination (MMSE) as a screening test (patient group should be at least ≥ 24).
3. Patients on medications influencing sleep as: benzodiazepines, barbiturates, etc...
4. History of other causes of Parkinsonism including toxic exposure, head injury, encephalitis, cerebrovascular disease, etc….
5. Metabolic causes as myxedema and hepatic or renal impairment.

Methods:

The following assessments were done to our subjects:

1- Complete history taking and neurological examination.
2- Patients diagnosed as having Parkinson's disease using Parkinson's Disease Society Brain Bank Diagnostic Criteria (PDSBBDC) [7].
3- Mini-mental state examination to exclude dementia.
4- Sleep questionnaire of Asaad and Kahla[8]: Sleep history was taken from all patients through the structured sheet for sleep disorders[9], which consist of 72 questions regarding the following:
   a. Personal sleep rituals (e.g. time to go to bed, sleep hours, and sleep naps), or other habits affecting sleep.
   b. Past or present history of sleep disturbance and medications used.
   c. Drug history for psychiatric or other physical disorders.
   d. Sleep disorders which are insomnia, hypersomnia, parasomnias, or dyssomnias.
   e. Effect of sleep disturbance on personal functioning.
5- Semi-structured clinical interview for DSM IV (SCID):- The structured clinical interview for DSM-IV axis I disorder (SCID-I) is a semi structured interview for making the major DSM-IV axis I diagnosis. Structured interview have been developed to increase diagnostic reliability through standardization of the assessment process and to increase diagnostic validity by facilitating the application of the DSM-IV diagnostic criteria and by systematically probing for symptoms that might otherwise be overlooked.
6- Brain computerized tomography (CT) or Magnetic resonance imaging (MRI) if possible.
7- Routine laboratory investigations to exclude other non primary Parkinsonism.

History of L- dopa administration in PD patients:

Patients were classified according to whether they were on L-dopa treatment or not into two groups:

* Group (A): It included 20 patients (66.7%) who were receiving L-dopa. The dose of L-dopa ranged from 250 to 750 mg/day with a mean of 443.8±148.9 mg/day and the duration of L-dopa treatment ranged from 1 to 10 years with a mean of 3.2±1.9.
* Group (B): It included 10 patients (33.3%). who were not receiving L-dopa.

Statistical Analysis

Data was collected, tabulated and analyzed by SPSS version 11.0 statistical package *(SPSS Inc. Chicago, Illinois, USA).* Quantitative data expressed as mean and standard deviation (X±SD). Student t-test was used to compare two groups of normally distributed variables and Mann-Whitney (U – test) for non – normally distributed variables. Qualitative data expressed as number and percentage and analyzed by Chi-square test with or without Yale's Continuity Correction when appropriate. Level of significance was set as P value < 0.05.

RESULTS

The main neurological manifestations in PD group showed that bradykinesia was present in all cases 100%, while tremors were present in 96.6%, and rigidity in 66.6%. Other manifestations as speech disorders (63.3%), postural instability (63.3%), and freezing (40% were also reported (Table 1).

Psychiatric manifestations in PD and control groups showed that all psychiatric disturbances were higher in PD group than control group; although it was significant only as regard depression (P<0.05) (Table 2).

Different types of psychiatric disorders were higher in L-dopa receivers than in L-dopa non receivers. There was highly significant difference (P<0.001) between two groups as regard major depression and significant difference (P<0.05) regarding dysthymia, but not significant for others (Table 3).

All sleep disorders were higher in PD patients (60%) when compared with control group. The difference between two groups was significant as regarding nocturia, sleep initiation problem, nocturnal restlessness and motor symptoms (tremor, cramps, dystonia) (P<0.05), but not significant for others (Table 4).

Higher sleep disorders were recorded in L-dopa receivers than in L-dopa non receivers. The difference between two groups was significant as regarding sleep initiation problem, nocturnal restlessness, nocturia and motor symptoms (tremor, cramps, dystonia) (P<0.05) but not significant for others (Table 5).

Significant difference was found between motor symptoms and psychiatric disturbances regarding depression (P<0.05), but it was not significant for anxiety and psychosis (Table 6).

Table 1. Neurological manifestations in Parkinsonian patients.

Neurological manifestations	Number (30)	Percentage
Main manifestation :		
Bradykinesia	30	100.0
Tremors	29	96.6
Rigidity	20	66.66
Others:		
Speech disorders	19	63.3
Postural instability	19	63.3
Freezing	12	40.0
Parathesia	6	20.0
Impotence	6(of 13 male)	(46.1)
Pain	4	13.3
Postural hypotension	4	13.3
Dyskinesia	3	10.0

Table 2. Distribution of psychiatric manifestations in PD patients (according to SCID diagnosis).

Psychiatric disturbances	Studied groups				χ^2	P-value
	PD patients		Controls			
	Number (30)	Percentage	Number (20)	Percentage		
Depression	18	60.0	4	20.0	4.8	< 0.05*
Major depressive	3	16.7	0	00.0	0.03	> 0.05
Dysthymic	15	83.3	4	100		
Anxiety	13	43.3	4	20.0		
Generalized anxiety	8	61.5	2	50.0	1.7	> 0.05
Social phobia	3	23.1	2	50.0	0.8	> 0.05
Panic disorder	2	15.4	0	0.0		
Mixed (anxiety and depression)	8	61.5	0	0	6.35	> 0.05
Psychotic features	6	20.0	0	0.0	2.4	> 0.05

PD Parkinson's disease, χ^2 chi square
* Statistically significant at p<0.05

Table 3. Distribution of psychiatric disorders in parkinsonian L-Dopa receivers and non-receivers (according to SCID diagnosis).

	Parkinsonian patients				χ^2	P-value
	Dopa receivers		Dopa non-receivers			
	Number	Percentage	Number	Percentage		
Depression	15	75.0	3	30.0	5.6	< 0.01**
Major depressive	1	6.7	2	66.7	2.9	< 0.05*
Dysthymic	14	93.3	1	33.3		
Anxiety	8	40.0	5	50.0	0.27	> 0.05
Generalized anxiety	5	62.5	3	60.0		
Social phobia	2	25.0	1	20.0	0.15	> 0.05
Panic disorder	1	2.5	1	20.0		
Psychotic features	4	20.0	2	20.0	0.0	> 0.05

χ^2 chi square
* Statistically significant at p<0.05 ** Statistically significant at p<0.01

Table 4. Distribution of sleep disorders among the PD patients and controls.

	Studied groups				χ^2	P value
	PD patients		Controls			
	Number (30)	Percentage	Number (20)	Percentage		
Bad Quality of sleep	7	23.3	4	20.0	0.05	> 0.05
Sleep initiation problem	6	20.0	0	0.0	2.90	< 0.05*
Sleep maintenance problem	8	26.7	8	40.0	0.64	> 0.05
Nocturnal restlessness	6	20.0	0	0.0	2.90	< 0.05*
Vivid dreams& hallucinations	4	13.3	0	0.0	1.50	> 0.05
Nocturia	12	40.0	4	20.0	2.60	< 0.05*
Motor symptoms during sleep	6	20.0	0	0.0	2.90	< 0.05*
Excessive daytime somnolence	5	16.7	2	10.0	0.26	> 0.05

PD Parkinson's disease, χ^2 chi square
* Statistically significant at p<0.05

Table 5. Distribution of sleep disorders among Parkinsonian L-dopa receivers and non-receivers.

	Parkinsonian patients				χ^2	P-value
	Dopa receivers		Dopa non-receivers			
	Number (20)	Percentag	Number (10)	Percentage		
Bad Quality of sleep	6	30.0	1	10.0	1.5	> 0.05
Sleep initiation problem	6	30.0	0	0.0	3.8	< 0.05*
Sleep maintenance problem:	7	35.0	1	10.0	2.1	> 0.05
Nocturnal restlessness	6	30.0	0	0.0	3.8	< 0.05*
Vivid dreams&hallucinations	4	20.0	0	0.0	2.3	> 0.05
Nocturia	12	60.0	0	0.0	10.0	< 0.05*
Motor symptoms during sleep:	6	30.0	0	0.0	3.8	< 0.05*
Excessive day-time somnolence	5	25.0	0	0.0	3.0	> 0.05
Total	20	100.0	10	100.0		----

PD Parkinson's disease, χ^2 chi square
* Statistically significant.

Table 6. Comparison between motor symptoms and psychiatric disturbances.

	Bradykinesia		Rigidity		Tremors	
	Number (30)	100%	Number (18)	66.6%	Number (29)	96.6%
Depression						
Present	18	60.0	13	72.2	18	62.06
Absent	12	40.0	5	27.7	11	
X^2	-		2.8		2.6	
P- Value	-		<0.05*		<0.05*	
Anxiety						
Present	13	43.3	7	38.8	13	44.8
Absent	17	56.6	11	61.1	16	55.2
X^2	-		0.36		0.79	
P- Value	-		>0.05		>0.05	
Psychosis						
Present	6	20	5	27.7	6	20.6
Absent	24	80	13	72.2	23	79.4
X^2	-		1.7		0.26	
P- Value	-		>0.05		>0.05	

χ^2 chi square
* Statistically significant at p<0.05

DISCUSSION

Several non-motor features of PD have attracted attention of physicians. These aspects are at least equally important to the motor aspects as regards to the overall morbidity of PD and the quality of life of the patient[9].

These aspects are common and have been strongly correlated with the quality of life of patients and their careers. They may precede motor symptoms and can be missed unless looked for. They may require the use of special scales for their detection. They are often multifactorial in etiology that is, disease duration and severity, drugs, and age all can contribute to their causation. Treatment of these aspects is often very difficult, but when successful, it can be very rewarding[10].

This study showed that depression is the most common psychiatric symptom (60%). This percentage is slightly higher than those reported by Ring and Serra-Mestres[11]and Tazi et al.[12] (50%) for both; and higher than those reported by Aarsland et al.[3]; (39%). This variation in results regarding may be attributed to the use of different diagnostic criteria and screening tools for depression, and also due to the clinical overlap between signs and symptoms of depression and some of those of PD manifisttions.

Depression in PD is usually linked to a reduction in brain catecholamines, serotonin (a decrease in the concentration of 5-hydroxy-indoleacetic acid in cerebrospinal fluid was reported), or dopamine (postmortem studies show dopamine depletion in the ventral tegmental area) [13].

A significant correlation was found between depressive symptoms and the degree of motor disability. This result is in contrast to several studies as those reported by Starkstein et al.[14] and Attain et al., [15]. These studies suggest that the mood changes in PD are not a reaction to motor disability but rather reflect a neurobiological based disorder of emotional dysfunctions. However, other studies do suggest such relation between both variables and attribute this relation to more pronounced changes in cerebral neurotransmitters (i.e. endogenous origin), or patient's response to their limited mobility and isolation in later stages of the disease (i.e. reactive origin) Slawek et al.[16].

In this study, Slight increase in the frequency of depressed patients after levodopa therapy might suggest the possibility that patients under levodopa therapy were more likely to experience depression. This agrees with the research of Daniel et al.[17] who reported an association between depression and higher levodopa.

This result not agree with Marsh and Lang,[18] who conducted a prospective study to evaluate depression in PD patients before and after 15 months of levodopa therapy, and reported that levodopa therapy did not alter Parkinsonian depression. Kosti et al.[19] found that PD with depression was also associated with longer duration of the disease, higher doses of L-dopa equivalents, patient's age, general impairment of daily living.

In this study, anxiety is reported in about (43%) of PD patients. Similar percentage was reported by Kelly et al.[1] and Michael et al.[2]. Most of these patients (61.5%) met the criteria of generalized anxiety disorder, 15.4% met the criteria of panic disorder and 23.1% of them met the criteria of social phobia. These percent go hand in hand with those reported by Rijk and Bijl[20] regarding generalized anxiety disorder 24.5% and social phobia 11.5%. , but they are slightly higher than those results regarding panic disorder 7.9%. This variation in the results may be attributed to the use of different diagnostic criteria and screening tools.

Several neurotransmitters have been implicated in the pathogenesis of generalized anxiety disorder in patients of PD. These include norepinephrine, serotonin, δ-aminobutyric acid (GABA), and dopamine. Anxiety in PD patients could involve a dopaminergic deficit directly or could be due to interactions between dopaminergic deficits and the variable deficits in norepinephrine and serotonin that are known to occur in PD. Significant changes in anxiety levels may accompany the "on-off" symptoms in patients with fluctuating PD. Most patients with "on-off" fluctuations experience greater anxiety during the off phase (when they are slower and more rigid[3].

Psychotic disorders were present in (20%) of the patient sample, most of them were visual. This percentage came in the same range reported by Holroyd et al.[21] who examined one hundred and two patients and found 29.4% of them having hallucinations or delusions. Twenty percent of L-Dopa receivers and L-Dopa non receivers had psychosis in the form of delusions and visual hallucination. So, these results deny any role for L-Dopa in the pathogenesis of any of the psychotic symptoms found in PD patients.

This agrees with other recently reported results of Holroyd et al. [21] who concluded that current L-Dopa use, total L-Dopa dose, duration of L-Dopa use, were not associated with hallucinations. They suggested that hallucinations in PD are not simply a medication effect, but a symptom related to the disease itself, although one that might be worsened by medication.

However, this goes against the common view that suggests a strong role of L-Dopa in the pathogenesis of psychotic symptoms in PD patients[22]. On the other hand Barnes and David[23] suggested that hallucinations might be explained by some unknown mechanism of interaction between dopaminergic drugs and PD to produce hallucinations.

In this study, sleep disturbances were found in about 60% of PD patients. This is slightly lower than what had been reported by other authors as Garcia Borreguero et al.[24] and Michael and Thorpy[25] who

found that sleep complaints were found in about 70% for both; and similar to that reported by Stocchi et al.[26] (about 60%).

In this study, overall sleep complaints occur more often in PD patients than in an age-matched elderly population. Previous studies found similar results. In particular, sleep fragmentation, due to impaired motor function, nocturia, altered dreaming, nocturnal vocalization and daytime hallucinations occurred more frequently in the PD group, suggesting that these symptoms were disease-related. It has also been suggested that pain and depression might be concurrent important factors resulting in sleep disruption in PD patients (Tandberg et al.[27], van Hilten et al.[28]).
However, Charles et al.[29] found in their study that many factors have been implicated to cause such complaints as: nocturnal motor dysfunctions (nocturnal akathesia and periodic limb movement), sleep apnea, anxiety disorders, REM sleep behavior disorder and the effects of medication.

This study showed that the most frequent cause of sleep disturbance was recurrent awakenings for urination (nocturia) 40%. This is in accordance with a community based surveys of 220 PD patients in UK where the commonest nocturnal symptom was nocturia in 60%[30], and with an Indian Hospital based study on sleep disturbances in PD patients, where the most common complaint was again nocturia (70.4%) and this was attributed to incomplete bladder emptying, which, in turn, probably reflects a high incidence of autonomic dysfunction in PD[31].

These results were close to those reported by Wetter et al.[32] who found that the main complaints were sleep fragmentation, probably due to an inability to turn over in the bed and nocturia. Urinary frequency may also occur as the dose of dopaminergic medication wears off, and therefore a change to a longer acting form of medication at night may be required [33].

In this study there was a statistically significant difference between the PD patients on medication and those not receiving L-dopa treatment in most of items, suggesting that sleep disturbances in PD patients is related to its pathological process as well as drug related factors.
These results were rather in contrast to those reported by Arnulf et al.[34] who found no correlation between sleepiness and dopamine agonist or levodopa-equivalent daily doses. The authors stated that absence of dose-related sleepiness is a strong argument against an effect of this class of drugs on the mechanisms of sleepiness and suggested that individual characteristics of the patients predominate.

This difference between our results and others could be due to in our patient the duration and severity of the disease that is more in L-Dopa receivers than non-receivers and due to methodological differences. This view is in agreement with Happe et al.[35] who found that the disease severity is strongly associated with sleep disorders in PD patients.

These findings may suggest that the pathologic process itself is a significant factor in causing disordered sleep in PD. In addition to striatal and mesencephalic dopaminergic depletion, serotonergic neurons of the dorsal raphe, noradrenergic neurons of the locus coeruleus, and cholinergic neurons of the pedunculopontine nucleus are also affected. Each of these neuronal populations is involved in control of the sleep-wake cycle[36].

However the underlying causes for sleep disorders in PD patients are still discussed controversially. It may be due the disease itself with its underlying immobility, the impact of dopaminergic medication or due to a concomitant depression[35].

In conclusion, several non-motor aspects of PD that is of at least equal importance in the management of these patients. They include wide range of psychiatric symptoms including depression, anxiety, delusions and hallucinations. Sleep disturbances are also common in PD. Causes of sleep disorders in PD is multifactorial. It is partially related to the disease and its symptoms, and to the treatment used.

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الملخص العربى

الاضطرابات العصبية والنفسية واضطرابات النوم في مرضى الشلل الرعاش

يعتبر مرض بار كينسون من أهم وأشهر الأمراض الحركية التي تصيب كبار السن ويعاني المرضى المصابون بمرض بار كينسون من العديد من الأعراض الحركية مثل الرعشة وبطء الحركة كما يعانون من العديد من الأعراض النفسية وكذلك اضطرا بات النوم.

لذا فقد استهدف البحث دراسة الأنواع المختلفة من اضطرابات النوم وكذلك دراسة الاضطرابات النفسية عند المصابين بهذا المرض . ولقد أجريت هذه الدراســة على ثلاثين مريضاً بمرض بار كينسون وتم تقسيمهم إلى مجموعتين (مجموعه تتلقى العلاج بعقار الليفودوبا وأخرى لا). وتمت مقارنتهم بمجموعة ضابطة من عشرين فردا من الأصحاء من عمر وجنس متقارب، ولقد أجريت لهم جميعاً الفحوصات والأبحاث التالية :

- ✓ استبيان لأخذ بيانات المرضى وتقييم إكلينيكي و فحص كامل للجهاز العصبى .
- ✓ أشعة مقطعية على المخ بالكمبيوتر أو أشعة بالرنين المغناطيسي على المخ إن أمكن.
- ✓ استبيان خاص بالنوم.
- ✓ الفحص المختصر للحالة العقلية MMSE.
- ✓ مقياس (SCID-1) لتقييم الأعراض النفسية.

ولقد تلخصت نتائج البحث فيما يلي :

جاءت مجموعتا المرضى متوازيتين مع بعضهما البعض ومع المجموعة الضا بطة من حيث السن والجنس. وقد وجد أن هناك أعراض نفسية يعاني منها المرضى بمرض باركينسون بنسبه أكبر من المجموعه الضابطة، وقد جاء الاكتئاب في المقدمة باعتباره أكثر الأعراض النفسية شيوعاً بنسبة 60%، وجاءت أعراض القلق في المرتبة الثانية بنسبة 43.3% ثم الهلاوس والضلالات بنسبة 20%. ولوحظت هذه الأعراض بنسبة أكبر في المرضى الذين يتلقون العلاج بعقار الليفودوبا منها في أولئك الذين لا يتلقون هذا العلاج، وقد وجد علاقة طرد ية بين الاكتئاب والمرض نفسه وأيضاً العلاج بعقار الليفودوبا.

وقد أظهرت الدراسةأيضا أن اضطرابات النوم كانت أكثر في المرضى عن المجموعه الضابطه وأيضاً أولئك الذين يتلقون العلاج بعقار الليفودوبا من أولئك الذين لا يتلقون العلاج. وقد تمثلت اضطرابات النوم في زيادة التبول الليلي، مشكلة الدخول في النوم، مشكلة الاستيقاظ مرات عديدة أثناء النوم ليلاً، النعاس كثيراً بالنهار، كثرة الحركة أثناء النوم، رؤية أحلام واضحة وهلاوس أثناء النوم.