European Journal of Molecular & Clinical Medicine

ISSN 2515-8260 Volume 08, Issue 03, 2021

Assessment of association between glycemic status and Parkinson disease

Gary Batra[1], Sourav Bansal[2], Jugraj Singh[3], Arshdeep Singh Samra[4]

[1,3,4]MBBS Intern, PIMS Jalandhar, Punjab, India;
[2]MBBS Intern, Govt. Medical College Amritsar, Punjab, India

Corresponding author: Dr. Sourav Bansal, MBBS Intern, GMC Amritsar, Punjab, India

ABSTRACT:

Background: Parkinson disease (PD) is also a major chronic disease, and its clinical significance is increasing worldwide. The present study was conducted to assess association between glycemic status and clinical stage of PD.

Materials & Methods: 89 patients of Parkinson disease (PD) of both genders were included. Sleep quality was assessed using Parkinson's disease sleep scale (PDSS). Presence of depression was assessed using Hamilton Depression (HAM-D) Rating Scale. Presence of motor and autonomic symptoms were recorded.

Results: out of 89, males were 49 and females were 40. Autonomic dysfunction was constipation in 42, urinary symptoms in 34, sexual dysfunction in 28 and postural giddiness in 53 patients. Non- motor symptoms was sleep disorder in 23, depression in 38, pain in 21, olfactory dysfunction in 45 and cognitive dysfunction in 61. Glycated hemoglobin <5.7% was seen in 18, 5.7- 6.4 % in 23 and >6.5% in 48. The difference was significant (P< 0.05).

Conclusion: Poor glycemic control was found in most of patients with PD.

Key words: Glycemic control, Parkinson disease, Cognitive dysfunction

Introduction

The prevalence of diabetes and related complications is increasing worldwide. Accordingly, the burden on global health care related to diabetes continues to increase. Meanwhile, various therapeutic interventions for diabetes have been developed, and the clinical course and quality of life of people with diabetes have improved.[1] However, it remains impossible to completely prevent the development of diabetes-related complications. Rather, the clinical significance of previously overlooked atypical complications has paradoxically increased.[2]

Parkinson disease (PD) is also a major chronic disease, and its clinical significance is increasing worldwide.[3] Damage to the dopaminergic neurons of the substantia nigra is known to be a major cause of PD, which is clinically characterized by a variety of neurologic symptoms. The prevalence of PD is expected to continue to increase as human life expectancy increases. However, curative treatment for this disease does not currently exist, and only symptomatic management is performed. Thus, the disease burden for PD is also expected to increase in the future.[4]

Various risk factors are being studied and postulated in PD. Gender, environmental factors such as rural living and pesticide exposure has been found as important risk factors in the previous studies. Clinical data on the risk of developing new diabetes in relation to Parkinson's disease are limited. Chronic systemic inflammation and mitochondrial dysfunction are common to pathogenesis of both diabetes and PD and the same being postulated as one reason for substantiating diabetes as a risk factor for PD.[5] The present study was conducted to assess association between glycemic status and clinical stage of PD.

Materials & Methods

The present study comprised of 89 patients of Parkinson disease (PD) of both genders. The diagnosis of PD was based on presence of bradykinesia and muscular rigidity, 4-6 Hz rest tremor, postural instability not caused by primary visual, vestibular, cerebellar, or proprioceptive dysfunction. All were taken in the study after their parent gave written consent.

Data such as name, age, gender etc. was recorded. A thorough clinical examination was performed. Sleep quality was assessed using Parkinson's disease sleep scale (PDSS). Presence of depression was assessed using Hamilton Depression (HAM-D) Rating Scale. Presence of motor and autonomic symptoms were recorded. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

Results

Table I Distribution of patients

Total- 89		
Gender	**Males**	**Females**
Number	49	40

Table I shows that out of 89, males were 49 and females were 40.

Table II Assessment of autonomic dysfunction

Autonomic dysfunction	**Number**	**P value**
Constipation	42	0.12
Urinary symptoms	34	
Sexual dysfunction	28	
Postural giddiness	53	

Table II, graph I shows that autonomic dysfunction was constipation in 42, urinary symptoms in 34, sexual dysfunction in 28 and postural giddiness in 53 patients. The difference was non- significant (P> 0.05).

Graph I Assessment of autonomic dysfunction

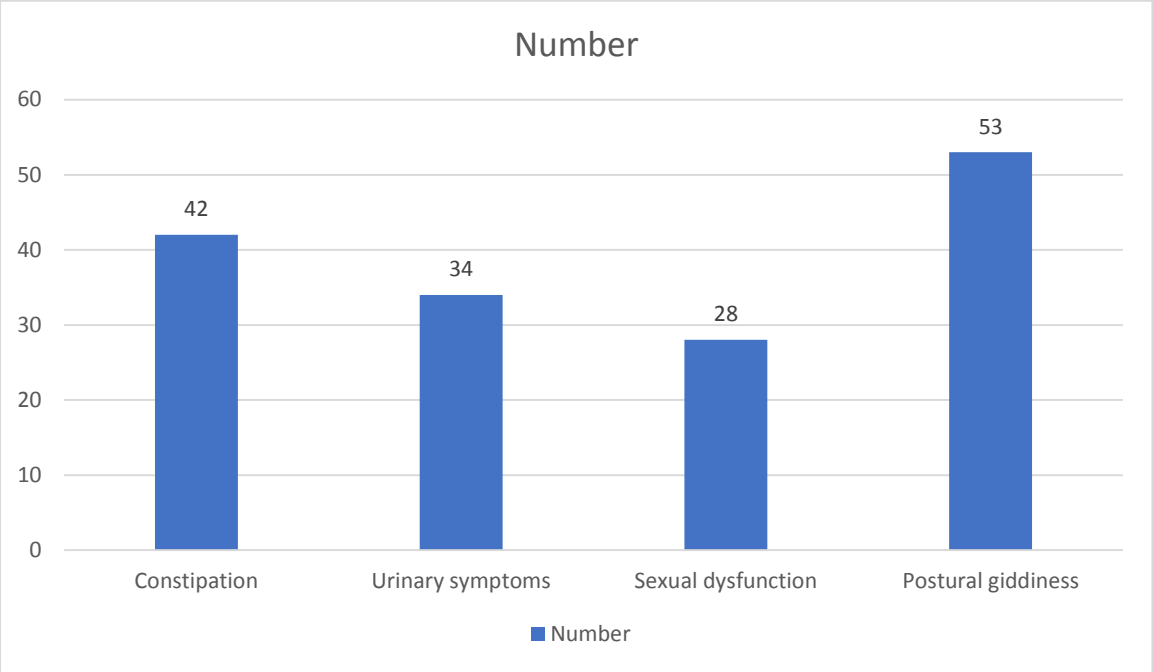

Table III Presence of non- motor symptoms

Non- motor symptoms	Number	P value
Sleep disorder	23	0.04
Depression	38	
Pain	21	
Olfactory dysfunction	45	
Cognitive dysfunction	61	

Table III shows that non- motor symptoms was sleep disorder in 23, depression in 38, pain in 21, olfactory dysfunction in 45 and cognitive dysfunction in 61. The difference was significant (P< 0.05).

Table IV Glycemic status and PD

Glycemic status (%)	Number	P value
<5.7 (Normal)	18	0.01
5.7- 6.4 (Pre- diabetic)	23	
>6.5 (Diabetic)	48	

Table IV shows that glycated hemoglobin <5.7% was seen in 18, 5.7- 6.4 % in 23 and >6.5% in 48. The difference was significant (P< 0.05).

Discussion

Various environmental and genetic factors are known to increase the risk of PD. In particular, recent reports have suggested that metabolic diseases, such as obesity, metabolic syndrome,

and diabetes, are important risk factors for PD.[6]This is because the pathophysiologic mechanism associated with insulin resistance plays an important role in the development and worsening of PD as well as diabetes.[7] However, there have been conflicting results from previous epidemiologic studies on the association between diabetes and PD. Moreover, any causal relationship between diabetes and PD needs to be more clearly defined.[8]

Both Parkinson's disease and diabetes are chronic illnesses which needs longterm medical care and most often result in significant morbidity.[9] Some of the past literature suggested that diabetes mellitus has been linked with PD, and they have in common similar pathogenic pathways. Contact to environmental factors and genetic susceptibility play an important role in the etiology and evolution of both diabetes mellitus and PD. It is also previously documented that insulin and dopamine may exert reciprocal regulation between PD and diabetes.[10] The present study was conducted to assess association between glycemic status and clinical stage of PD.

In present study, out of 89, males were 49 and females were 40. ELangowan et al[11] in their study consecutive patients with a diagnosis of idiopathic parkinson's disease (PD) satisfying UK Parkinson's disease society brain bank clinical diagnostic criteria attending the department of neurology during the period January 2019 to December 2019 were included. Total of 70 patients were enrolled in the study. Tremor was the most common motor symptom. 53.3% of subjects had one or the other non motor symptoms. Diabetes was present in 38.6% of subjects. Mean FBS among the cases was 112. 36% of the subjects had poor control of diabetes based on their HbA1C values (HbA1C >7). There was statistically significant relation between glycemic control and clinical stage of PD.

We found that autonomic dysfunction was constipation in 42, urinary symptoms in 34, sexual dysfunction in 28 and postural giddiness in 53 patients. Lin Lu et al[12] they found that diabetic individuals may have a reduced incidence of PD despite significant heterogeneity. Various case control studies had contrasting finding on association of diabetes and PD.

We observed that non- motor symptoms was sleep disorder in 23, depression in 38, pain in 21, olfactory dysfunction in 45 and cognitive dysfunction in 61. A study by Xiaoyan Guo et al[13] in Chinese population found lower levels of total cholesterol, LDL-C and TG than controls, they also found high levels of total cholesterol and LDL-C may be associated with low prevalence of PD.

We found that glycated hemoglobin <5.7% was seen in 18, 5.7- 6.4 % in 23 and >6.5% in 48. Rhee et al[14] subjects were classified into the following groups: no diabetes, impaired fasting glucose (IFG), diabetes duration <5 years, and diabetes duration ≥5 years. They analyzed the adjusted hazard ratio of PD for each group. PD occurred in 31,577 patients. Compared with the non- diabetes group, the adjusted hazard ratio was 1.038 (95% CI, 1.009–1.067) in the IFG group, 1.185 (95% CI, 1.143–1.229) in the diabetes duration <5 years group, and 1.618 (95% CI, 1.566–1.672) in the diabetes duration ≥5 years group. These results were consistent with those of the subgroup analysis, and the presence of diabetes further increased the risk of PD regardless of comorbidities such as cardiovascular, cerebrovascular, and chronic kidney diseases.

Conclusion

Authors found that poor glycemic control was found in most of patients with PD.

References

1. Craft S, Watson GS. Insulin and neurodegenerative disease: shared and specific mechanisms. Lancet Neurol 2004;3:169–178.
2. Ravan A, Ahmad F, Chabria S, Gadhari M, Sankhla C. Non-motor symptoms in an Indian cohort of Parkinson's disease patients and correlation of progression of non-motor symptoms with motor worsening. Neurology India. 2015;63:166–74.
3. Behari M, Srivastava AK, Das RR, Pandey RM J Neurol Sci. 2001;190:49-55.
4. Priyadarshi A, Khuder SA, Schaub EA, Priyadarshi SS Environ Res. 2001;86:122-7.
5. Seidler A, Hellenbrand W, Robra BP, Vieregge P, Nischan P, Joerg J, Oertel WH, Ulm G, Schneider E. Possible Environmental, Occupational, and Other Etiologic Factors for Parkinson's Disease: A Case-Control Study in Germany. Neurology. 1996; 46:1275- 84.
6. Nanditha A., Snehalatha C., Satheesh K., Susairaj P., Simon M., Vijaya L., Raghavan A et al. Secular trends in diabetes in India (STRIDE–I): Change in prevalence in 10 years among urban and rural populations in Tamil Nadu. Diabetes Care 2019;42:476-485.
7. Comella CL, Nardine TM, Diederich NJ, Stebbins GT. Sleep-related violence, injury, and REM sleep behavior disorder in Parkinsons disease. Neurology 1998; 51: 526–529.
8. Gibb WR, Lees AJ. The relevance of the Lewy body to the pathogenesis of idiopathic Parkinson's disease. J Neurol Neurosurg Psychiatry 1988;51:745–752.
9. Park JH, Kim DH, Park YG, et al. Cancer risk in patients with Parkinson's disease in South Korea: a nationwide, population-based cohort study. Eur J Cancer 2019;117:5–13.
10. Nam GE, Kim NH, Han K, et al. Chronic renal dysfunction, proteinuria, and risk of Parkinson's disease in the elderly. Mov Disord 2019;34:1184–1191.
11. Elangovan, Shameer Palliyali, Jithin Antony Bose, Jayasankar VR. A study on diabetes and parkinson's disease in a tertiary care centre at central Tamilnadu. International Journal of Contemporary Medical Research 2020;7(6):1-4.
12. Lu L, Fu D-l, Li H-q, Liu A-j, Li J-h, et al. Diabetes and Risk of Parkinson's Disease: An Updated Meta- Analysis of Case-Control Studies. PLoS one 2014;9: 857-81.
13. Santiago JA, Potashkin JA. Shared dysregulated pathways lead to Parkinson's disease and diabetes. Trends Mol Med 2013;19:176–186.
14. Rhee SY, Han KD, Kwon H, Park SE, Park YG, Kim YH, Yoo SJ, Rhee EJ, Lee WY. Association Between Glycemic Status and the Risk of Parkinson Disease: A Nationwide Population-Based Study. Diabetes Care. 2020 Sep 1;43(9):2169-75.