Clinical features of metabolic syndrome in patients with Parkinson's disease

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Introduction. Focus on the metabolic causes underlying dopaminergic cell loss in Parkinson's disease (PD) has increased lately. Glucose imbalances have been shown to be present in patients with PD. A syndrome characterized principally by insulin resistance and glucose dysregulations is metabolic syndrome. Scarce literature has evaluated the relation between these two diseases.

Aim. To determine the prevalence and clinical features of metabolic syndrome and its components in patients with PD.

Patients and methods. We analyzed data from 99 patients with PD diagnosis. Scales that evaluate motor, non-motor, and cognitive function, as well as sleep disorders and quality of life were registered. Metabolic syndrome was diagnosed according to the World Health Organization criteria.

Results. Metabolic syndrome was reported in 8% of the population. When subdividing patients based on positivity to metabolic syndrome criteria, no significant differences in motor and cognitive function, as well as quality of life and sleep disorders were observed between groups. However, patients with metabolic syndrome showed worse scores in Non-Motor Symptom Scale compared to patients without the syndrome, especially gastrointestinal, mood/apathy, sexual function, perceptual and miscellaneous symptoms. No significant differences in clinical correlates were observed when grouping patients based on which single metabolic syndrome component was present.

Conclusion. Metabolic syndrome might have an effect on non-motor symptomatology in PD, as patients with metabolic syndrome showed worse scores in Non-Motor Symptom Scale.

Key words. Cognition. Insulin resistance. Metabolic syndrome. Non-motor symptoms. Obesity. Parkinson's disease.

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Introduction

Parkinson's disease (PD) is the second most frequent neurodegenerative disease [1-3], after Alzheimer's disease. Epidemiologically, its incidence has been associated positively with age, affecting over 1% of the population over 60 years [4]. Clinically, the most common symptoms referred to PD are tremor, bradykinesia and rigidity, all motor type [2,3]. However, PD has a spectrum of non-motor symptoms, which include cardiovascular, sleep, gastrointestinal, cognitive disorders, among others. These have brought more and more attention to research due to the impact of non-motor symptomatology on function and wellbeing of patients with PD [5].

Despite all the effort put into the understanding of the pathogenesis of the disease, no full mechanism that explains the exact reason underlying dopaminergic cell loss in PD has been established. Lately, focus has been put on the metabolic causes of neurodegenerative diseases. A link between diabetes and earlier onset of PD has been demonstrated in population studies [6,7]. However, a pre-diabetic phase, that implies glucose dysregulations and imbalances, have also been shown to be present in PD patients [8], related to impaired insulin adaptive response. One disease that is caused by insulin resistance and abnormal adipose function is metabolic syndrome.

Metabolic syndrome is characterized by the presence of high blood pressure, high glucose levels, increased abdominal fat, and abnormal cholesterol or triglycerides levels. These glucose imbalances and high lipid levels have been associated with lipid peroxidation and lower antioxidant levels, which cause oxidative stress, leading to neuronal cell damage [9], explaining this way the possible relation between PD and metabolic syndrome. Scarce literature has evaluated the relation between these diseases. However, one study published recently suggested that metabolic syndrome and its components might be risk factors for PD [10]. In this study, we determined the prevalence of metabolic syndrome

and its components in patients with PD in our outpatient clinic and evaluated clinical features in this population.

Patients and methods

Our study was based using a database collected from all PD patients that attend Neurology Department outpatient clinic of the Hospital Universitario Dr. José E. González. The database is updated yearly and includes all information regarding sociodemographic characteristics, PD treatment, complications, and clinometric evaluations, obtained during assessment of disease. Within these evaluations, the following were completed: Movement Disorder Society-Unified Parkinson's Disease Rating Scale (MDS-UPDRS), the Non-Motor Symptom Scale for PD (NMSS), the Montreal Cognitive Assessment (MoCA), the Parkinson's Disease Questionnaire-8 (PDQ-8) scale for quality of life, and Parkinson's Disease Sleep Scale-2 (PDSS-2). Assessment involved disease staging, which was evaluated by the modified Hoehn and Yahr (HY) staging, measuring both impairment and disability. Among the patients collected in the database, no age restriction was applied for selection in this study. From the total data, we excluded those patients who had any missing variable (n = 18). We conducted a cross-sectional study with the remaining patients (n = 99) analyzing metabolic syndrome prevalence and clinical features of the PD population with this syndrome.

For metabolic syndrome prevalence analysis in the population we used WHO criteria [11], considering that their definition for metabolic syndrome could be covered best by variables in the database: type 2 diabetes mellitus (T2DM) or impaired glucose tolerance plus ≥ 2 of the following:

- Body mass index (BMI) > 30 kg/m^2 or waist to hip ratio (WHR) > 0.85.
- HDL-cholesterol < 40 mg/dL.
- Triglycerides ≥ 150 mg/dL.
- Blod pressure ≥ 140/90 mmHg or use of blood pressure medication.
- Microalbuminuria > 20 pg/min.
- Albumin/creatinine ≥ 30 mg/g.

All procedures performed in the study were in accordance with the ethical standards in compliance of the 1964 Helsinki declaration and its later amendments and received approval by the ethics committee of our institution. No written informed consent was required for the study.

Statistical analysis

Data was first tested for normality using Kolmogorov-Smirnov test. All continuous variables were thus expressed using mean± standard deviation (SD) or as median (interquartile range, IQR). Where appropriate, data were analyzed using student's t test or Mann-Whitney U-test. For multiple groups comparison one-way ANOVA test or Kruskal-Wallis test were used, using Bonferroni correction with an alpha value of 0.05. All categorical variables were expressed as percentages and for their analysis Chi-square or Fisher exact test were used. Linear regression analysis was conducted to assess the contribution of metabolic syndrome to MDS-UPDRS and NMSS scores. Metabolic risk factors for non-motor symptom severity were studied by logistic regression analysis with a 95% CI. A $p < 0.05$ was considered significant. All statistical analyses were assessed using the SPSS v. 23.0.

Results

Population baseline characteristics

We included a total of 99 patients, with a median age of 66 (17) years, most of the patients were male (68%), and had a tremor dominant subtype (61%), with a median of years with diagnosis of 7 (8) years. Median total MDS-UPDRS score was 57 (58), median NMSS score was 41 (55), median PDSS-2 score was 14 (17), while MoCA median score was 2 (4). The rest of baseline characteristics are shown in table I.

Metabolic syndrome and its components in patients with PD

Among components of metabolic syndrome, arterial hypertension showed the greatest prevalence among patients with PD with 30%. T2DM patients corresponded to 23% of the population, whereas obese patients corresponded to 27%. Dyslipidemia was present in 7% of the population. Analyzing prevalence of positivity to metabolic syndrome components, most patients fulfilled 0 to 1 criterion (42% and 37%, respectively), whereas the prevalence of metabolic syndrome was reported to be 8%.

Clinical features based on positivity to metabolic syndrome criteria

Based on the results obtained from positivity to metabolic syndrome criteria, we subdivided the

population in three groups: a) 0-1 criteria, b) 2 criteria, c) metabolic syndrome diagnosis to analyze clinical features for each group (shown in table II). There were no significant differences in gender, and motor subtype, but significances were observed in years of diagnosis and age (p = 0.014; p = 0.039). There was no significant difference in either HY stage or in presence of cognitive impairment per MoCA scores, sleep disturbances per PDSS-2 scores and motor impairment per MDS-UPDRS scores between groups, but non-motor symptomatology per NMSS scores did differ significantly (p = 0.009).

Post hoc analysis was carried out to assess which groups differed significantly in NMSS scores, showing that patients with metabolic syndrome had significantly higher scores compared to patients with 2 (p = 0.005), or 0-1 criteria (p = 0.013). Comparing NMSS scores in patients with 0-1 criterion to patients with 2, no significant difference was observed. Moreover, metabolic syndrome contributed significantly to NMSS score in a linear regression model adjusted for age and disease severity stage (B = 38.011; t = 2.564; p = 0.012), but did not affect motor symptomatology significantly (p = 0.119).

Metabolic risk factors for non-motor symptom severity

Based on the subclassification of NMSS of Chaudhuri et al [12], we subdivided the population's non motor symptomatology on severe versus non severe. Then, we proceeded to analyze metabolic predictors for presence of severity by logistic regression analysis with 95% CI. On univariate analysis, no factor was significant: presence of T2DM had an odds ratio of 1.436 (95% CI: 0.562-3.668), obesity 1.444 (95% CI: 0.597-3.492), arterial hypertension 1.120 (95% CI: 0.470-2.666), and dyslipidemia 1.115 (95% CI: 0.236-5.273).

Clinical features and metabolic syndrome component present in PD population

To evaluate any differences in clinical features based on metabolic syndrome component present, we subdivided the population with 1 criterion in groups: T2DM (n = 7), arterial hypertension (n = 13), dyslipidemia (n = 2), and obesity (BMI > 30; n = 15). No significant differences were observed in any clinical feature between groups. Total scores in MDS-UPDRS ranged from 41 to 73, NMSS total scores from 40 to 46, PDQ-8 scores from 11 to 28, PDSS-2 scores from 16 to 20, whereas MoCA scores ranged from 22 to 26. Most of the population in each group corresponded to tremor predominant motor subtype at onset.

Table I. Baseline characteristics in population with Parkinson's disease (n = 99).

Gender	Male	67 (68%)
	Female	32 (32%)
Age [a]		66 (17)
Years with diagnosis [a]		7 (8)
Years of education [a]		10 (7)
Motor subtype of onset	Tremor	60 (61%)
	Rigidity/bradykinesia	32 (32%)
	Instability gait	6 (6%)
	Mixed	1 (1%)
Smoking habit		35 (35%)
Treatment of Parkinson's disease	Treatment with levodopa	86 (87%)
	Other treatments (agonists, MAOIs)	13 (13%)
Hoehn & Yahr scores	0	2 (2%)
	1	11 (11%)
	2	45 (45%)
	3	32 (33%)
	4	9 (9%)
	5	0
MDS-UPDRS score [a]		57 (58)
MoCA score [a]		25 (4)
PDQ-8 score [a]		28 (31)
PDSS-2 score [a]		14 (17)
NMSS score [a]		41 (55)

MAOIs: monoamine oxidase inhibitors; MDS-UPDRS: Movement Disorders Society-Unified Parkinson's Disease Rating Scale; MoCA: Montreal Cognitive Assessment; PDQ-8: Parkinson's Disease Questionnaire-8; PDSS-2: Parkinson's Disease Sleep Scale-2; NMSS: Non-Motor Symptoms Scale. [a] Median (interquartile range).

Table II. Sociodemographic and clinical features based on positivity to metabolic syndrome criteria.

		0-1 criteria fulfilled (*n* = 79)	2 criteria fulfilled (*n* = 11)	Metabolic syndrome (*n* = 8)	*p*
Age [a]		66 (17)	65 (16)	56 (21)	0.039
Gender	Male	55 (70%)	7 (64%)	4 (50%)	0.691
	Female	24 (30%)	4 (36%)	4 (50%)	
Years with diagnosis [a]		5 (6)	11 (7)	8 (8)	0.014
Motor subtype of onset	Tremor	48 (60%)	7 (63%)	5 (63%)	0.875
	Rigidity	26 (33%)	3 (27%)	3 (37%)	
	Instability gait	4 (5%)	1 (9%)	0	
	Mixed	1 (1%)	0	0	
Hoehn & Yahr scores	1	9 (11%)	1 (9%)	1 (13%)	0.511
	2	37 (47%)	6 (55%)	3 (38%)	
	3	27 (34%)	3 (27%)	2 (25%)	
	4	6 (8%)	1 (9%)	2 (25%)	
MDS-UPDRS total score [a]		62 (53)	49 (21)	75 (89)	0.330
MDS-UPDRS part II score [a]		12 (16)	13 (13)	19 (16)	0.700
MDS-UPDRS part III score [a]		35 (29)	33 (13)	43 (51)	0.663
MDS-UPDRS part IV score [a]		0 (4)	2 (7)	0 (10)	0.684
PDQ-8 score [a]		25 (31)	16 (31)	38 (22)	0.063
MoCA score [a]		25 (5)	24 (6)	26 (9)	0.172
PDSS-2 score [a]		14 (15)	6 (16)	26 (24)	0.220
NMSS score [a]		40 (51)	17 (32)	76 (208)	0.009

MDS-UPDRS: Movement Disorders Society-Unified Parkinson's Disease Rating Scale; MoCA: Montreal Cognitive Assessment; PDQ-8: Parkinson's Disease Questionnaire-8; PDSS-2: Parkinson's Disease Sleep Scale-2; NMSS: Non-Motor Symptoms Scale. [a] Median (interquartile range).

Non motor phenotypes in PD population with metabolic syndrome

When comparing non motor symptoms in PD patients with metabolic syndrome versus patients without the diagnosis using NMSS (Table III), we observed significantly higher scores on the following domains in patients with metabolic syndrome: mood/apathy, perceptual problems, sexual function, gastrointestinal tract and miscellaneous. Cardiovascular, sleep/fatigue, urinary and attention/memory domains showed no significant differences between groups.

Discussion

In this study, we found that metabolic syndrome has a prevalence of 8% in patients with PD based on criteria from WHO. To our knowledge, scarce literature has approached to determine the prevalence of this syndrome and assess its clinical features in this population. One study found the prevalence to be 23% [13], using for the diagnosis National Cholesterol Education Program's Adult Treatment Panel III (NCEP ATP III), whereas another found the prevalence to be 7.5% [14], similar to the one in our study, but no criteria used to diagnose metabolic syndrome was mentioned.

One of the most important findings in the present study is the fact that non motor symptomatology, measured by the NMSS total score, showed worse scores in patients with metabolic syndrome compared to patients without metabolic syndrome or 1 to 2 components of metabolic syndrome, these differences showing significance ($p = 0.009$). To our knowledge, no other study has shown an effect of metabolic syndrome on non-motor symptoms measured by the NMSS. However, there are studies that have shown a relationship between metabolic syndrome and increased risk on cognitive impairment, as well as reduced risk of falls [13,15]. These support partially our findings, as patients with metabolic syndrome in our study had higher scores on the mood/apathy domain, whereas no differences in scores were observed between groups in the cardiovascular domain, which includes falls.

As the logistic regression model showed, single components of metabolic syndrome showed no association with severity of NMSS. Furthermore, when comparing patients with only one metabolic syndrome criteria, no differences were observed in NMSS scores, which raises the idea that no single component has greater contribution, but their rather combined presence could be key on the difference in non-motor symptomatology. A study showed no difference in non-motor symptomatology in PD patients with insulin resistance compared to patients without this [16]. Moreover, another study demonstrated that patients with T2DM had similar results in clinical correlates compared to non-diabetic PD patients [17]. However, a higher than nor-

mal BMI in PD patients at diagnosis had a protective effect in cognitive decline in other study [18], suggesting a single component contribution to this non-motor symptom.

Our study found no difference in motor function comparing patients with metabolic syndrome to patients without the syndrome or having 1 to 2 components of this. This contrasts what other studies have shown, that metabolic syndrome played a role in motor function, by showing a greater increase in UPDRS total score in PD patients with metabolic syndrome compared to controls through follow-up [19] and showing variability in motor function based on metabolic syndrome criteria present, having high triglycerides a protective effect, whereas high blood pressure and low HDL-cholesterol levels were associated with poorer motor function [20].

The link between metabolic syndrome and neurodegenerative disorders has been the focus of research lately, and studies suggesting possible mechanisms for their relation have been published [21,22]. The main problematic of this relation is establishing whether metabolic syndrome is a cause or consequence of PD. A study showing glucose imbalances in patients with PD proposed this syndrome as a novel non motor consequence of PD associated dysautonomia [8]. However, two studies addressing metabolic syndrome as risk factor for PD have been published. One cohort-study showed an increase risk in PD incidence in patients with metabolic syndrome in all components of the syndrome [10], whereas the other cohort-study showed reduced risk in PD incidence in patients with metabolic syndrome with high triglyceride and fasting glucose, but increased risk with high BMI [23].This is interesting as meta-analysis of the single components of metabolic syndrome and future PD risk have shown that obesity and hypertension may increase future risk of PD [24,25], whereas controversy exist whether diabetes contributes to the presence of this disease [26].

Considering the results in our study, and the neuroprotective effect that has been attributed to insulin [27], agents that improve its sensitivity could play a major role in PD, as they could serve as a promising complementary treatment alongside levodopa, aiming to aid on ameliorating non-motor symptomatology. A study showed better outcomes in mood/apathy domain of NMSS in PD patients treated with exenatide compared to controls [28], however, no significant differences were observed for other domains. Another insulin sensitizing agent, glitazone, showed no effect on dementia or depression in PD patients compared to placebo [29]. Nonetheless, considering the contribution of non-motor symptomatology to the great burden on PD patients [30,31], more studies need to be developed in order to study the exact role metabolic syndrome could play on PD, as this could help develop new ways to treat this disease. On the other hand, a study aimed at assessing the effect of different oral antidiabetics on non-motor symptoms in PD patients should be considered for future research plans.

This study had limitations, some associated with its cross-sectional design. For example, we cannot conclude whether metabolic syndrome is a cause or consequence of PD. On the other hand, the prevalence of metabolic syndrome could have been underestimated considering that we used WHO criteria for diagnosis since it best fitted the variables in the database, instead of using other criteria more used in research. This is of importance as the prevalence of metabolic syndrome in our population is low (8%), and this could bias our results obtained. However, this study intends to describe and compare the findings in our database, and we refer to the data found on it. A future project with an increased sample size of patients with metabolic syndrome and PD and a prospective cohort methodology to deepen the understanding of this relation

Table III. Non-Motor Symptoms Scale in Parkinson's disease (PD) patients with and without metabolic syndrome.

	PD with metabolic syndrome (*n* = 8)	PD without metabolic syndrome (*n* = 91)	*p*
Cardiovascular [a]	1 (3)	1 (4)	0.936
Sleep/fatigue [a]	15 (25)	8 (12)	0.099
Mood/apathy [a]	14 (44)	5 (13)	0.023
Perceptual problems [a]	0 (7)	0 (0)	0.027
Attention/memory [a]	2 (18)	1.5 (5)	0.065
Gastrointestinal [a]	8 (17)	2 (8)	0.042
Urinary [a]	4 (14)	2 (7)	0.244
Sexual function [a]	5 (21)	0 (1)	0.030
Miscellaneous [a]	9 (15.5)	3.5 (12)	0.047
Hyposmia [a]	4 (9)	0 (2)	0.047

[a] Median (interquartile range).

could be promising. Furthermore, we focused on describing non-motor symptomatology only per NMSS as we considered it a useful tool for assessment of these symptoms, however, more scales assessing non motor symptoms could have been used.

In conclusion, this study shows a possible interaction of metabolic syndrome in PD via non motor symptomatology, which should be further assessed in a greater sample size and in a prospective methodology, in order to understand in a deeper way the relationship between these diseases. Nonetheless, this study serves as a stimulus for more research regarding the relation between these two diseases.

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Características clínicas del síndrome metabólico en pacientes con enfermedad de Parkinson

Introducción. Recientemente, se ha incrementado la atención hacia causas metabólicas de la pérdida de células dopaminérgicas en la enfermedad de Parkinson (EP), dada la intolerancia a la glucosa que pueden presentar estos pacientes. Un síndrome caracterizado por resistencia a la insulina es el síndrome metabólico.

Objetivo. Determinar la prevalencia y las características clínicas del síndrome metabólico y sus componentes en pacientes con EP.

Pacientes y métodos. Se analizaron variables de 99 pacientes con EP. Se registraron escalas que evalúan las funciones motora, no motora y cognitiva, los trastornos del sueño y la calidad de vida. El síndrome metabólico se diagnosticó según los criterios de la Organización Mundial de la Salud.

Resultados. La prevalencia de síndrome metabólico se notificó en un 8%. Al subdividir a los pacientes en función de los criterios positivos de síndrome metabólico, no se observaron diferencias significativas en las funciones motora y cognitiva, la calidad de vida ni los trastornos del sueño entre los grupos. No obstante, pacientes con síndrome metabólico mostraron peores puntuaciones en la escala de síntomas no motores en comparación con pacientes sin el síndrome, especialmente en cuanto a tracto gastrointestinal, estado de ánimo/apatía, función sexual, problemas perceptivos y misceláneos. No se observaron diferencias significativas en las características clínicas al agrupar a los pacientes sobre la base del componente único de síndrome metabólico presente.

Conclusión. El síndrome metabólico podría tener un efecto sobre la sintomatología no motora en la EP, ya que los pacientes con este síndrome mostraron peores puntuaciones en la escala de síntomas no motores.

Palabras clave. Cognición. Enfermedad de Parkinson. Obesidad. Resistencia a la insulina. Síndrome metabólico. Síntomas no motores.