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Original article

Low cardiometabolic risk in Parkinson's disease is independent of nutritional status, body composition and fat distribution☆

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SUMMARY

Background & aims: To investigate if the reduced cardiometabolic risk in Parkinson's disease (PD) is independent of nutritional status, body composition and fat distribution.
Methods: We designed a case–control study comparing 80 non underweight PD patients with 80 controls matched for sex, age and body mass index (BMI). Nutritional assessment included: anthropometry (BMI and waist circumference [WC]), body composition estimated by impedance and biochemistry (fasting glucose, serum lipids and transaminases). The presence of arterial hypertension, diabetes mellitus and metabolic syndrome (MetS) were noted.
Results: Compared to controls and independently of gender, PD patients showed lower percentage of body fat ($P < 0.001$) and biochemical parameters (glucose, $P < 0.001$; total cholesterol, $P < 0.001$; LDL, $P < 0.001$; triglycerides, $P = 0.002$; alanine aminotransferase, $P < 0.001$ and aspartate aminotransferase, $P = 0.015$) but similar WC ($P = 0.324$). The prevalence of hypertension and MetS was similar in the two groups, as well as the frequency and the number of MetS criteria. The relationship between PD and low cardiometabolic profile was independent of age, gender, current smoking and BMI. After adjusting for WC and body fat, most of the associations remained significant.
Conclusions: PD patients seem to have a more favorable cardiometabolic risk profile, independently of nutritional status, body composition and fat distribution.

1. Introduction

Parkinson's disease (PD) is a chronic neurodegenerative motor disorder affecting 1 out of 800 people all over the world.[1]

Typical motor symptoms (bradykinesia, resting tremor, rigidity and postural instability), together with complications or consequences of both pharmacological and non-pharmacological treatments may cause changes in food intake, lifestyle and energy requirements associated with weight loss or weight gain.[2] Previous studies have reported that the prevalence of overweight and obesity among PD patients changes during the course of disease.[2–4] An Italian population study has shown that the prevalence of obesity among PD patients is about 50% higher than that in the general reference population.[3] However, it has also been observed that body mass index (BMI) progressively decreases during the course of the disease and that undernutrition may also occur.[3] Indeed, the limited literature available suggests that fluctuations in body weight are mainly due to changes in fat mass, which are gender-specific to some extent.[5–7]

Cardiovascular disease (CVD) still remains the main cause of mortality in the general population and the occurrence has been linked to body weight excess through the adverse effect of adipose-tissue-related complications such as hypertension, diabetes and dylipiudemia.[8,9] Despite the higher prevalence of overweight and obesity, PD patients appear to be less susceptible to CVD,[10,11] but the mechanisms behind this protection have been scantily investigated. In a previous retrospective case–control study[12] it was found that the prevalence of CV risk factors in PD patients is significantly lower than in sex and age-matched controls and the

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reduced autonomic activity has been fingered as one of the potential explanations for the phenomenon.[12,13] However, the effect of other confounders, such as nutritional status, adiposity and its distribution over the body, has not been investigated. This is particularly intriguing because not only total and abdominal adiposity have been associated with cardiometabolic risk and the risk of death[14,15] but also because body fat mass, particularly visceral adipose tissue (VAT), have been associated also with chronic neurodegeneration.[16]

Aim of our case–control study was to evaluate the relationship between low cardiometabolic risk in PD and nutritional status, body composition and body fat distribution.

2. Methods and procedures

The study was performed in agreement with the principles of the Declaration of Helsinki. The protocol was approved by the local Ethics Committees and written informed consent was obtained from every patient recruited. All patients underwent a complete nutritional assessment and medical examination, including blood pressure measurement. Information on smoking history and pharmacological treatment was also collected.

All the evaluations were performed early in the morning and in fasting conditions. PD patients were assessed after taking PD medications in order to avoid any bias due to movement disorders (e.g. rigidity or resting tremor) which could theoretically affect some evaluations (e.g. height and body composition).

The exclusion criteria were: refusal to give informed consent, use of lipid-lowering medications, thyroid disease, viral hepatitis (B or C; by appropriate serological markers), body weight changes in the previous 6 months, neurosurgical procedure for PD (deep brain stimulation or pallidotomy). Patients who were overtly underweight (BMI < 18.5 kg/m^2)[17] were also excluded, because they are obviously less likely to present an adverse cardiometabolic risk profile. Along with this, low body weight or malnutrition in PD patients is likely to be due to the combination of increased energy expenditure produced by dyskinetic movements and inadequate food intake,[3,18] while in the general population it usually is the result of disease-related inflammatory disorders and inadequate food intake.[19]

2.1. PD patients

From January 2008 to January 2010, eighty patients with a diagnosis of PD according to UK Brain Bank criteria[20] living in the community, who were admitted to the Parkinson Institute for periodic disease reassessment (elective hospital stay), and fullfilled the inclusion criteria, were consecutively recruited.

2.2. Controls

During the same period, sedentary controls ($n = 80$) matched in pairs for age (± 3 years), gender and BMI (± 1 kg/m^2) were recruited among the population of subjects attending the International Center for the Assessment of Nutritional Status for weight concerns.

2.3. Nutritional assessment

The procedures included were:

Anthropometry. Subjects were wearing only underwear. Height (to the nearest 0.5 cm) and body weight (to the nearest 0.1 kg) were measured by the same calibrated scale equipped with a telescopic vertical steel stadiometer (SECA 711; Germany). For those patients with evident spinal cord deformities height was derived by validated equations.[21] Afterward, the body mass index (BMI) was calculated as the ratio between weight [kg] and height [m] squared (kg/m^2).[17] Waist circumference (WC; to the nearest 0.5 cm) was measured through plastic flexible tape-measures at the midpoint between the lowest rib and the iliac crest, placing the tape perpendicular to the long axis of the body and parallel to the floor.[17,22] Also skinfold thickness (SFT; by a Holtain caliper to the nearest 0.2 mm – Holtain LTD, Crymych, UK) was measured at four sites (biceps, triceps, subscapular and suprailiac) to better evaluate subcutaneous fat deposition. The sum of all skinfolds was considered in the analysis. All anthropometric measurements were performed in triplicate according to standard procedures[17] and the mean of three values was considered in the analysis.

Biochemistry. Venous blood samples were drawn after 8–12 h of fasting and the following parameters were assessed using conventional automated analyzers: glucose, total cholesterol, high and low density lipoprotein cholesterol (HDL and LDL, respectively), triglycerides, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) values.

Body composition. Whole-body resistance was measured at frequencies of 50 kHz (R_{50}), following international guidelines and using four-polar impedance meters (Tanita Segmental Multifrequency Body Composition Monitor, MC 180 MA, Sensormedics, Milan, Italy [at the Parkinson Institute] or Human IM Scan, DS-Medigroup, Milan, Italy [at the International Center for the Assessment of Nutritional Status]). Fat-free mass (FFM; kg) was then calculated using R_{50} values according to the formula for healthy adults proposed by Deurenberg et al.[23] FFM was then normalized for height to calculate the FFM index (FFMI; FFM [kg] and height [m] squared [kg/m^2]) according to Pichard et al.[24] Percentage of body fat mass (BF%) was also derived.

2.4. Cardiometabolic profile

The cardiometabolic profile was based on biochemical parameters (total cholesterol, HDL, LDL, triglycerides, glucose, ALT and AST), established comorbidities (arterial hypertension and diabetes mellitus) and the presence of metabolic syndrome (MetS+). Comorbidities were defined as follows: arterial hypertension as repeated blood pressure measurements $\geq$140/90 mmHg or the reported use of antihypertensive medications; diabetes as at least two blood glucose measurements $\geq$126 mg/dL or reported antidiabetic treatment. The National Cholesterol Education Program's Adult Treatment Panel III criteria[25] were used to define MetS+. Accordingly, subjects had to have $\geq$3 of the following: (1) WC $>$102 cm in men and $>$88 cm in women; (2) serum triglycerides $\geq$150 mg/dL; (3) HDL cholesterol $<$40 mg/dL in men and $<$50 mg/dL in women; (4) blood pressure $\geq$130/85 mmHg; and (5) fasting plasma glucose level $\geq$110 mg/dL. The total number of criteria was also considered in the analyses.

2.5. Statistical analysis

Statistical analyses were performed by MEDCALC® for Windows Version 11.3.0.0 (MedCalc Software, Mariakerke, Belgium), setting the level of significance at a two-tailed P-value <0.05.

Continuous variables are reported as mean and standard deviation (in case of normal distribution), or median and interquartile range (25th–75th percentile; in case of non normal distribution). The Kolmogorov–Smirnov test was used to test for normal distribution of the data. Categorical variables were presented as counts and percentages.

Descriptive and parametric statistics were initially considered before and after categorizing both PD patients and controls on the basis of: