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Research Article

Lower Body Parkinsonism Patients: Increased Blood Flow in Posterior Cerebral Arteries

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Abstract

Introduction: Lower Body Parkinsonism (LBP) is a bilateral, symmetric lower limbs Parkinsonism without a resting tremor, a poor L-dopa response and with a minimal upper limbs involvement. Patients usually demonstrate gait difficulties due to freezing gait and falls, causing a major grade of disability when compared to idiopathic Parkinson's Disease patients (PD). Small vessel disease has been related to the pathophysiology of this type of secondary Parkinsonism. We decided to study the hemodynamic characteristics of the cerebral arteries of patients affected with LBP and compare them to PD patients using a Transcranial Doppler.

Patients and Methods: We enrolled 12 LBP, 15 PD patients and 15 age-matched healthy control subjects. A Transcranial Doppler was performed on patients and subjects in order to determine the systolic and diastolic velocities and pulsatility index (PI) in the anterior and posterior circuit cerebral arteries.

Results: The LBP patients consisted of 7 men and 5 females; mean age 74 years; Hoenh and Yarh stages 2 and 3. PD patients consisted of 9 males and 6 females; mean age 70 years; Hoehn and yahr stages 2 and 3. LBP patients showed a mean systolic velocity of 193 cm/s, diastolic velocity 64cm/s and PI 2.22 (p: 0.01) in the Posterior Cerebral Arteries (PCA), The remaining cerebral arteries showed normal velocities and PI in LBP and PD patients as well as in normal subjects.

Conclusion: The LBP patients of this study showed a statistically significant difference in the mean systolic velocity and PI of the PCA compared to PD patients and normal subjects.

Keywords: Transcranial doppler; Lower body parkinsonism; Vascular parkinsonism

Introduction

Parkinsonism due to cerebrovascular disease or Vascular Parkinsonism (VP) is a heterogeneous clinical entity representing 4-12% of all cases of Parkinsonism [1]. Lower Body Parkinsonism (LBP) is a recognized clinical expression of VP and is characterized by rigidity and bradykinesia, mainly affecting the lower limbs. Patients with LBP demonstrate a great difficulty for walking; postural instability and falls are the most remarkable clinical symptoms [2,3]. According to some authors, patients with the clinical criteria of LBP show a poor response to L-dopa therapy and resting tremor is an uncommon finding [4,5]. LBP is considered for some authors as a variety of vascular Parkinsonism, with other clinical presentations of VP found in the medical literature [6,7]. The etiology of LBP is unknown, but it has been classically related to vascular risk factors for stroke [8, 9]. As with other secondary Parkinsonism, the diagnosis is based on clinical criteria and few paraclinical tools are useful nowadays to confirm LBP. For the time being, there are no biomarkers for LBP, neither electrophysiological nor radiological biomarkers for LBP have been reported in the medical literature [10,11]. In view of the high incidence of vascular risk factors for stroke in LBP patients, we designed a clinical-sonographic study to determine the hemodynamic characteristics of the intracranial arteries of patients with LBP.

Patients and Methods

We defined LBP as a lower body Parkinsonism (rigidity and bradykinesia in the lower limbs) disproportionate in relation to the upper limbs and an L-dopa test improvement of less than 30% in the Part III of the UPDRS scale.

12 patients with the clinical criteria of LBP, 15 Parkinson's Disease (PD) patients and 15 aged matched-non ill subjects with similar demographic characteristics were enrolled. Other secondary causes of Parkinsonism were ruled out. Patients with a clinical history of ischemic stroke were excluded.

The clinical evaluation consisted in total UPDRS, Hoen and Yahr stages and Swab and England Scales. Determination of Vascular risk factors for stroke was asked systematically to patients and subjects. A cranial angio-MRI was performed in all the patients, focusing on the brainstem.

A continuous Transcranial Doppler with a 2Hz transducer was performed on the LBP, PD patients and on normal subjects. PD and LBD patients were under l-dopa effects when the Transcranial Doppler was performed. The mean time duration between the l-dopa intake and the Transcranial Doppler was 2 ± 0.5 hours.

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Transtemporal and sub-occipital acoustic windows were used to evaluate the anterior and posterior brain vascular circuit (bilateral) and posterior circuit. Systolic and diastolic velocities, as well as Pulsatility Index (PI) were measured in all the arteries evaluated. We carefully measured the following: Middle Cerebral Artery (MCA), Anterior Cerebral Artery (ACA), Anterior Communicating Artery (AcoA) Posterior Communicating Artery (PcoA), Basilar Artery (BA), Vertebral Arteries (VA) and Posterior Cerebral Arteries (PCA) of both hemispheres.

Clinical findings

LBP patients consisted of 5 women and 7 men, a mean age 74 years and a mean disease onset of 5 years. H-Y scale: 9 patients showed stage II and the remaining 3 showed stages III. S-E scale showed a mean of 70% and a mean L-dopa dose of 845 mg. PD patients consisted of 6 women and 9 men, a mean age 70 years with a mean disease onset of 6 years. H-Y scale: 10 patients showed a stage II and 5 showed a stage III. S-E scale showed a mean of 80% and a mean L-dopa dose of 650mg. High blood pressure was present in all LBP patients (100%, p:0.02) with only 20% of PD patients and 30% of normal subjects exhibiting high blood pressure. Diabetis Mellitus was also present in 50% of LBP patients (p: 0.04) and only 20% of PD patients (Table 1).

LBD patients showed a mean l-dopa dose of 845 Mg ± 20 and PD patients showed a mean l-dopa dose of 645 Mg ± 14 (*P*: 0.07).

Transcranial doppler findings

In the posterior circuit, the basilar artery in LBP patients had a mean systolic velocity of 78 cm/sec. (p: 0.06), mean diastolic velocity of 23 cm/sec (p: 0.7) with a mean of PI of 0.76 cm/sec. (p: 0.6); the PCA showed a mean of systolic velocity of 196 cm/sec (p: 0.04), mean of diastolic velocity 64 cm/sec (p: 0.03) and a PI of 2.23 cm/sec (p: 0.02). The arteries of the anterior circuit evaluated (MCA and ACA) of both hemispheres showed normal values of mean systolic and diastolic velocities as well as PI values with no statistical significance when compared to normal subjects (Table 2).

Cranial MRI findings

The cranial angio-MRI of our LBP patients showed in the majority, a global brain hypotrophy (9/12, 75 %, p: 0.02),and in 8/12 of the patients (61%, p: 0.04) hyper-intensity signals in T2 sequence in brainstem, particularly in the pons. 11 of the patients (91%, p: 0.02) showed a reduction in SNPc with statistical significance when compared to PD patients and normal subjects (p: 0.02) (Table 3).

Table 1: Demographic characteristics of LBD and PD patients.

	VP-LBP N=12	PD N=15	Normal subjects N= 15
Age	74	72	76
Female/male	5/7	9/6	8/7
Onset (years)	5	6	-
H-Y stage	7 (III) 5 (II)	8 (III) 7(II)	-
S-E scale	70%	80%	-
L-dopa dose	845 ± 20 Mg	650 ± 14 Mg	***P*: 0.07**
Hypertension	12 (100%) ***p*: 0.01**	2 (13%)	3 (20%)
Diabetis	6 (50%) ***P*: 0.04**	2 (13 %)	1 (6 %)
Dislipemia	7 (58%) ***p*: 0.03**	5 (33 %)	3 (33%)

Table 2: Transcranial Doppler in LBP patients. PCAs showed increased systolic and diastolic velocities and PI.

Cerebral arteries	**SV (Mean) *p***	**DV (Mean) *p***	**PI (Mean) *p***
MCAs	69 *p*: 0.7	18 *p* :0.6	0.72 *p*:0.6
ACAs	96 *p*: 0.6	22 *p*: 0.6	0.8 *p*:0.06
PCAs	**196 *p*: 0.04**	**64 *p*: 0.03**	**2.34 *p*: 0.02**
BA	78 *p*: 0.6	23 *p*:0.7	0.76 *p*: 0.6
VAs	42 *p*: 0.8	9 *p*:0.8	0.63 *p*: 06

Table 3: Transcranial Doppler in PD patients. Normal Velocities and PI.

Cerebral arteries	**SV (Mean) *p***	**DV (Mean) *p***	**PI (Mean) *p***
MCAs	74 *p* : 0.09	18 *p* :0.3	0.34 *p* : 0.6
ACAs	80 *p*: 0.8	19 *p*: 0.8	0.35 *p*:0.8
PCAs	**101 *p*: 0.07**	**45 *p*: 0.07**	**0.78 *p*: 0.05**
BA	45 *p* :0.7	15 *p* :0.7	0.75 *p*:0.7
VAs	40 *p*: 0.6	12 *p*: 0.6	0.67 *p*: 0.9

Discussion

Vascular Parkinsonism patients are a challenge for clinicians and researchers. The lack of a deep knowledge and a poor understanding of the pathophysiology of this uncommon type of Parkinsonism is a reality that is reflected in the lack of a widely accepted clinical criterion and other diagnostic tools published in the medical literature.

We decided to study the hemodynamic characteristics of the intracranial arteries in LBP and PD patients given the high association of cerebrovascular risk factors in LBP patients reported in the past. The Transcranial Doppler can show dynamic characteristics of the blood flow into the intracranial arteries [12,13], Such characteristics are often difficult to obtain with other neuro-imaging technics such as angio-MRI or functional neuroimaging (eg. Positron emission tomography, single-photon emission computed tomography), as these tend to show morphological and metabolic features more than the hemodynamic characteristics of intracranial arteries (Table 4).

More LBP patients in this study had hypertension and diabetes than PD patients and normal subjects as has been reported by other authors. PD and LBD patients were under l-dopa effects when the Transcranial Doppler was performed. It is known that l-dopa is a vasomotor drug and maybe could play a role in systemic hemodynamic changes in PD and other parkinsonian patients; however our patients showed no blood pressure changes during the Transcranial Doppler performance. LBP patients also showed an increased mean systolic and diastolic velocities, as well as an increased mean PI in the PCA when compared to PD patients and normal subjects. The remaining

Table 4: Cranial MRI findings LBP and PD patients. LBP patients showed: SNPc reduction, Brainstem hyper intensity and Global hypo trophy statistically significance compared to PD patients.

	SNPc reduction	Brainstem hyper intensity	Global hypo trophy
LBP n=12	11 (91%), **p:0.02**	11 (91%) %, p=**0.01**	10 (83%), p:**0.02**
PD n= 15	40%, p: 0.06	10%, p: 0.07	30%, p: 0.05

intracranial arteries studied in LBP patients of this study showed normal values of velocities and PI.

The increased values of systolic and diastolic velocities, as well as PI in the PCA of LBP patients in this study is a remarkable finding because such values represent a moderate to severe (> 50%) vascular stenosis according to standardized values reported in the past [14]. The PI represents the vascular resistance to the blood flow and the PCA supplies the mesencephalus; particularly the Substantia Nigra Pars compacta (SNPc) and part of the striatum, and the basilar artery supplies to a major part of the brainstem.

We hypothesize that the vascular resistance that shows the PCA could cause a chronic sub-cortical ischemia, particularly in SNPc and striatum, and such chronic ischemia could cause a reduction of SNPc as we showed in the cranial MRI of LBP patients of this study. Such findings have been published in previous studies of cranial MRI in vascular Parkinsonian patients in the past [15,16]. Simultaneously, a disruption in the dopamine receptors of the striatum, probably due to the sub-cortical ischemia, could explain the poor clinical response to L-dopa in LBP patients. As discussed above, the cranial Angio-MRI of our LBP patients showed in the majority (10/12), a global hypotrophy and in 11 of these patients (91%), hyper-intensity signals in T2 sequence, particularly in the pons. 11 of these patients (91%) showed a statistically significant reduction in SNPc when compared to PD patients and normal subjects (p: 0.02).

LBP patients have a high incidence of vascular risk factors for stroke and a moderate to severe stenosis of PCA in these patients could play an important role in the pathophysiology of a possible chronic ischemia in the striatum and brain stem; however we could not explain why the PCAs of our LBP patients showed increased mean in the velocity of blood flow and PI, and not the rest of intracranial arteries. Further studies are needed to better understand the pathophysiology of this type of Parkinsonism.

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