eScientific Library

Volume 1 Issue 1
www.escientificlibrary.com

ES Journal of Neurology

Parkinson's disease in the Differential Diagnosis of Dizziness: Accuracy the Anamnesis

Case Report

Alberto Vargas Cañas[1*] and Francisca Mendoza[2]

[1]*Neurology Unit, Hospital Santiago Oriente, Chile*
[2]*Neurologist´s ResidentProgram de Neurología, Clínica Alemana-Universidad del Desarrollo, Chile*

Received: Jan 28, 2020; **Accepted:** Feb 24, 2020; **Published:** Feb 25, 2020

***Corresponding author:** Dr. Alberto Vargas Cañas, Neurology Unit, Hospital Santiago Oriente "Dr. Luis TisnéBrousse": Av. Las Torres 5150, Peñalolén, Santiago 7930124, Chile

Abstract

Parkinson's disease (PD) is one of the most frequent chronic neurodegenerative disorders in neurological practice. This condition has well recognized diagnostic criteria such as at least bradykinesia with one or more of the following: 4 to 6 hertz rest tremor, muscular rigidity, and loss of postural reflexes. However, on unusual occasions, the classical clinic is not very evident, and the initial symptoms of PD can lead to diagnostic confusion. One of these symptoms is Postural Instability (PI); which is classically presented in advanced stages; but can also do it early in this condition; and can be confused with dizziness and vertigo; delaying the diagnosis of PD, and the onset of symptomatic treatment. We present three patients with nonspecific clinic, who did consulting for second medical opinion for dizziness, vertigo or PI; and after a detailed anamnesis and physical examination, the diagnosis of early PD was concluded. We review the pathophysiology of PI in PD, and want to keep attention - especially to non-specialist physicians - that in the differential diagnoses of dizziness-vertigo, PD may be included; and keep in mind, that this form of presentation, with early PI, may indicate a variant of the PD that predicts a greater PD severity.

Keywords

Vertigo; Dizziness; Postural instability; Parkinson´s disease; Differential diagnosis

Introduction

Parkinson's disease (PD) is one of the most frequent neurological, chronic, and neurodegenerative disorders, affecting 1% of individuals over 60 years of age [1]. The clinical diagnosis of PD is made on the identification of cardinal motor signs such as tremor, bradykinesia, rigidity and Postural Instability (PI) [2,3]. This last symptom becomes more prevalent and worsens as the disease progresses, and is one of the most disabling symptoms associated with falls and loss of independence [4]; and it can be described by patients as dizziness, an unspecific term. On the other hand, one of the most common disorders in clinical practice is vertigo [5], this is defined as the illusion of movement (of objects or body in space) [6], and has clinical characteristics that can orient it to a peripheral or central origin.

IP is defined as the alteration of balance control, which consists of the ability to maintain the balance of the body in relation to the gravitational force, thus compromising the ability to maintain or change the position when

walking or standing [4]. The pathophysiology of PI in PD has not been well understood. Through static and dynamic posturography, several factors that can contribute to IP have been identified, such as: alteration of the automatic postural response (reactive and anticipatory), bradykinesia in the correction of gait, arm abnormality and axial rigidity. However, there is a lack of understanding of neurochemical and neuropathological changes [7]. There are reasons to believe that not all manifestations of PD result only from the loss of dopamine in the nigrostriatal pathway; and it can explain why the PI is refractory to treatment with dopaminergic drugs. Even balance problems can be aggravated by the adverse effects of dopaminergic drugs such as dyskinesia. Then, IP could be the result of "extranigral" or "non-dopaminergic" lesions. Postmortem studies have clearly identified the presence and extent of non-dopaminergic lesions, which typically develop in the elderly with longstanding PD. For example, loss of noradrenergic neurons has been seen in the Coeruleus Locus (LC) [7].

In animal studies, it is suggested that the noradrenergic system is related to the gait ignition, possibly with control of the balance and contribute to the control of the vestibulospinal reflex of the extremities, which are severely altered in PD [7]. Also, it has been seen in series of cases of patients with newly started PD, evidence of findings that support a polyneuropathy, characterized primarily by a loss of distal sensitivity that could contribute to balance and balance alterations in these patients [8]. Since "dizziness" is a frequent reason for consultation, and usually does not include PD as a differential diagnosis, this was the symptoms in the 3 cases related a continuation.

Clinical Case 1

A 72-year-old woman without morbid history has complaints of a 02 previous years of a sensation of loss of equilibrium. She was evaluated by otorhinolaryngology, and treated with Diphenidol 75mg per day and Cinnarizine 75 mg daily for possible diagnosis of Benign Paroxysmal Postural Vertigo (BPPV), and was studied with tests all them in normal range. In view of the increase of his symptoms, he requests a second opinion to neurology. In a directed anamnesis refers no response to treatment; and note slow march, and increase of the sensation of IP since the beginning of the anti-vertiginous treatment. On physical examination, was evident a facial hypomimia, non nystagmus, less arm movement, cogwheel in upper right extremity and hypokinesia. It is studied with brain images, blood count, vitamin B12 levels, vdrl and biochemical profile, all them normal. A diagnosis of PD was made, the others therapies were stopped, and it was indicated Levodopa / Carbidopa (200/50). It evolves with fewer motor symptoms.

Clinical Case 2

An 85 years old women with previous diagnosis of arterial hypertension, consults to neurology for a second opinion. Her symptoms, has about 2 months of slow installation of progressive PI not associated with tremor. She was studied by general practionary who starts Ondasentrone 12 mg daily and Cinnarizine 75 mg QD. Soon; from the onset of the anti-vertiginous treatment, a gait more slowness and persistence of the IP were more notorious. At physical examination, it was evidenced a facial hypomimia, less arm movement during gait test, ligth cogwheel and bradykinesia. A cerebral imaging was made, complete blood count, vitamin B12, vdrl and biochemical profile, all without alterations. It was decided to perform a therapeutic trial with Levodopa / Carbidopa (200/50) and stop previous treatments indicated. In subsequent control, reduction of hypokinesia is evidenced.

Clinical Case 3

A 55-year-old woman who consults neurology, for dizziness not exacerbated by postural change. In the specific anamnesis (Table 1), her husband reports that about a year ago there is a loss of balance and insecurity walk. The finds at neurologic examination were facial hypomimia, less arm balance during walking, and a pull-test (+). It is evaluated with cerebral imaging, total blood count, vitamin B12 levels, vdrl and biochemical profile, all normal. Therapy is started with Levodopa / Carbidopa (200/50). Her symptoms reduce in a posterior evaluation.

Discussion

Occasionally, PD starts with IP, a symptom that can be a cause of diagnostic confusion and consequently delay the start of treatment. This would be explained due the cardinal symptoms of IP can be confused to conditions like peripheral vertigo, more specifically, BPPV; so it is common that in early stages the first consultation not be done to a neurologist but other specialties or general medicine. This means that the diagnoses made are mainly of other specific pathologies in the area of specialization of the attending physician, sometimes producing, in addition to the delay

in the diagnosis, an exacerbation of the symptoms due to the prescribed medications, the above is observed in two of the clinical cases described.

Although PI usually does not appear until late stages of PD evolution, three cases that started their symptoms with this manifestation are described, which led to initial diagnostic confusion? In the same context, the evidence indicates that patients with early PI should be considered an atypical parkinsonian (APD) disorder such as progressive supranuclear paralysis or multisitemic atrophy [4]; but in our patients, contrary to the habitual seen in APD, there were a good response to L-Dopa. One observation that we think in our three patients, is that their type of PD, perhaps due to the precocity of the diagnosis, or its predominant symptoms, could be a "rigid-akinetic" variant; and be a more aggressive PD that in early stages of the disease they already have IP; this would be a factor of poor prognosis in the progression to long-term severity, as was verified in a study by Rajput et al. [10].

PD is still a clinical diagnosis; and it can be supported by tests that confirm the diagnosis, such as transcranial Doppler; but this test requires even more protocolization, and has shown diagnostic specificity near to 80% [8]. By this reason is that the diagnosis of PD depends on the physician's ability to perform a right anamnesis and recognize the cardinal signs, especially in the early stages of the disease. Many times the patients consult for "dizziness"; which is a non-specific term, and it is necessary to keep warm to the differential diagnosis such peripheral or central vertigo, presyncopal symptoms, imbalance [5]; and include the suspected IP of early PD.

Conclusions

The correct anamnesis is still the basis of a right and prompt diagnosis; and in patients with nonspecific symptoms like dizziness, vertigo or postural instability has a primordial importance, specially if has clinically findings that suggest a early PD like hypomimia, hypokinesia, frequents falls and bad response to anti-vertiginous treatment.

Ethical issues

Patients were asked to authorize the use of data from their clinical files, without using names or other data, maintaining their confidentiality; and signed an informed consent.

Acknowledgment

Acknowledgments to our families, their love is essential; to our patients, their truth is our strength; to the hospital authorities, who support our initiatives.

References

1. Parkinson Disease: Practice Essentials, Background, Anatomy. 24 de agosto de (2016). Disponible en
2. Hughes AJ, et al. UK Parkinson`s Disease Society Brain Bank Clinical Diagnosis Criteria. J NeurolNeurosurg Psychiatry 55 (1992): 181-184.
3. Gelb DJ, Oliver E, Gilman S. Diagnosticcriteriaforparkinsondisease. ArchNeurol. 1 de enero de 56 (1999): 33-39.
4. Kim SD, Allen NE, Canning CG, et al. Postural instability in patients with Parkinson's disease. Epidemiology, pathophysiology and management. CNS Drugs. febrero de 27 (2013): 97-112.
5. Approach to the patient with dizziness (2016).
6. Jason JS, Barton JMF. Evaluation of the patient with vertigo. UpToDate. :25
7. Grimbergen YA, Langston JW, Roos RA, et al. Postural instability in Parkinson's disease: the adrenergic hypothesis and the locus coeruleus. Expert Rev Neurother. 1 de febrero de 9 (2009): 279-290.
8. Pogdorny PJ, Suchowersky O, Romanchuk KG, et al. Evidence for small fiber neuropathy in early Parkinson`s disease. Parkinsonism and Related Disorders XXX (2016): 1-6.
9. The specificity and sensitivity of transcranial ultrasound in the differential diagnosis of Parkinson's disease: a prospective blinded study. The Lancet Neurology 7 (2008): 417-424.
10. Rajput AH, Rajput ML, Ferguson LW, et al. Baseline motor findings and Parkinson disease prognostic subtypes. Neurology 89 (2017): 1-6.