Del Chicca M, et al., J Clin Stud Med Case Rep 2024, 11: 238
DOI: 10.24966/CSMC-8801/1000238

HSOA Journal of Clinical Studies and Medical Case Reports

Case Report

Levodopa-Induced Dyskinesias in Corticobasal Degeneration: A Case Report and Review of the Literature

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Abstract

Background

Corticobasal degeneration (CBD) is a neurodegenerative disorder characterized by several clinical features, including progressive asymmetric parkinsonism, dystonia, myoclonus and cortical sensory signs. This clinical presentation is referred to as Corticobasal Syndrome (CBS), considering the diverse underlying pathologies associated CBS.

Levodopa (LD) responsiveness plays a crucial role in diagnosing various parkinsonian conditions, including CBD. CBD is excluded as a diagnosis when there is a "sustained responsiveness to LD" and motor improvements due to LD are typically modest and transient.

Case report and literature review

This article presents a case report of a patient with probable CBS and possible CBD who developed LD-induced oro buccal dyskinesia. A review of the literature reveals the rarity of LD-induced dyskinesia in CBD, with five cases of pathologically confirmed CBD and one clinically diagnosed CBS, all experiencing this phenomenon.

Conclusion

A hypothesis has been proposed to explain the presence, though rare, of LD-induced dyskinesias in CBD: it has been observed that dyskinesias arise from dopaminergic nigrostriatal denervation in the presence of a relatively intact striatum, however the exact mechanism leading to LD-induced dyskinesias in CBD patients remains unknown. Further research is needed to better understand these motor complications, elucidate their pathologic basis in atypical parkinsonism, particularly in CBD and explore potential disease-modifying treatments.

Keywords: Corticobasal degeneration; Corticobasal Syndrome; Levodopa responsiveness; Levodopa-induced dyskinesias

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Citation: Del Chicca M, Del Prete E, Benevento E, Bergamin E, Frosini D, et al. (2024) Levodopa-Induced Dyskinesias in Corticobasal Degeneration: A Case Report and Review of the Literature. J Clin Stud Med Case Rep 11: 238.

Received: May 15, 2024; **Accepted:** May 22, 2024; **Published:** May 30, 2024

Introduction

Corticobasal degeneration (CBD) is a rare neurodegenerative disorder characterized by progressive and asymmetric parkinsonism along with dystonia, myoclonus and cortical sensory signs [1]. This clinical presentation is now referred to Corticobasal Syndrome (CBS) as it has become evident that various underlying pathologies may give rise to this condition. Conversely, also CBD can manifest with different phenotypes beyond CBS.

Levodopa (LD) responsiveness is considered a key feature in published, widely-used clinical diagnostic criteria for several parkinsonian conditions including Parkinson's disease (PD), multiple system atrophy (MSA), progressive supranuclear palsy (PSP) and CBD [2]. Specifically, CBD is excluded as a diagnosis when there is a "*sustained responsiveness to LD*" [1]. In atypical parkinsonism motor improvements due to LD, when present, are generally modest and transient. LD-induced dyskinesias are a common occurrence in PD but they are believed to be less frequent in other disorders within the parkinsonian spectrum, such as PD dementia (PDD) and Dementia with Lewy Bodies (DLB). LD-induced dyskinesias are exceptionally rare in PSP and CBD, even at high LD doses [3]. The variations in dyskinesias frequency between different parkinsonian disorders likely result from multiple factors, including LD dosage, age, treatment duration, follow-up length, and potentially selective neuroanatomic degenerative differences and etiologies.

In this paper, we present the case of a patient with probable CBS and possible CBD that developed LD-induced dyskinesias. Additionally, we found five cases in the literature with pathologically confirmed CBD and one case of clinically diagnosed CBS, all of which presented LD-induced dyskinesias.

Case Presentation

We present the case of a 69-year-old man who initially presented difficulty using his right arm and stiffness, followed by postural instability and dysarthria. The patient had thus undergone a neurological assessment and the diagnostic suspicion of parkinsonism had been raised. A Brain and Cervical Spine Magnetic Resonance Imaging (MRI) were performed without significant findings. A Dopamine

Transporter Single Photon Emission Computed Tomography (DaT-SPECT) analysis did not reveal nigrostriatal degeneration. Three years after the onset of symptoms, he also reported difficulty in fine movements with his left upper limb and he underwent a brain 18FluoroDeoxyGlucose Photon Emission Tomography (18FDG-PET), which indicated mild hypometabolism in the left parieto-occipital cortex and the ipsilateral striatum. In the same year, a diagnosis of Atypical Parkinsonism was made and Pramipexole was introduced, without evident clinical benefits. Subsequently, this drug was discontinued, and treatment with LD at a dose of 300 mg/day, along with Trihexyphenidyl, was initiated, yet without clear clinical efficacy.

The patient's medical history included previous bilateral saphenectomy, knee surgery, and cataract surgery. There was no notable family history for neurological disorders.

The patient was then referred to our clinic, at the Neurology Unit of Pisa Hospital and we visited him four years after symptoms onset. Neurological assessment revealed moderate dysarthria, slowed and hypometric saccades in both horizontal and vertical directions, a "*round the houses*" sign during downward gaze, bilateral palmomental reflex, orobuccofacial and ideomotor apraxia in the left arm. Apraxia could not be assessed in the right upper limb due to severe dystonia; this arm also exhibited stimulus-sensitive myoclonus. While walking, the right upper limb was held abducted with fingers extended in a dystonic posture, and an alien limb phenomenon was present. Postural instability and severe plastic hypertonia were revealed in the right upper limb, along with moderate plastic hypertonia in the other limbs. The patient exhibited bilateral astereoagnosia, agraphesthesia, and digital agnosia.

A follow-up brain MRI was conducted at a higher magnetic field (3 Tesla) and revealed bilateral fronto-parietal atrophy, more pronounced on the left side, and an absence of the physiological hyperintensity of the intermediate portion of the left substantia nigra (Figure 1). Neuropsychological evaluation showed visuoperceptive, executive and planning difficulties. A lumbar puncture yielded normal values of Tau and Beta-amyloid proteins. Consequently, the patient was diagnosed with probable CBS and possible CBD. LD dosage was gradually increased to 800 mg/day, but no improvement in motor symptoms was reported. Neurological evaluation 4 months after LD increase showed a worsening in speech and in gait; the patient presented significant dysartria, hypokinetic gait with severe postural instability and mild to moderate oro buccal dyskinesias and choreoathetotic movements in the left hand, which the patient reported to have started after LD increase. These hyperkinetic movements were consistently reported throughout the day by the caregiver, while the patient was not always aware of them. However, oro buccal dyskinesias were described by the patient as disabling, especially because they resulted in the misalignment of the denture and difficulties with eating and drinking. A gradual reduction of the LD dosage to 600 mg/day, and later to 400 mg/day, was recommended. This reduction did not bring about significant changes in the overall motor function, but it did lead to a substantial decrease in oro buccal dyskinesias and the disappearance of dyskinesia in the left hand. As a result, there was an improvement in the patient's ability to eat and drink comfortably, and the denture remained well- positioned within the oral cavity.

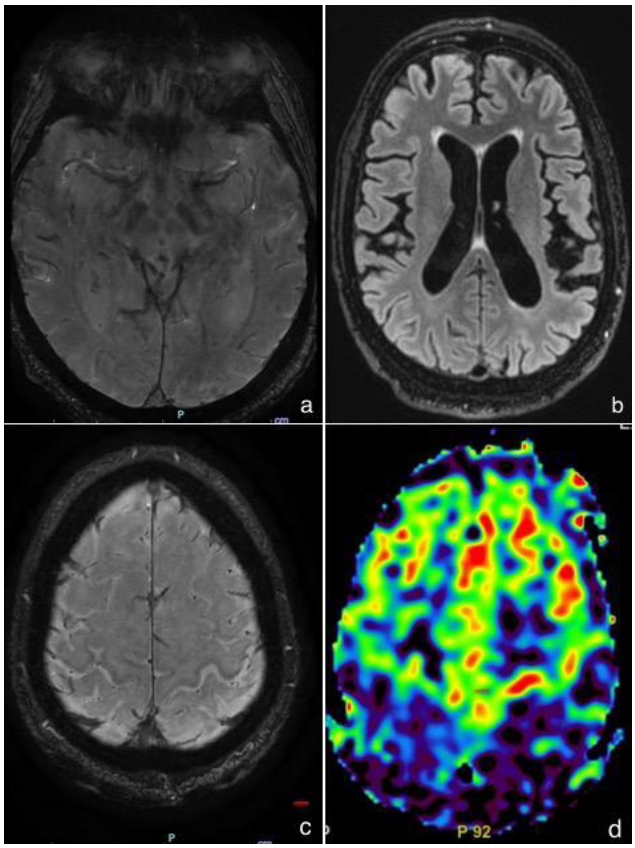

Figure 1: Follow up 3-Tesla brain MRI. a) Axial SWAN sequence showing the absence of the physiological hyperintensity in the intermediate portion of the left substantia nigra, indicating signs of degeneration in the nigrostriatal system at that level, while it remains recognizable on the right side. b) Axial FLAIR sequence highlighting bilateral frontoparietal atrophy, with greater severity on the left side. c) Axial SWAN sequence showing subtle cortical hypointensity in the bilateral primary motor cortex, the anterior post central gyri, and the posterior middle frontal gyri. d) Axial CBF image revealing hypoperfusion in the parietal lobes, notably on the left side.

Abbreviations: MRI: Magnetic Resonance Imaging; **SWAN:** Susceptibility-Weighted Angio graphy; **FLAIR:** Fluid Attenuated Inversion Recovery; **CBF:** Cerebral Blood Flow

Literature review and Discussion

The asymmetric presentation of dystonia, myoclonus associated with ideomotor apraxia and alien limb phenomenon with a history of the disease of at least four years led us to diagnose probable CBS and possible CBD in our patient according to Armstrong et al. diagnostic criteria [1]. Our patient also presented other clinical features typical of CBD including cortical sensory loss (in particular astereoagnosia, agraphesthesia and digital agnosia) and cognitive impairment (especially deficits in executive and visuospatial domains with relatively preserved episodic memory). 3-Tesla brain MRI, carried out later, 4 years after the onset of symptoms, showed asymmetrical fronto- parietal atrophy and pointed out the absence of the physiological hyperintensity of the intermediate portion of the left substantia nigra (Figure 1). At the beginning of the disease, however, DAT imaging did not reveal nigrostriatal degeneration. In degenerative parkinsonism, it has been demonstrated that CBS can present normal DAT imaging during the initial assessment, despite the presence of evident parkinsonism, highlighting the need for clinical follow-up. This may be attributed to a delayed loss of substantia nigra neurons. Therefore, clinical diagnosis should continue to be the primary standard in clinical practice, and

the utilization of imaging should be considered judiciously, following a comprehensive clinical evaluation [4]. In our patients, moreover, we also performed the analysis of CSF revealing normal values of Beta-amyloid and Tau proteins. All these clinical, laboratory and instrumentals findings suggest us that tauopathy is the possible pathogenic mechanism underpinning our patient clinical presentation.

Typically, CBD patients do not improve in motor symptoms when LD treatment is started, with only sporadic and transient improvements reported [2]. In our patient after a progressively increasing the LD dosage up to 800 mg/day, four years after symptoms onset, involuntary movements appeared, including oro buccal dyskinesias and choreoathetotic movements in the left hand. Subsequent neurological evaluation, after the reduction of LD dosage to 600 mg/day and then to 400 mg/day, showed a clear decrease of these involuntary movements also perceived by the patient.

In the literature, five pathologically confirmed CBD cases and one clinically diagnosed CBS case presented LD-induced dyskinesia (Table 1). Unfortunately, in only one case we find detailed clinical and pharmacological description of the patient, in other cases there is a lack of information, such as patient demographics, LD dosages, or changes in involuntary movements following LD dosage adjustments.

Frucht et al. reported a case of a patient with pathologically confirmed CBD who developed mild to moderate dyskinesias after being prescribed a daily dose of 800 mg of LD. These movements were mainly triggered by voluntary actions and included dystonic blinking, neck and torso wiggling, as well as mild choreic movements in the right arm and both legs. They were not coordinated or patterned, indicating they were not simply mirror movements. Dyskinesias improved when the LD dosage was reduced to 600 mg [5].

Ling et al. described a case initially diagnosed as CBS and later confirmed as CBD through pathological examination. This patient developed foot dystonia because of LD treatment. Additionally, they documented two cases initially diagnosed as PSP, pathologically confirmed as CBD, both of which exhibited LD-induced dyskinesias [6]. Unfortunately, the specifics of these involuntary movements, including their type and location, are not available.

Martin et al. reported a patient clinically diagnosed with parkinsonism and later confirmed to have CBD through anatomopathological diagnosis. This patient also experienced LD-induced dyskinesias. However, the exact LD dosage taken by the patient is not known, only that it fell within the range of 450-1500 mg [7]. Detailed information about the type and location of the involuntary movements and whether there were any changes in the clinical presentation, particularly regarding dyskinesias after LD dosage adjustments, are also lacking.

Lastly, Lang et al. described a patient with CBS (we do not know whether CBD was confirmed on post-mortem histopathological examination) who developed disabling large-amplitude, bizarre limb movements, particularly in the arms, after the introduction of LD (the dosage is unspecified). These movements ceased completely when LD was discontinued. It is worth noting that these involuntary movements were reported by family members but not observed by physicians [3].

In PD, deficiency of striatal dopamine is the anatomopathological basis of the parkinsonism which can be improved through dopamine replacement therapy, as the dopamine receptors and regions downstream from the striatum remain intact. Therefore, improvement with LD strongly suggests that the underlying pathology of parkinsonism is the damage to substantia nigra neurons. In atypical parkinsonism the diffusion of pathology beyond the dopaminergic substantia nigra reduces the potential for a positive response to dopaminergic therapy. In the case of CBD, which exhibits more severe and widespread pathology, especially involving neocortical regions, it results in LD-resistant parkinsonism and additional non-parkinsonian and complex clinical features [3].

It is worth noting that a favorable response to LD does not necessarily indicate PD. Mild or modest efficacy for parkinsonian features has been observed in atypical parkinsonism, and also in some cases of CBD. Kompoliti et al. described 147 individuals clinically diagnosed (confirmed by autopsy in only 7) with CBD. Symptomatic improvement with LD was noted in 24% of these CBD cases with a median dose of 300 mg/day. When benefit occurred, it typically involved an improvement in parkinsonism, although isolated cases reported improvements in dystonia or alien limb features. However, benefits were generally modest and transient, and some patients experienced worsened symptoms on these medications [8]. Thus, CBD patients, similarly to those with MSA and PSP, experienced a response to LD, typically modest and often transient, in approximately one-third of patients [2].

Compared to PD, LD-induced dyskinesias, are less common in other disorders within the parkinsonian spectrum, such as PDD and DLB. In a population-based cohort of individuals with parkinsonism, dyskinesias were reported in about 30% of PD patients compared to 12.6% in those with PDD or DLB [9]. One possible reason for the lower occurrence of dyskinesias in DLB and PPD could be the use of lower LD doses in these patients when compared to PD patients. However, there may be other factors beyond lower LD doses in PDD and DLB that contribute to the reduced risk of dyskinesias compared to PD. LD-induced dyskinesias, primarily orofacial in nature, have been reported to occur into up to 27% of patients with MSA, particularly those with a long-term response to LD [10]. LD-induced dyskinesias are exceptionally rare in PSP and CBD, even at high LD doses [3].

It was observed that, unlike PD, which spares the globus pallidus and striatum, these regions in PSP and CBD undergo severe degeneration, limiting their ability to generate dyskinesias despite LD presence [11]. Additionally, cortical hypometabolism in CBD may prevent the development of involuntary movements. The existing model explaining the pathophysiology of LD-induced dyskinesias postulates a sequence of events at the striatal level, encompassing pulsatile stimulation of dopamine receptors, subsequent alterations in the post-synaptic compartment, and irregularities in non-dopaminergic neurotransmitters. These events collectively induce changes in firing patterns and oscillatory activity between the basal ganglia and the motor cortex, resulting in excessive disinhibition of thalamocortical neurons and heightened activation of the motor cortex. Consequently, cortical areas, particularly the primary motor and sensory cortex, along with network dysfunction in these regions, may contribute to the onset of LD-induced dyskinesias. Therefore, degeneration of cortical neurons appears to impede the development of these LD-induced involuntary movements [5].

Author, year	Number of patients	Age/sex	Clinical Diagnosis	Anatomo- pathological diagnosis	Type of involuntary movements	LD (mg)	Follow up after LD reduction/ suspension
Frucht, 2000	1	74/W	Parkinsonism	CBD	Mild to moderate dyskinesias (dystonic blinking, neck and torso wiggling, and mild choreic movements of the right arm and both legs)	800	600 mg provided relief from dyskinesias
Ling, 2010	1	NA	CBS	CBD	Foot dystonia	NA	NA
Ling, 2010	2	NA	PSP	CBD	Dyskinesias not better described	NA	NA
Martin, 2022	1	NA	Parkinsonism	CBD	Dyskinesias not better described	Range 450-1500*	NA
Lang, 2005	1	NA	CBS	NA	Large amplitude bizarre movements of her limbs (particularly the arms)	NA	Involuntary movements resolved completely when LD was withdrawn

Table 1: Case report/case series of LD-induced movement disorder.

*LD range of the CBD group of 8 patients, including the patient who presented LD-induced dyskinesias.

Abbreviations: LD: Levodopa; **NA:** Not applicable; **W:** woman; **CBS:** corticobasal syndrome; **PSP:** progressive sopra-nuclear palsy; **CBD:** corticobasal degeneration.

It is also possible that CBD patients who do not present a rapid response to LD may discontinue it before dyskinesias can be recognized by the patient or physician. Furthermore, due to the limited or absent response to LD, this therapy is often not initiated in patients with CBD or is administered at low doses. For CBD, a specific dose threshold for LD responsiveness has not yet been established.

A hypothesis has been proposed to explain the presence, though rare, of LD-induced dyskinesias in CBD. It has been observed that dyskinesias arise from dopaminergic nigrostriatal denervation in the presence of a relatively intact striatum. In fact, in their original description of the disorder, Reibez et al. noted widespread nigral cell loss in three patients, with the striatum relatively preserved [12].

This pattern has also been reported on PET scans of patients with CBD, particularly early in their illness. In literature, in some reports of CBD, striatal FDG uptake remains preserved, while caudate and putaminal fluorodopa uptake is reduced [13].

In our patient, 18-FDG PET scan revealed hypometabolism in the left cortex and ipsilateral striatum. Previous PET studies have demonstrated that in patients with CBD, there are relative metabolic reductions in several cortical areas and the basal ganglia contralateral to the most affected side, mirroring the findings in our patient. Specifically, the most common hypometabolism pattern associated with CBS includes asymmetrical hypometabolism in the parietal and frontal cortex, thalamus and basal ganglia, while the occipital cortex and cerebellum are typically spared [14].

The presence of such a hypometabolism pattern in our patient on 18-FDG PET, along with the degeneration of the substantia nigra evident in the 3-Tesla brain MRI, contradicts the earlier hypothesis that LD-induced dyskinesias might stem from nigrostriatal denervation in the presence of a relatively intact striatum.

Therefore, the exact mechanism leading to LD-induced dyskinesias in CBD patients remains unknown.

Although rare, recent interest is growing in these motor complications in CBS; Chahine et al. have included these involuntary movements among its clinical features [15].

Atypical parkinsonism can serve as a model for further understanding LD-induced dyskinesias, potentially leading to the identification of more effective treatments. Exploring the mechanisms behind LD-induced dyskinesias can also enhance our comprehension of the functioning of the basal ganglia and their distinct involvement in various neurodegenerative diseases, including synucleinopathies and tauopathies. Nevertheless, larger-samples studies are required to determine the true frequency of LD-induced dyskinesias in atypical parkinsonism, especially in CBD, compared to PD.

To comprehend dyskinesias in CBD patients and forecast LD-induced involuntary movements, efforts should concentrate on delineating LD trial parameters, standardizing assessment methods for involuntary movements, and monitoring responses to LD dosage adjustments.

Declarations

Ethics approval

Approval was obtained from the ethics committee of University of Pisa. The participant's consent was obtained according to the Declaration of Helsinki.

Funding Sources and Conflict of Interest

No specific funding was received for this work. The authors declare that there are no conflicts of interest relevant to this work.

Financial Disclosures for the previous 12 months

The authors declare that there are no additional disclosures to report.

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