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Diagnosis and management of corticobasal syndrome in a phenotypic presentation of Parkinson's Disease

Diagnóstico e manejo da síndrome corticobasal em uma apresentação fenotípica da Doença de Parkinson

Diagnóstico y manejo del síndrome corticobasal en una presentación fenotípica de la Enfermedad de Parkinson

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ABSTRACT

Objective: To report a case of Corticobasal Syndrome (CBS), a rare neurodegenerative disease that is frequently misdiagnosed as other Parkinsonian syndromes, such as Parkinson's Disease (PD). The article highlights the challenges in differential diagnosis and the importance of a multidisciplinary approach. **Case Details:** Male patient, 61 years old, with a history of parkinsonism, cognitive decline, and behavioral changes. He began treatment with levodopa and showed slight motor improvement but continued to experience cognitive deficits. Over the course of follow-up, he developed asymmetric motor symptoms, apraxia, and sleep disturbances. Neuropsychiatric assessments suggested progressive deterioration, leading to the suspicion of CBS. Limited access to advanced neuroimaging within the public health system (SUS) hindered a definitive diagnosis. **Final Considerations:** The case underscores the need for an accurate diagnosis, with access to imaging exams, and for individualized treatment. The limited response to levodopa and rapid progression of symptoms differentiates CBS from other Parkinsonian syndromes, highlighting the importance of a multidisciplinary approach to improve patient quality of life.

Keywords: Parkinsonism, Dementia, Behavioral symptoms.

RESUMO

Objetivo: Relatar um caso de Síndrome Corticobasal (SCB), uma doença neurodegenerativa rara que tem seu diagnostico frequentemente confundido com outros parkinsonismos, como a Doença de Parkinson (DP). O artigo visa destacar os desafios no diagnóstico diferencial e a importância de uma abordagem multidisciplinar. **Detalhamento do caso:** Paciente masculino, 61 anos, com histórico de parkinsonismo e declínio cognitivo, além de alterações comportamentais. Iniciou tratamento com levodopa e apresentou melhora motora discreta, mas continuou com déficits cognitivos. Ao longo do acompanhamento, desenvolveu sintomas motores assimétricos, apraxia e distúrbios do sono. Exames neuropsiquiátricos sugeriram deterioração progressiva, levando à suspeita de SCB. A dificuldade de acesso a neuroimagem avançada no sistema público de saúde (SUS) dificultou o diagnóstico definitivo. **Considerações finais:** O caso reforça a necessidade de um diagnóstico preciso, com acesso a exames de imagem, e de um tratamento individualizado. A resposta limitada à levodopa e a evolução rápida do quadro diferenciam a SCB de outras síndromes parkinsonianas, destacando a importância de uma abordagem multidisciplinar para melhorar a qualidade de vida dos pacientes.

Palavras-chave: Parkinsonismo, Demência, Sintomas de comportamento.

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RESUMEN

Objetivo: Reportar un caso de Síndrome Corticobasal (SBC), una rara enfermedad neurodegenerativa cuyo diagnóstico muchas veces se confunde con otros parkinsonismos, como la Enfermedad de Parkinson (EP). El artículo tiene como objetivo resaltar los desafíos del diagnóstico diferencial y la importancia de un enfoque multidisciplinario. **Detalles del caso:** Paciente masculino, 61 años, con antecedentes de parkinsonismo y deterioro cognitivo, además de cambios conductuales. Inició tratamiento con levodopa y presentó ligera mejoría motora, pero continuó con déficit cognitivo. Durante el seguimiento, desarrolló síntomas motores asimétricos, apraxia y trastornos del sueño. Los exámenes neuropsiquiátricos sugirieron un deterioro progresivo, lo que llevó a sospechar de SCB. La dificultad de acceso a neuroimagen avanzada en el sistema público de salud (SUS) dificultó el diagnóstico definitivo. **Consideraciones finales:** El caso refuerza la necesidad de un diagnóstico certero, con acceso a exámenes de imagen y tratamiento individualizado. La respuesta limitada a la levodopa y la rápida evolución de la afección diferencian al CBS de otros síndromes parkinsonianos, destacando la importancia de un enfoque multidisciplinario para mejorar la calidad de vida de los pacientes.

Palabras clave: Parkinsonismo, Demencia, Síntomas conductuales.

INTRODUCTION

Parkinsonism is a heterogeneous group of neurological disorders that share common features such as bradykinesia, muscle rigidity, resting tremors, and postural instability. Among the diseases in which parkinsonism is evident, Parkinson's disease (PD) is the most common, followed by less prevalent conditions such as progressive supranuclear palsy (PSP), Dementia with Lewy Bodies (DLB), and corticobasal syndrome (CBS), among others. Each of these conditions has distinct characteristics in terms of symptomatology, disease progression, and response to treatment, representing a significant challenge for differential diagnosis and effective clinical management (UTIUMI MA, et al., 2012; KO T, et al., 2021).

PD is typically characterized by the onset of motor symptoms, with patients generally developing significant cognitive complications after a decade or more. Approximately 40% of patients with PD develop dementia around 10 to 15 years after the initial diagnosis, one of the most severe and significant complications of the disease. This fact underlines the importance of longitudinally monitoring the progression of patients with PD (MELO PHM, et al., 2023).

In contrast to PD, atypical parkinsonism, such as CBS, frequently exhibit cognitive and behavioral symptoms that precede motor symptoms. CBS is particularly notable due to its association with tau protein deposition in the brain, distinct from the predominant accumulation of synuclein in PD. Patients with CBS may present a varied range of neurological symptoms, including apraxia, rigidity, dystonia, and the alien limb phenomenon, where limbs may act as if they have a will of their own (CAMPOS AC, et al., 2023; OLIVEIRA LM, et al., 2016; PARMERA JB, et al., 2016).

Epidemiologically, while PD accounts for approximately 75% of Parkinsonism cases, conditions such as CBS, PSP, and MSA make up a smaller fraction of diagnoses—about 25%. CBS has an estimated prevalence in patients ranging from 4.9 to 7.3 cases per 100,000 people, with an incidence of less than 5 cases per 100,000 inhabitants, highlighting the need for clinical awareness and specialization to ensure proper recognition and management (VAN AGT TFA, et al., 2022; OLIVEIRA RV, PEREIRA JS, 2017; PEDROSO JL, et al., 2023). From a therapeutic standpoint, levodopa is the standard treatment for Parkinson's disease (PD), acting as a metabolic precursor that converts into dopamine in the central nervous system. This conversion is essential to supplement dopamine, whose production is reduced due to the progressive degeneration of dopaminergic neurons in the substantia nigra, a crucial brain region for motor control. Levodopa administration significantly improves motor symptoms in most PD patients, reflecting the predominantly dopaminergic pathological basis of this disease (DUNCAN W, et al., 2021). In contrast, in atypical parkinsonism such as corticobasal syndrome (CBS), the response to levodopa is generally minimal. CBS is characterized not only by the loss of dopaminergic neurons but also by the extensive accumulation of pathological tau protein in cortical and subcortical regions (PAMERA JB, et al., 2022). These pathological changes involve multiple

neuronal systems beyond the dopaminergic ones, limiting the effectiveness of levodopa since the therapy does not address the diverse neurodegenerative components present in CBS. Thus, the limited response to levodopa in CBS cases reveals substantial differences in the neuropathological pathways between CBS and PD (CONSTANTINIDES VC, et al., 2019).

The differential diagnosis of various forms of parkinsonism requires rigorous clinical evaluation, complemented by advanced neuroimaging techniques such as positron emission tomography (PET) and single-photon emission computed tomography (SPECT). These tools are fundamental for visualizing specific neuroanatomical and functional changes, providing essential data for accurate diagnosis (SMITH J, et al., 2015). Histopathological confirmation, when accessible, is considered the gold standard, directly identifying pathological changes such as tau and alpha-synuclein protein deposits.

Managing patients with CBS requires a multidisciplinary approach to address motor and nonmotor complications, including cognitive and behavioral disorders, as well as autonomic and musculoskeletal symptoms (CONSTANTINIDES VC, et al., 2019; PARMERA JB, et al., 2022). This article aims to discuss a clinical case of CBS, illustrating not only the complexity of diagnosis and management of these patients but also highlighting the importance of a multidisciplinary approach that includes geriatricians or neurologists, physical therapists, occupational therapists, and mental health professionals, essential to maximize quality of life and minimize the consequences of the rapid progression of these neurodegenerative conditions (CAMPOS AC, et al., 2023; OLIVEIRA LM, et al., 2016; PARMERA JB, et al., 2016). This is a case study assessed and approved by the Research Ethics Committee (CEP) (Opinion 7.476.715 and CAAE 86007024.2.0000.5149).

CASE REPORT

A 61-year-old male patient with a history of femoral neck fracture in 2006, chronic alcoholism until 2020, and smoking until 2006 (76 pack-years) presented at a Primary Health care in Belo Horizonte in June 2022, reporting a 6-month history of memory loss and dizziness, along with episodes of falling from standing height. Additionally, he also reported mood changes, fluctuating from agitation to psychomotor slowing, associated with constant nightmares. Upon investigation, his companion reported significant cognitive and physical impairments affecting his ability to perform both basic and advanced activities of daily living (ADLs), marked by depression symptoms and motor slowing, which began in 2018 and had progressively worsened. In that year, he also began to experience insomnia, irritability and aggressive behavior, which led to his fired from work.

During the consultation, upper limb rigidity and difficulty walking were noted, initially attributed to the previous femoral fracture. Physical examination revealed bradykinesia, cogwheel rigidity in the upper limbs, and a shuffling gait, without tremors. A provisional diagnosis of Parkinsonian syndrome was made, and he was prescribed Levodopa 250mg + Carbidopa 50 mg, 1/4 tablet four times daily, and referred to neurology.

In March 2023, the patient developed tremors, with progressive worsening. His companion reported that he frequently talked to himself about past experiences and uttered phrases out of context, with a lack of response to external stimuli and failure to recognize familiar people. He remained unable to perform ADLs, with progressively impaired social interaction, hearing loss, and lack of appetite. Family history revealed Parkinson's disease (father). In the Mini-Mental State Examination (MMSE), he showed orientation in time and space but had deficits in attention, calculation, recall memory, language, and visuospatial skills. Pfeffer score: 26.

Parkinsonism persisted, and additional hypotheses of dementia to be clarified and major depression were considered. An increase in Levodopa + Carbidopa (250 mg/50 mg) to 3/4 tablet three times daily and discontinuation of carbamazepine were made. In April 2023, the patient reported progressive improvement in tremors and mood, though memory worsened. Random chewing movements were observed. Physical examination showed mild bilateral cogwheel rigidity. MMSE revealed deficits in attention, calculation, recall memory, language, and visuospatial skills, with a total score of 16 points. Pfeffer score: 30. Diagnosis included major depression, parkinsonism, and undetermined dementia.

In August 2023, insomnia, sleep agitation, forgetfulness, and inability to perform ADLs persisted. His appetite improved, tremors decreased, he had intense dreams with verbalization at night, and continued difficulty moving. Physical examination showed a typical, slightly slowed gait with preserved balance. MMSE: 22 points, with deficits in calculation, recall memory, and visuospatial skills. The differential diagnosis was well controlled parkinsonism, dementia with preserved orientation, and major depression. The patient reported unchanged sleep latency of two hours and agitation. Treatment adjustments included increasing Levodopa + Benserazide to 1 tablet three times daily. Treatment included prescribing Donepezil 5 mg at midday, maintaining Levodopa/Benserazide (200/50) mg 1 tablet three times daily. Physical examination revealed a patient with low mood, quiet, fixed gaze on the floor, bradykinesia, rigidity, gait with little arm movement, block turning, and rigidity in the upper limbs. MMSE: 23 points.

To assist with neuropsychiatric complaints related to depressive symptoms, the patient was initially prescribed nortriptyline, followed by fluoxetine at a dose of 20 mg daily, as it was available in the SUS network. Escitalopram was later introduced as a substitute, as it was better accepted by the patient and more effective in managing symptoms. Regarding mood complaints and the patient's sleep quality, medication interventions were made. Melatonin was initially prescribed but did not yield a good response. Subsequently, clonazepam, obtained from the spouse, was self-administered with a moderate response, and the dosage was adjusted to 8 drops. Ultimately, clonazepam was replaced by ramelteon at a dose of 8 mg, with a positive response in managing initial insomnia. He noted improvement in memory and mood with Donepezil. Moreover, he showed improvement in ADLs, being able to prepare meals independently, although he remained unable to perform basic self-care, such as dressing or bathing, without assistance due to physical limitations from shoulder arthralgia. He reported partial improvement in rigidity and constant jaw movements. Physical examination indicated difficulty walking due to hip joint wear, bradykinesia, asymmetric rigidity, tongue dyskinesia, slowed gait, limited arm swing (especially on the left side), block turning, limb asymmetry, with hyperreflexia in the left lower limb and right upper limb. Based on these findings, corticobasal syndrome. Then he was referred to neurology for sedated MRI.

DISCUSSION

Corticobasal Syndrome (CBS) is a rare, progressive neurodegenerative disease characterized by a combination of cognitive and motor symptoms. These symptoms result from the progressive degeneration of the cerebral cortex and subcortical structures, with emphasis on the basal ganglia. CBS is classified among atypical parkinsonian syndromes, marked by the presence of parkinsonism, which includes bradykinesia associated with resting tremor, rigidity, or postural instability (ARMSTRONG MJ, et al. 2013). The prevalence is low, affecting fewer than 5 individuals per 100,000 population, and its life expectancy ranges from 2.5 to 12.5 years after diagnosis, with a usual onset between 50 and 70 years of age (CONSTANTINIDES VC, et al., 2019). Major differential diagnoses include Parkinson's Disease (PD), Dementia with Lewy Bodies (DLB), and Progressive Supranuclear Palsy (PSP).

The terms Corticobasal Syndrome (CBS) and Corticobasal Degeneration (CBD) designate distinct entities; while the former refers to the clinical phenotype, the latter, diagnosed exclusively through post-mortem neuropathological analysis (ALMEIDA IJ, et al., 2020), is considered a pathological entity, with an estimated impact showing that, of patients with clinical symptoms of CBS, up to 50% have a post-mortem diagnosis of CBD, although CBD is also found in patients with diagnoses of other syndromes (GRIJALVO-PEREZ AM, LITVAN I, 2014; PARMERA JB, et al., 2016).

Armstrong MJ, et al., (2013) define the diagnostic criteria for CBS, as exemplified in **Table 1**. According to these foundations, "probable CBS" is determined when there is bilateral asymmetry and at least two of the following motor symptoms: (A) limb rigidity or akinesia; (B) limb dystonia; (C) limb myoclonus; in association with at least two of the following symptoms: (D) Oro buccolingual or limb apraxia; (E) cortical sensory deficits; (F) alien hand phenomenon (ALMEIDA IJ, et al., 2020). Additionally, possible Corticobasal Syndrome, is characterized by the presentation of the same symptoms, requiring one among (a), (b), and (c), either symmetrical or asymmetrical, along with one of the symptoms described in (d), (e), or (f).

Table 1 - Proposed clinical phenotypes (syndromes) associated with the pathology of corticobasal degeneration (adapted).

Syndrome	Features
Probable corticobasal Syndrome	Asymmetric presentation of 2 of: • (A) Limb rigidity or akinesia • (B) Limb dystonia • (C) Limb myoclonus Plus 2 of: • (D) Oro buccal or limb apraxia • (E) Cortical sensory deficit • (F) Alien limb phenomena (more than a simple levitation)
Possible corticobasal Syndrome	May be symmetric: presentation 1 of: • (a) Limb rigidity or akinesia • (b) Limb dystonia • (c) Limb myoclonus Plus 1 of: • (d) Oro buccal or limb apraxia • (e) Cortical sensory deficit • (f) Alien limb phenomena (more than a simple levitation)

Source: Armstrong, M. J., et al. Criteria for the diagnosis of corticobasal degeneration, *Neurology*, 2013, 80.

In the speech profile of a patient with parkinsonism, there is impairment of phonation and word articulation, constituting a type of dysarthria known as hypokinetic dysarthria. In this dysarthria, the reduction in speech volume is prominent, which may be limited to small whispers, along with decreased or lost voice inflection capacity, making the speech monotonal, and rhythm disturbances, characterized by episodes of initial hesitation and a slowed cadence, which may have inappropriate pauses or involuntary accelerations. The decline in patient speaking ability, beyond cognitive factors, was marked by the presence of a repetitive, chronic mandibular movement, impairing the coordination needed for phonetic-lexical articulation and hesitation in initiating conversations.

The patient, presented with bradykinesia, muscle rigidity with marked asymmetry, showing greater unilateral intensity on the right side of the body, and a block gait. Additionally, significant cognitive impairments were observed, including deficits in attention, memory, visuospatial functions, and language, which are commonly associated with dysfunction of the parietal lobe and frontal cortex, regions affected in CBS (LING H, et al., 2010). These clinical findings are consistent with the diagnostic criteria established by Armstrong et al., particularly the presence of asymmetric rigidity and apraxia, along with progressive cognitive impairment, as indicators for diagnosing the patient in the probable CBS category (ARMSTRONG MJ, et al., 2013).

In Corticobasal Syndrome (CBS), voluntary action disorders, such as alien limb syndrome and apraxia, may be central to most diagnostic criteria (GRIJALVO-PEREZ AM, LITVAN I, 2014). Alien limb syndrome, although not identified in this patient, involves involuntary and semi purposeful movements, resulting from lesions in areas such as the posterior parietal cortex and the supplementary motor area (PARMERA JB, et al., 2016). For this patient, apraxia was evident in the impairment of performing complex daily motor tasks, reflecting cortical dysfunctions typical of CBS. These symptoms indicate lesions in cortical areas involved in motor planning and control, aligning with the expected clinical profile in CBS (PARMERA JB, et al., 2016; GRIJALVO-PEREZ AM, LITVAN I, 2014). The asymmetry of rigidity, with a predominant onset on one side of the body, is a common feature in PD and CBS, setting it apart from other parkinsonisms, such as Multiple System Atrophy (MSA), Progressive Supranuclear Palsy (PSP) and Vascular Parkinsonism. However, PD is more robustly described in the literature evidence as exhibiting motor decline preceding the onset of cognitive impairment symptoms (PARMERA JB, et al., 2016). This pattern is primarily due to the degeneration of dopaminergic neurons in the substantia nigra, which initially impacts motor control. Only with PD progression do cognitive functions begin to be compromised, often leading to deficits in executive function, memory, and attention (DAG AARSLAND, et al., 2021). In contrast, in Corticobasal Syndrome (CBS), cognitive symptoms tend to appear before motor alterations.

This is due to the underlying pathology that initially affects cortical brain areas responsible for complex cognitive functions, such as behavior and personality (ZHANG Q, et al., 2020). Therefore, the temporal onset of symptoms is crucial for the differential diagnosis between Corticobasal Syndrome (CBS) and Parkinson's Disease (PD). In the patient's case, cognitive factors, particularly personality changes, were reported by his wife as conflicts at work due to exacerbated aggressive behavior, which ultimately led to his work suspension. In this context, it is suggested that cognitive, behavioral, and personality alterations manifested before motor limitations and dysfunctions, highlighting the complexity of diagnosing CBS.

Moreover, given the symptom phenotype overlap among of clinical features of parkinsonian disorders, such as CBS, Lewy Body Disease (LBD), and PSP, differentia diagnosis becomes imperative. In this case, the absence of visual hallucinations and cognitive fluctuations common in LBD (TATSCH MF, et al., 2002), along with the lack of typical PSP oculomotor dysfunction, such as vertical gaze palsy (LITVAN I, et al., 1996), supports the diagnosis of CBS. In addition, the pattern of asymmetric rigidity and the presence of apraxia observed in the patient are more frequently reported in diagnostic descriptions of CBS cases, further indicating CBS as the probable diagnosis. These findings contrast with LBD, where psychiatric symptoms predominate, and with PSP, which presents more symmetric and early parkinsonism (LITVAN I, et al., 1996).

The introduction of levodopa, a dopamine precursor, in PD treatment by Cotzias and colleagues in 1967 represented a considerable improvement in the quality of life for Parkinsonian patients. Although the therapeutic effect of levodopa in Parkinson's Disease (PD) is well-documented in the literature, comparison between CBS and other neurodegenerative diseases, such as LBD and PSP, reveals that, for LBD and PSP, the response to levodopa tends to be absent or merely transient. In these cases, limb rigidity may progress to a global involvement often described as "lead-pipe" rigidity, with or without the "cogwheel" phenomenon (GRIJALVO-PEREZ AM, LITVAN I, 2014). Other studies indicate an excellent response in PD and a moderate response in CBS (PARMERA JB, et al., 2016).

The administration of levodopa + carbidopa promoted a marked improvement in the patient’s motor symptoms, including mitigation of resting tremor, reduction of asymmetric rigidity, and improvement of gait disturbances. Prior to treatment, gait was characterized by hesitation, small steps, episodes of freezing, and involuntary acceleration to maintain balance, particularly due to reduced mobility of the right lower limb. After pharmacological intervention, increased joint fluidity, greater range of motion on the right side, and reduction of compensatory contralateral movements were observed. These changes contributed significantly to the recovery of postural control and the performance of automatic and daily tasks, such as walking, feeding, dressing, and personal hygiene, improving the patient’s functional independence.

Positron emission tomography (PET) allows the study of dopaminergic activity at the striatal level, serving as a valuable tool for detecting dopaminergic insufficiency and identifying variants or degeneration areas in patients with Corticobasal Syndrome (CBS) (PARMERA JB, et al., 2016). However, access to this technology is restricted within the Sistema Único de Saúde (SUS) in Belo Horizonte, MG. This poses a significant barrier to the accurate and early diagnosis of neurodegenerative conditions. In the patient’s case, a PET scan was not performed, even though its usefulness is well-documented in the literature for differentiating CBS from other pathologies (PARMERA JB, et al. 2016). Additionally, citing claustrophobia, the patient refused to undergo cranial magnetic resonance imaging (MRI) without sedation, further complicating diagnostic accuracy.

Among neuropsychiatric symptoms, the most common neurodegenerative diseases include depression, apathy, irritability, and sleep disturbances, with depression being the most prevalent (GRIJALVO-PEREZ AM, et al., 2014). These phenomena are closely related to dynamic factors of cortical anatomical degeneration. However, the relationship between neuropsychiatric symptoms and anatomical brain changes still requires further study to clarify their interrelations. Despite this gap, it is known that these symptoms predispose individuals to the development of psychosomatic comorbidities (PARMERA JB, et al., 2016).

In summary, this case reinforces the need for precise diagnosis and a multidisciplinary approach to maximize the quality of life for patients with Corticobasal Syndrome, providing individualized care for motor, cognitive, and behavioral symptoms. In this case report, the diagnosis would be that of possible Corticobasal

Syndrome. Personalized therapeutic strategies and psychological support are essential to slow disease progression and improve quality of life (VAN AGT, et al., 2022; PEDROSO JL, et al., 2023; DUNCAN W, et al., 2021). Therefore, the diagnostic limitations encountered were due to the difficulty of imaging studies, which remain limited in the SUS network or, as with PET scans, are restricted to specific scientific research situations.

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