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Association between Vitamin D deficiency and Parkinson's disease: systematic review of the evidence on therapeutic potential and causal relationship

Associação entre deficiência de Vitamina D e doença de Parkinson: revisão sistemática das evidências sobre potencial terapêutico e relação causal

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ABSTRACT

Introduction: Parkinson's disease (PD) is a neurodegenerative disorder characterized by the loss of dopaminergic neurons in the substantia nigra and the formation of Lewy bodies. Genetic mutations and oxidative stress contribute to its progression. Vitamin D may play a neuroprotective role in PD, but its therapeutic potential remains uncertain.

Objective: This review aims to assess the relationship between vitamin D deficiency and the development, prognosis, and therapeutic potential in PD.

Method: A systematic review was conducted using the descriptors "vitamin D" and "Parkinson's disease" in the PubMed and MedLine databases. Fourteen studies published in the last 5 years, focusing on human clinical and epidemiological data, were selected.

Result: The studies suggest that low levels of 25(OH)D increase the risk of PD, although there is no association with symptom severity. Vitamin D influences neurotrophic factors, aiding in the maintenance of dopaminergic neurons and improving tissue microcirculation. The neuroprotective role of vitamin D includes the regulation of neurotrophic factors and the protection of neurons against oxidative stress.

Conclusion: Although there is no consensus on the therapeutic role of vitamin D in PD, evidence suggests its influence on the development of the disease. Investigating vitamin D levels at the onset of PD is crucial, especially to prevent bone density loss and fall-related complications.

KEYWORDS: Vitamin D deficiency. Parkinson's disease. Therapy.

Central Message

Parkinson's disease (PD) involves loss of dopaminergic neurons, and vitamin D deficiency may increase its risk. Although there is no consensus on its therapeutic role, evidence suggests that low vitamin D levels influence the development of PD.

Perspective

Vitamin D deficiency appears to be linked to an increased risk of Parkinson's disease, but its therapeutic potential is still uncertain. Further studies are needed to clarify its real influence in the treatment of PD.

RESUMO

Introdução: A doença de Parkinson (DP) é doença neurodegenerativa caracterizada pela perda de neurônios dopaminérgicos na substância negra e pela formação de corpos de Lewy. Mutações genéticas e estresse oxidativo contribuem para sua progressão. A vitamina D pode desempenhar um papel neuroprotetor na DP, mas seu potencial terapêutico permanece incerto.

Objetivo: Esta revisão tem como objetivo avaliar a relação entre a deficiência de vitamina D e o desenvolvimento, prognóstico e potencial terapêutico na DP.

Método: Foi realizada uma revisão sistemática utilizando os descritores "vitamina D" e "doença de Parkinson" nas bases de dados PubMed e MedLine. Quatorze estudos publicados nos últimos 5 anos, com foco em dados clínicos e epidemiológicos humanos, foram selecionados.

Resultado: Os estudos sugerem que baixos níveis de 25(OH)D aumentam o risco de DP, embora não haja associação com a gravidade dos sintomas. A vitamina D influencia os fatores neurotróficos, auxiliando na manutenção dos neurônios dopaminérgicos e melhorando a microcirculação tecidual. O papel neuroprotetor da vitamina D inclui a regulação de fatores neurotróficos e a proteção dos neurônios contra o estresse oxidativo.

Conclusão: Embora não haja consenso sobre o papel terapêutico da vitamina D na DP, evidências sugerem sua influência no desenvolvimento da doença. Investigar os níveis de vitamina D no início da DP é crucial, especialmente para prevenir a perda de densidade óssea e complicações relacionadas a quedas.

PALAVRAS-CHAVE: Deficiência de vitamina D. Doença de Parkinson. Terapia.

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INTRODUCTION

Parkinson's disease (PD) is a neurodegenerative disease characterized by the reduction of dopaminergic neurons present in the substantia nigra of the midbrain and the deposition of alpha-synuclein proteins in the cytoplasm of neurons, thus forming Lewy bodies. Although it is an idiopathic condition, it is noted that mutations in genes responsible for the production of alpha-synuclein, LRRK2, parkin, and PINK1 proteins trigger mitochondrial changes which, consequently, cause oxidative stress in neuronal tissue, with a subsequent increase in reactive oxygen species, thus contributing to the progression of PD.[1,2]

The corresponding neuromotor dysfunction is multifactorial, being mainly associated with genetic predisposition, advanced age, male gender, use of medications, exposure to chemicals, and smoking. Clinical manifestations arise with the progression of the disease, when the majority of dopaminergic neurons have been damaged, being manifested by motor symptoms — tremors, bradykinesia, postural instability, and rigidity — and non-motor symptoms — cognitive decline, dysfunction of the autonomic nervous system, psychiatric disorders, and sleep disorders.[3]

PD management is multidisciplinary and must consider the clinical stage of the pathology, symptoms, and the individuality of the patient. The available and commonly prescribed pharmacological options are levodopa, MAO-B inhibitors, glutamate pathway antagonists, catechol-O-methyltransferase (COMT) inhibitors, and non-ergot dopaminergic agonists.[3]

The numerous advances related to knowledge of the pathophysiology and therapy of PD are notable. Some studies show a possible association between vitamin D deficiency and this disease. The corresponding vitamin can be obtained through diet or sun exposure, requiring a series of reactions to activate. The first step takes place in the liver and involves the hydroxylation of cholecalciferol (D3) or ergocalciferol (D2), resulting in the formation of 25-hydroxyvitamin D. Subsequently, the same process takes place in the kidneys, but to generate 1,25-hydroxyvitamin D, the active form of vitamin D, which is capable of binding to the specific receptor and carrying out its actions. It is worth noting that this vitamin also has receptors in various brain regions, thus exerting a neuroprotective function by inhibiting the activation of microglia and, in this way, protecting dopaminergic neurons and increasing the neurotrophic factor expression. Despite the existence of some divergences regarding this association, there is some evidence of the influence of hypovitaminosis on the individual's prognosis, mainly in the maintenance of postural instability and impairment of motor condition.[4]

Therefore, to clarify the role played by vitamin D in development, prognosis, and therapeutic potential, this paper summarize the main scientific data that allow critical evaluation and understanding of this possible association.

METHOD

The method used in this systematic review has as its starting point the formulation of the following research question: What is the relationship between vitamin D and Parkinson's disease? To address this question, the descriptors "vitamin D" and "Parkinson's disease" were used during the bibliographic search. The search was conducted in the VHL (MedLine) and PubMed databases, following specific selection criteria. Were included studies with full text available in Portuguese and English, published in the last 5 years, and which focused on clinical, epidemiological, and observational approaches, all aimed at exploring the relationship between vitamin D and Parkinson's disease in humans. In vitro and animal studies and those that expanded the scope to other neurodegenerative diseases were excluded, to maintain the focus on the proposed theme. Data extraction covered essential information, such as author, year of publication, title, objectives, methods, sample size, results, and conclusions. The methodological quality of the selected studies was carefully evaluated. In the initial search, 84 results were identified in PubMed and 142 in MedLine. Of these, 9 and 5, respectively, were selected for analysis, following the previously established inclusion criteria, totaling 14 articles for the present review (Table).[5-17]

RESULT

Metadata and content analysis of articles (n = 14) (Tabla)

DISCUSSION

Vitamin D plays a significant role in synaptic plasticity by modulating the neurotrophic factors responsible for the process of neuronal proliferation and differentiation, such as glial cell-derived growth factor (GDNF) and neural growth factor (NGF), and these mediators are fundamental for the maintenance, respectively, of dopaminergic and cholinergic neurons. These factors exert neuroprotection by interfering with the activity of glutamyl transpeptidase gamma and free radicals produced by reactive oxygen species (ROS) and nitric oxide. Furthermore, it is known that the human brain has numerous receptors for vitamin D (VDR), mainly in the hypothalamus and neurons of the substantia nigra. This binding structure is responsible for mediating the biological effects of ^{1,25}(OH), highlighting the importance of this substance in the cognitive functions of individuals and, consequently, in the development of neurodegenerative diseases, such as PD.[5-9]

From this perspective, several studies have highlighted the relationship between vitamin D levels and the development of PD; however, there has yet to be a consensus on the mechanism that triggers this interference. Some authors have demonstrated the influence of abnormal levels of this vitamin in increasing the amount of ultrasensitive C-reactive protein (hs-CRP), with an indirect action of vitamin D being perceived

TABLE — Metadata and content analysis of articles (n = 14)5-17

Authors	Title	Journal	Objectives	Conclusion
de Siqueira et al.[5]	Vitamin D3 actions on astrocyte cells: a target for therapeutic strategy in Parkinson's disease?		Investigate alterations in the vitamin D pathway in astrocytes and neurons and their correlation with αsynuclein aggregates in human brains obtained from patients with PD and healthy control patients.	The presence of CYP27B1 astrocytes distinguishes PD patients and suggests their contribution to protecting neurons and improving neuropathological features.
Redenšek et al.[6]	Genetic variability of the vitamin d receptor affects susceptibility to Parkinson's disease and dopaminergic treatment adverse events	Frontiers in Aging Neuroscience	Investigate the association of VDR (vitamin D receptor) genetic variability with the occurrence of adverse events (AEs) from treatment with levodopa and dopamine agonists (DAs) and evaluate whether VDR genetic variability influences the daily dose requirements of dopaminergic treatment necessary to adequately control PD symptoms in an individual patient. Additionally, assess the association between VDR SNPs and the risk of PD in a Slovenian cohort of PD patients.	The study may support a personalized approach to PD treatment, especially in terms of monitoring vitamin D levels and vitamin D supplementation in patients with high-risk VDR genotypes.
Ogura et al.[7]	Circulatory 25(OH)D and 1,25(OH)2D as differential biomarkers between multiple system atrophy and Parkinson's disease patients	eNeurologicalSci	This study aimed to evaluate whether serum 25(OH)D and 1,25(OH)2D can be used as biomarkers to differentiate healthy subjects (HS), multiple system atrophy (MSA), and Parkinson's disease (PD) in patients of both sexes.	Serum 25(OH)D and 1,25(OH)2D could be used as biomarkers for MSA and PD. 25(OH)D and H&Y provided the best sensitivity and group classification by characteristics.
Lien et al.[8]	Correlation between hypovitaminosis D and nutritional status with the severity of clinical symptoms and impaired cognitive function in patients with Parkinson's disease.	Acta Neurologica Taiwan	To evaluate the relationship between the severity of clinical symptoms and cognitive function in patients with Parkinson's disease (PD) and their serum vitamin D levels and nutritional status.	It was revealed that PD patients at risk of malnutrition have impaired cognitive function, but patients with abnormal serum vitamin D levels did not show such an influence. However, PD patients with abnormal vitamin D levels have higher levels of hs-CRP, which affects the cognitive function of PD patients. Therefore, abnormal serum vitamin D levels may have an indirect influence on the cognitive function of PD patients through their effect on hs-CRP levels.
Lv et al.[9]	Assessing the effects of vitamin D on neural network function in patients with Parkinson's disease by measuring the fraction amplitude of low-frequency fluctuation.	Frontiers in Aging Neuroscience	To explore the relationship between Parkinson's disease (PD) levels and vitamin D (VD), as well as to analyze the effects of VD on spontaneous brain activity and investigate the possible mechanisms of its involvement in the risk of PD.	Patients with Parkinson's disease (PD) exhibited lower serum levels of vitamin D (VD) compared to the healthy control group, and VD may have a potential dose-dependent effect on the risk of PD. Lower serum levels of VD may affect spontaneous network neuronal activity in the default mode and visual pathway neurons in PD patients, providing a possible mechanism for its effect on the risk of PD.
Ozturk et al.[10]	Bone mineral density and serum vitamin D status in Parkinson's disease: Are the stage and clinical features of the disease important?	Neurology India	To evaluate the relationship between bone mineral density (BMD) and serum vitamin D levels and the stage or clinical characteristics of Parkinson's disease (PD).	All patients with Parkinson's disease (PD) should be screened for the development of osteoporosis and for sufficient vitamin D levels in the early stages of the disease, as these are related. Preventive methods for bone health should be considered at the onset of PD.
Hiller et al.[11]	A randomized, controlled pilot study of the effects of vitamin D supplementation on balance in Parkinson's disease: Does age matter?	Public Library of Science One, PLoS 1	To explore whether short-term supplementation of high doses of vitamin D is safe and improves balance in individuals with Parkinson's disease (PD).	High-dose vitamin D supplementation in the short term appears to be safe in individuals with Parkinson's disease (PD), but it did not significantly improve balance as measured by the Sensory Organization Test in this pilot study population. A post hoc analysis suggests that vitamin D may have potential to improve balance in a younger population with PD. High-dose vitamin D supplementation in PD requires further studies, especially in light of new research suggesting that megadoses and even moderate doses (as low as 4,000 IU per day) may increase falls in older populations.
Al-Amin et al.[12]	Vitamin D deficiency is associated with reduced hippocampal volume and disrupted structural connectivity in patients with mild cognitive impairment	Human Brain Mapping	Mild cognitive impairment (MCI) is prodromal to neurocognitive disorders, and neuroimaging studies suggest that, in older adults, this cognitive impairment is associated with a reduction in hippocampal volume and structural integrity of white matter. To test whether vitamin D is associated with the neuroanatomical correlates of mild cognitive impairment (MCI), the study analyzed an existing dataset of structural and diffusion magnetic resonance imaging from elderly patients with MCI.	Low vitamin D levels are associated with reduced volumes of hippocampal subfields and connection deficits in older adults with MCI, which may exacerbate neurocognitive outcomes. Longitudinal studies are now needed to determine whether vitamin D can serve as a biomarker for Alzheimer's disease and whether intervention can prevent the progression from MCI to severe cognitive disorders.
Kuhn, Karp e Muller[13]	No vitamin D deficiency in patients with Parkinson's disease.	Degenerative neurological and neuromuscular disease		
Bariche Lv et al.[14]	Clinical correlates of serum 25-hydroxyvitamin D in Parkinson disease	Nutritional Neuroscience	A decrease in vitamin D levels has already been reported in patients with Parkinson's disease, along with correlations to the clinical severity of the disease. This case-control study found higher but not statistically significant plasma levels of 25-OH-vitamin D in patients with Parkinson's disease compared to age- and sex-matched controls, with no association found with clinical parameters such as disease severity scores or cognitive function assessments..	Our results warrant further confirmatory research with a strict matching design between patients and controls, which was not implemented in previous investigations. We emphasize that this case-control study does not allow for any comments on the supposed beneficial effects of vitamin D supplementation, such as on bone mass or bone mineral density, in patients with Parkinson's disease.
Zhang et al.[15]	Relationship between 25-hydroxyvitamin D, bone density, and Parkinson's disease symptoms.	Acta Neurologica Scandinavica	A cross-sectional observational study to investigate whether previous studies were correct in suggesting a negative association between 25(OH)D levels and clinical characteristics of Parkinson's disease (PD), as the data are inconsistent.	Serum 25(OH)D levels were negatively correlated with disease severity and symptoms, as well as with overall cognitive functions. Our study adds evidence that low levels of 25(OH)D may negatively affect the progression of Parkinson's disease (PD). Intervention studies in this area are needed.
Wu et al.[16]	Correlation between serum 25(OH)D and cognitive impairment in Parkinson's disease.	Journal of Clinical Neuroscience	Vitamin D deficiency is widespread in patients with Parkinson's disease (PD). Our aim was to determine whether serum vitamin D levels were correlated with bone mineral density (BMD) and non-motor symptoms in patients with PD..	In patients with Parkinson's disease (PD), vitamin D levels were significantly correlated with falls and some non-motor symptoms. However, no associations were found between bone mineral density (BMD) and serum 25(OH)D levels in patients with PD. Thus, vitamin D supplementation is a potential therapy for the non-motor symptoms of PD..
Marshal et al.[17]	Mechanistic insights into the role of vitamin D and computational identification of potential lead compounds for Parkinson's disease.	Journal of Cellular Biochemistry	This study aimed to investigate the relationship between serum 25(OH)D levels and cognitive impairment in patients with Parkinson's disease (PD), in the hope of providing possible insights for the diagnosis and prevention of PD with cognitive impairment.	These findings support the relationship between cognitive impairment and vitamin D in patients with Parkinson's disease (PD). Serum 25(OH)D may be a useful biomarker for diagnosing cognitive impairment in patients with PD.
Mazzetti et al.[1]	Astrocytes expressing vitamin D-activating enzyme identify Parkinson's disease.	CNS Neuroscience & Therapeutics	The present study aims to evaluate the neuroprotective activity of vitamin D3 in astrocytes following exposure to rotenone (ROT), a natural pesticide known for its neurotoxic potential through the inhibition of mitochondrial complex I.	Therefore, treatment with vitamin D3 protected astrocytes from ROT-induced damage by reducing oxidative stress, decreasing the expressions of NF-kB and Nrf2, and improving mitochondrial function. However, further investigations are needed regarding the involvement and mechanism of action of vitamin D3 in this cellular model of Parkinson's disease (PD), focusing on the crosstalk between Nrf2 and NF-kB.

in the impairment of patients' cognitive functions, as it is capable of acting as an anti-inflammatory factor. However, this function is not perceived in the control of neuroinflammation of microglia. That is, vitamin D deficits do not seem to influence the severity of symptoms but are related to the development of the disease.[8,9,17]

Furthermore, a contribution of astrocytes positive for the vitamin D-activating enzyme (CYP27B1) in neuroprotection was observed. CYP27B1 assists in the second hydroxylation of 25-hydroxyvitamin D into the active metabolite, that is, 1,25-hydroxyvitamin D. These types of astrocytes appear to carry out the uptake and clearance of synuclein, preventing accumulation and sensitization of the dopaminergic neuron to oxidative stress and formation of Lewy bodies. Another aspect noted is the influence of VDR genetic variability on PD susceptibility due to the presence of polymorphisms, such as VDR rs2228570, which has been shown to increase the chances of motor fluctuations, highlighting the role of vitamin D in achieving neuromuscular function.[4,7,10,12-14,18]

Hypovitaminosis can cause a reduction in bone mineral density, proximal muscle weakness, pain, difficulty walking, and postural instability. Therefore, there is evidence of the importance of investigating vitamin D levels in the early stages, to prevent the patient's condition from worsening due to the intense reduction in bone mass as the disease progresses. Supplementation of this vitamin proved to be safe but did not provide considerable improvements in the balance of older patients. However, the possibility of improving balance in young individuals has been reported.[10,12-14]

Vitamin D deficiency in elderly patients with mild cognitive impairment is associated with reduced hippocampal volume and brain structural connectivity. A smaller total hippocampal volume is observed in those with deficient serum 25-OH D levels, which was considered in later studies due to the smaller volumes of the CA1, molecular layer, dentate gyrus, and fimbria subfields. This atrophy occurs through some mechanisms, including increased pro-inflammatory cytokines, increased oxidative stress, reduced level of neurotrophic factors, decreased synaptic protein, and increased excitotoxicity, all of which can result in reductions in subfield volumes of the hippocampus. Disruption of brain structural connectivity in 13 regions has been reported in patients with deficient serum vitamin D levels. Vitamin D improves hippocampal synaptic function in rats. Therefore, disturbances in the structural connectivity of the hippocampus and the reduction in its volume may be related to the loss of synapses and the reduced level of synaptic protein. This low level of synaptic protein is a cause of reduced hippocampal volume, which leads to reduced neuronal connectivity. Network disruption was most evident in the right hemisphere, with the right hippocampus as the center, producing significantly more severe neurocognitive outcomes than patients who were not vitamin D deficient, as reflected in scores from a cognitive battery including MMSE, CDR, CDR-SOB, and GDS.[12]

It is known that increased vitamin D changes blood flow parameters, improving tissue microcirculation. This increases oxygen transport and tissue perfusion, improving mitochondrial function and defense against oxidative stress. As already mentioned, mitochondrial impairment and reduced capacity to eliminate free radicals are closely linked to the pathophysiology of chronic neurodegenerative disorders. Thus, it can be hypothesized that higher vitamin D levels contribute to better coping with PD symptoms, such as cognitive impairment and age-related comorbidities such as diabetes mellitus or cardiovascular diseases.[13]

However, in Kuhn, Karp, and Müller's 2022 case-control study13, they concluded that vitamin D levels did not vary significantly between PD patients and matched controls, and did not show any relationship with the severity of the disease, in contrast to other clinical investigations. However, they report that participants were assessed only once and were not taken off PD medication. Therefore, correlation analysis cannot provide profound value in assessments of functional deficits about vitamin D measurement in this PD cohort.

It is already recognized that patients with PD have reduced serum levels of 25(OH)D when compared to the general population. Furthermore, it was discovered that the deficiency state is mainly associated with reduced sun exposure and food intake, which, in turn, does not appear to be related to the severity of diseases or their progression and cognitive decline. However, the study suggested the existence of confounding factors, as well as endogenous changes in vitamin D metabolism in PD, as serum 25(OH)D levels in PD patients were lower than in controls, even with comparable food ingestion and sunlight exposure. Thus, preliminary evidence suggests that the combination of greater sun exposure and vitamin D supplementation is the most effective strategy, having greater potential benefits from food fortification than dietary modifications alone.[14]

It was also reported that PD patients with low serum levels of 25(OH)D had a higher frequency of falls, sleep problems, depression, and anxiety, in addition to lower BMD in the lumbar spine and femoral neck. Together with the other results, these relationships reinforce that vitamin D deficiency plays a role in the pathogenesis of PD, while vitamin D supplementation can be used to treat the non-motor symptoms of PD.[9]

Furthermore, it has been reported that reduced vitamin D levels may occur in the early stages of the disease, even before its onset. Other studies have found that vitamin D deficiency is present in patients with early, untreated PD. Furthermore, the risk of PD increases significantly as vitamin D levels decrease, and it is important to highlight that this phenomenon is complex and is influenced by several environmental and genetic factors already discussed.[9]

CONCLUSION

The analysis of the scientific productions allowed to conclude that there is no consensus on vitamin D's causal relationship and therapeutic potential in PD. Still, there is strong evidence of its relevance in developing the disease. In this sense, most studies found that the risk of PD increases proportionally to the reduction in serum levels of 25(OH)D, that is, it appears to influence the development of the pathology but is not associated with the severity of the symptoms manifested by the patient. Among the reasons is that vitamin D is capable of modulating neurotrophic factors essential for the maintenance of dopaminergic neurons and even influencing the improvement of tissue microcirculation. Another interesting point is that vitamin D deficiency can occur in the early stages of the disease, demonstrating the importance of investigating levels at the beginning of the disease due to the possibility of reducing bone mineral density and worsening the condition due to falls. Although vitamin D has not demonstrated benefits in improving the balance of older patients, benefits have been seen from administering a moderate dose in young individuals; however, it is essential to carry out other studies on the causal relationship and therapeutic possibilities of the corresponding vitamin D.

Author´s contribution

Nicole Custódio Porto Silva: Formal analysis, Methodology
Rafael badalotti: Project administration
Laís Gabriel Inácio da Silva Dantas: Data curation, Writing – original draft
Nathália Carolinne Rabêlo de Souza: Validation, Writing – review & editing

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