

Original Article

Evaluating Prevalence and Risk Factors of Tooth Wear in Parkinson's Disease: A Narrative Review

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ABSTRACT

Tooth wear has emerged as a notable oral health concern that can negatively impact quality of life. Although Parkinson's disease (PD) is associated with poorer oral health compared to healthy populations, research specifically addressing tooth wear in PD remains scarce. Data on its prevalence and contributing factors in this group are particularly limited. This review examines the existing evidence on tooth wear in PD, highlighting reported prevalence, potential risk factors, and avenues for future investigation. A search of PubMed yielded four relevant studies, comprising one case report and three surveys. These studies indicate that PD patients may experience more pronounced tooth wear than healthy individuals. Additionally, connections between tooth wear and factors such as bruxism, temporomandibular disorder (TMD) pain, and oral health-related quality of life (OHRQoL) have been suggested. Due to the limited number of studies, definitive conclusions about the scope of tooth wear in PD cannot be drawn. However, the following propositions can be considered: (1) tooth wear may be more frequent in PD patients compared to healthy controls, (2) risk factors common in the general population could be more prevalent among PD patients, and (3) multiple overlapping factors may contribute to tooth wear, influencing its development and severity in this population.

Keywords: Quality of life, Tooth wear, Oral health, Parkinson's disease

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Introduction

Tooth wear, characterized by the gradual loss of dental hard tissues, becomes increasingly common with age and tends to worsen over time [1]. In some individuals, this process may progress to pathological tooth wear, where moderate tissue loss is accompanied by clinical signs such as tooth sensitivity [2]. Severe forms of tooth wear affect roughly 3–17% of adults [3]. Although single causes may occasionally dominate, tooth wear usually arises from multiple interacting factors, including mechanical and chemical processes of both intrinsic and extrinsic origin [4]. This multifactorial nature makes diagnosis and management challenging, while the loss of dental tissue can significantly impair oral health-related quality of life

(OHRQoL) [5]. Therefore, understanding and addressing tooth wear is a crucial aspect of oral health care.

Parkinson's disease (PD) is a progressive neurodegenerative disorder primarily affecting the substantia nigra and other brain regions [6]. It manifests with a range of motor and non-motor symptoms, including tremors, facial masking, pain, and cognitive deficits [7]. While PD can occur in younger adults, its incidence rises with age [8]. Standard treatment focuses on symptom management through medications that elevate dopamine levels, such as levodopa, alongside supportive interventions like physiotherapy, speech therapy, and occasionally neurosurgical procedures [9].

Evidence suggests that overall oral health is poorer in PD patients compared to healthy individuals [10]. Despite the recognized prevalence of tooth wear in the general population, its occurrence and risk factors in PD remain largely unexplored. This raises key questions: is tooth wear more common or severe in PD patients, and do conventional risk factors for tooth wear also apply to this population? Accordingly, this review has two objectives: to summarize existing evidence on tooth wear prevalence and risk factors in PD patients, and to propose hypotheses to guide future research.

Material and Methods

To investigate the first objective, a search of PubMed was conducted on January 2, 2024, using the MeSH terms “Parkinson’s disease” and “tooth wear,” supplemented by free-text keywords such as “tooth wear” and “dental wear.” Only original research articles were considered, without restrictions on language or publication date. References from included studies were also screened for additional relevant publications.

Results

The search identified eight articles, of which four provided data on tooth wear in PD patients. Magee reported a case of a 53-year-old woman with PD who developed tooth wear attributed to “pursing mouth movements” caused by high-dose levodopa therapy (4.5 g). Despite lowering the dose, dental grinding persisted, and after ten months, further damage was deemed unacceptable. The patient declined cessation of levodopa due to symptom relief, and splint therapy was initiated, successfully halting progression after seven months [11].

In addition, a pilot questionnaire study found a significant link between PD and self-reported tooth wear, though the authors cautioned that the reliability of self-reported measures was limited [12]. Secondary analysis of this cohort showed associations between tooth wear and both sleep and awake bruxism in PD patients [13]. Another case-control questionnaire study suggested that PD patients with tooth wear experienced worse OHRQoL compared to matched controls [6].

Overall, the evidence on tooth wear in PD is limited, largely based on self-reported data, and in two instances, derived from the same participant cohort. While these studies highlight associations with factors such as medication, bruxism, and OHRQoL, reliable prevalence data remain scarce, emphasizing the need for more robust investigations.

Discussion

This review sought to summarize the existing evidence on tooth wear in Parkinson’s disease (PD) and to outline directions for future research. The limited studies available indicate that PD patients may experience tooth wear more often than healthy individuals, yet these findings are largely based on self-reported information. To date, only a few factors—namely medication intake, bruxism, and oral health-related quality of life (OHRQoL)—have been consistently associated with tooth wear in this population.

Tooth wear is a complex condition, encompassing multiple domains such as detection, prevalence, causative mechanisms, clinical consequences, and therapeutic approaches. Literature analysis shows that most of these domains remain unexplored in the context of PD. Reliable data on the prevalence of tooth wear in PD are scarce, emphasizing the need for studies with representative patient samples and standardized assessment methods [4]. Furthermore, potential risk factors for tooth wear, which are relevant across all five domains, have received little attention, representing a major gap in knowledge. Investigating known risk factors from the general population [14–18] and evaluating their interaction with PD-specific characteristics may help clarify the mechanisms driving tooth wear in this patient group.

The following sections focus on medical, social, and other miscellaneous risk factors for tooth wear identified in healthy individuals, considering their possible relevance to PD. This approach aims to generate testable hypotheses and provide a framework for future research to better understand the prevalence, causes, and progression of tooth wear in people with PD.

Medical risk factors

According to Slater *et al.* [17], multiple medical conditions in the general population are associated with tooth wear, including genetic predisposition, orofacial pain, psychological factors, salivary flow abnormalities, sleep disturbances, medication intake, and acid reflux (**Table 1**). To date, no studies have specifically examined genetic contributions that might explain the increased susceptibility to tooth wear in patients with Parkinson’s disease (PD). Pain is a prominent non-motor symptom in PD, affecting 68–85% of individuals [18], yet orofacial pain such as temporomandibular disorder (TMD) pain and its potential link to bruxism has not been extensively evaluated. Recent evidence indicates that both TMD pain and bruxism are more frequently observed in PD

patients than in healthy individuals, with reported prevalence ranging from 0–33% for TMD pain and 2–57% for bruxism [19, 20], implying a potentially greater burden of tooth wear related to bruxism in this group.

Sleep-related conditions in PD extend beyond bruxism. Both central and obstructive sleep apnea are reported, with prevalence rates between 0–49% and 42–60%, respectively, though the research remains limited [21–26]. Some studies have also noted correlations between sleep metrics—such as mean oxygen saturation and the proportion of REM sleep—and PD severity [24], although the mechanisms remain unclear. Brain degeneration patterns may influence these associations [27], and increased sleep disturbances could lead to more frequent arousals and bruxism episodes, thereby raising the likelihood of tooth wear.

REM sleep behavior disorder (RBD), a parasomnia commonly seen in PD, affects 3–60% of patients [28]. Among these individuals, approximately 25% experience sleep bruxism [19, 29], which is substantially higher than in the general population, suggesting heightened vulnerability to tooth wear.

PD patients also exhibit higher rates of psychological disorders, including depression and anxiety (38% and 31%, respectively) [30, 31]. Since these conditions are linked to bruxism in the general population, they may similarly elevate tooth wear risk in PD.

Salivary dysfunction is more pronounced in PD patients than in healthy individuals. A systematic review revealed that PD patients tend to have reduced salivary flow [32], which may partly result from medication use. On average, PD patients take 7.4 ± 2.5 medications daily, with a median intake of five times per day [33]. Dopaminergic treatments, commonly used in PD, have been associated with both bruxism and TMD pain [20], suggesting that medication may

indirectly contribute to tooth wear alongside salivary issues.

Gastroesophageal reflux disease (GERD) has also been linked to PD, with odds ratios of 1.29–4.05 [34, 35]. Maeda *et al.* found GERD in 26.5% of PD patients [34], and gastrointestinal disorders—including dysphagia, constipation, and GERD—were reported to increase by 65% within four years of PD diagnosis [36], reinforcing reflux as a potential risk factor for dental wear.

Finally, awake bruxism affects nearly half of PD patients (46%) [14]. Because PD is a movement disorder, involuntary movements such as dyskinesias—which are repetitive, jerky, or dystonic in nature—may increase tooth-to-tooth contact [37, 38]. This heightened craniofacial muscle activity could further contribute to the loss of dental hard tissue in PD compared with healthy individuals.

Social risk factors

Table 1 outlines the social factors that may influence tooth wear and the proposed mechanisms through which they act. Evidence from the literature suggests that alcohol consumption is inversely linked to the likelihood of developing PD [39, 40], while caffeine intake [41] and smoking [40] may also reduce PD risk. In otherwise healthy individuals, these behaviors are considered contributors to tooth wear because they can trigger bruxism. Considering the inverse relationship between alcohol and caffeine use and PD, it is plausible that PD patients consume these substances less frequently, although this is not conclusively demonstrated. As a result, the contribution of these factors to tooth wear in PD patients may be similar to that in healthy populations. However, data on the prevalence of alcohol, caffeine, and tobacco use in PD patients remain unavailable, so interpretations should be made cautiously.

Table 1. Factors Contributing to Tooth Wear in Healthy Individuals and Their Underlying Mechanisms

Category	Risk Factor	Underlying Mechanism
Medical Factors	Genetics	Variations in genes, such as those affecting amelogenin (a protein critical for enamel formation), may contribute to the presence or severity of tooth wear.[15, 42]
	Pain	Orofacial pain serves as an indirect indicator of tooth wear, often linked to bruxism, which increases the risk of tooth wear.[15, 17]
	Sleep Disorders	Bruxism, now recognized as a potential protective mechanism in sleep apnea, is associated with a higher risk of tooth wear. Additionally, rapid eye movement sleep behavior disorder (RBD) is linked to bruxism.[17, 29]
	Psychological Factors	Conditions like ADHD, anxiety, or stress increase the likelihood of bruxism, thereby elevating the risk of tooth wear.[15, 17]
	Saliva	Saliva protects dental tissues, but reduced salivary flow or poor saliva quality (e.g., low buffering capacity) increases susceptibility to tooth wear.[14, 15, 17]

	Medication Use	Certain medications may exacerbate bruxism or decrease salivary flow, contributing to tooth wear.[17, 43]
	Acid Reflux	Gastroesophageal reflux lowers oral pH, leading to erosive tooth wear. This risk is heightened in individuals with sleep disorders (e.g., obstructive sleep apnea) or through dietary factors, though diet alone can also affect oral pH.[14, 15, 17, 44]
Social Factors	Stimulant Use (e.g., alcohol, caffeine, drugs, smoking)	Stimulants are linked to increased bruxism and reduced oral pH, both of which elevate the risk of tooth wear.[17, 18]
	Erosive Diet	See the explanation under acid reflux for details on how dietary factors contribute to tooth wear.[14, 15, 17, 44]
	Sports	Physical activity can lead to dry mouth, and some athletes, particularly those involved in strength training, may be prone to clenching, increasing tooth wear risk.[23]
Miscellaneous Factors	Ageing	Tooth wear progresses with age, with prevalence increasing over time.[17, 44]
	Occlusion	Excessive occlusal forces or premature contacts can influence the presence or severity of tooth wear.[16, 17, 44]
	Oral Hygiene	The type of toothbrush and frequency of oral hygiene practices can affect the presence or severity of tooth wear.[14]

Abbreviations: RBD – rapid eye movement sleep behavior disorder; ADHD – attention deficit hyperactivity disorder.

No studies have documented the dietary habits of individuals with PD, yet certain nutrients, such as vitamin C, are known to provide health benefits. However, some dietary components, including high-protein foods and ferrous sulfate supplements, may interfere with dopaminergic therapy [45], requiring physicians to guide patients on which foods to limit or encourage. Diet plays a central role in tooth wear, particularly when acidic foods are involved, but data on the specific dietary tendencies of PD patients remain unavailable. Nonetheless, plausible assumptions can be made. For instance, since loss of smell is a common non-motor symptom of PD [7], this sensory deficit can lead to a reduced sense of taste, prompting individuals to prefer stronger, more acidic flavors, which in turn may elevate the likelihood of erosive tooth wear. Furthermore, because vitamin C can enhance the pharmacokinetics and therapeutic action of dopaminergic medication [45], clinicians may recommend its supplementation or increased consumption of vitamin C-rich fruits. While beneficial pharmacologically, such habits expose teeth to acidic substances that could heighten the risk of erosion.

PD patients are also encouraged to maintain physical activity, though little information exists regarding their consumption of sports drinks or participation in endurance exercise. Among healthy individuals, these behaviors have been linked to tooth wear, partly due to the acidity of sports drinks and the frequent clenching observed during endurance sports. While PD patients are advised to engage in such activities, their participation level is unlikely to differ substantially from that of healthy populations.

Miscellaneous risk factors

Beyond social and medical contributors, elements such as aging, occlusal characteristics, and oral hygiene habits also play a role in tooth wear (**Table 1**). Although the incidence of PD among younger adults is rising, the condition remains more prevalent in older individuals [9]. Research indicates that oral health generally deteriorates with age—a trend mirrored in PD patients, who often experience tooth loss or retain root remnants [10]. Diminished self-care ability, stemming from PD-related motor challenges, may further exacerbate poor oral health. Nevertheless, reviews on oral hygiene practices have found no significant difference in oral care frequency between PD patients and healthy counterparts [10]. It is conceivable that awareness of their heightened oral health risk encourages PD patients to improve self-maintenance. Still, the involuntary motor symptoms characteristic of PD may cause erratic movements during oral care, contributing to abrasion, a type of tooth wear resulting from mechanical forces other than tooth contact. Despite these observations, specific data on oral hygiene routines in PD patients remain unavailable.

Conclusions

Current evidence regarding the prevalence and determinants of tooth wear in PD patients is scarce. However, it can be inferred that overlapping factors such as reduced salivary secretion, bruxism, medication intake, and gastric reflux likely increase

susceptibility to tooth wear in this population. Consequently, dental professionals should be alert to the heightened risk of extensive tooth wear in PD patients, which may adversely affect their oral health-related quality of life (OHRQoL). Nonetheless, it remains uncertain whether PD patients or their dentists perceive tooth wear as a significant concern.

Future investigations could be guided by several hypotheses: (1) severe tooth wear occurs more frequently in PD patients compared to healthy individuals; (2) risk factors commonly associated with tooth wear in the general population may be more pronounced among those with PD; and (3) the coexistence of multiple contributing factors in PD may collectively influence both the prevalence and progression of tooth wear in affected individuals.

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