Special Care in Dentistry

WILEY

REVIEW OPEN ACCESS

Huntington's Disease and Dentistry: A Review of Its Etiology, Clinical Presentation, Symptomatic Pharmacotherapy, and Dental Management

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Received: 20 October 2024 | **Revised:** 23 February 2025 | **Accepted:** 17 March 2025

Keywords: dental treatment | drugs | huntington's diseases | oral modifications | pharmacology

ABSTRACT

Aims: Patients with Huntington's Disease (HD) seeking dental care often present with poor oral health. Dentists often report that few protocols exist and there is a lack of understanding on how to manage these patients in the dental office. The aim of this review is to discuss the etiology and pathophysiology, clinical presentation, and management of this condition primarily from the dental perspective.

Methods: A detailed literature review was conducted including articles searched on PubMed and Google Scholar using relevant keywords. Inclusion criteria prioritized studies based on relevance to oral care and HD with topics including caregiver-assisted oral hygiene, fluoride, dental preventative approaches, and case studies.

Results: Research reveals the significant oral health issues patients with HD suffer from including dysphagia, xerostomia, and a high caries risk. Studies illustrate the need for tailored care emphasizing long term treatment planning, preventive, and clinically relevant approaches.

Conclusion: This review presents the importance of a proactive, multidisciplinary strategy to dental care in HD patients. Early preventive strategies can greatly postpone the decline of oral health in this population. Future studies should focus on a conclusive dental care guideline, caregiver education initiatives, and improving access to care in this patient population.

1 | Introduction

Huntington's Disease (HD) is a rare inherited condition manifesting with neurological, motor, and psychiatric impairments that gradually worsen throughout an individual's lifespan [1]. The disease was first described in 1872 by George Huntington [2]. The global prevalence of HD is estimated at 4.88 per 100 000 according to a 2022 meta-analysis, and an estimated pooled incidence of 0.48 per 100 000 person-years [3]. About 4700 Canadians are currently living with HD with approximately 1 in 5500 Canadians at risk of developing HD [4–6]. In the United States, around 200 000 Americans are at risk of developing HD [7]. If a single parent has HD, the child has a 50% chance of acquiring the disease [5, 8]. The age of onset for HD can range from ages 2 to 87, but more commonly occurs between ages 30 and 50 with both males and females having a similar chance of developing HD [5, 8–10]. Individuals presenting with symptoms under the age of 21 can develop a rare form called Juvenile onset Huntington's Disease (JHD), affecting up to 10% of all HD patients [10]. Symptoms can also begin in the elderly age > 60 years old, defined as late-onset HD (LoHD) [11]. Following onset, individuals have a life expectancy of around 15–18 years [8]. As the disease has

significant effects on the individual's oral care maintenance and accessibility to care, alongside its increasing prevalence, dentists should be aware of specific protocols for managing patients with this disease.

1.1 | Etiology and Pathophysiology

The Huntington gene (HTT) is located on chromosome 4 and is represented by a large stretch of CAG (polyglutamine) repeats. An abnormal repetition of polyglutamine is key in the etiology of HD, and its number can predict the clinical presentation/diagnosis of HD in susceptible individuals. As HD follows an autosomal dominant inheritance pattern, only 1 allele with a varying CAG repeat is sufficient for individuals to symptomatically present HD [8]. Individuals with 6–35 CAG repeats typically do not present with HD. The presence of 27–35 CAG repeats constitutes an intermediate HTT allele (IA) and IA carriers are unlikely to develop HD. However, the presence of IAs promotes an increased risk for progeny to develop HD [12]. Reduced penetrance of HD is often seen in individuals with 36–39 CAG repeats where some individuals may or may not develop HD symptoms within this range [13]. Disease presence is often associated with 40 or more CAG repeats. While the number of CAG repeats can signify the presence and severity of HD, it can also determine the onset of symptoms [13, 14]. In approximately 80% of individuals with JHD, 60 or more CAG repeats are present [15].

Concerning the neuropathology of HD, the most common area of degeneration in the brain is the basal ganglia (BG). The medium spiny neurons (MSNs) of the striatum are the most targeted by the mutant HTT (mHTT) proteins. Loss of MSNs is associated with decreases in neurotransmitters such as GABA and NMDA. The striatum is a crucial region involved in the motor and reward system that accepts primary inputs of the BG and houses the expression of dopamine receptors via MSNs [8, 13, 16, 17]. Bilateral atrophy of the dorsal striatum, more prominent in the caudate than putamen, is a common pathology observed in HD patients, and this structure is important for its role in cognitive and motor function [18]. On magnetic resonance imaging and brain scans, enlargement of the lateral ventricles associated with caudate atrophy is a clinical feature in diagnosing HD. Other areas of the brain can also be affected by HD, including the cortex, cerebellum, thalamus, and hypothalamus [16]. There have been theories proposed of mHTT protein pathogenesis on a cellular level such as mitochondrial dysfunction or forming clusters on neuronal cells leading to their death [8, 17].

1.2 | Clinical Presentation

HD can present with symptoms that primarily affect motor, neurological, and psychiatric function [1]. Of the three, the most prominent and last impairment to appear is motor, specifically chorea. Chorea is defined as short irregular, involuntary, and spontaneous muscle movements of varying degrees. In the early stages of HD, motor impairment primarily manifests as chorea (Table 1). The chorea initially presents as athetosis with jerk-like movements that are localized to the face and extremities. Progression of the disease increases the frequency and duration of the chorea while also spreading it throughout the whole body [8, 17, 19]. Chorea in HD can alter various bodily functions, such as an individual's gait or eye movement. Eye impairments can present in early stages such as difficulty in tracking with eyes, termed saccadic impairments that correct in more advanced stages [20, 21]. The early motor symptoms of HD only appear to stop during rest. As the disease terminally progresses, diminishing motor activity is often seen such as presented in parkinsonism including bradykinesia, akinesia, dystonia, and muscle rigidity [17, 22].

TABLE 1 | Stages of Huntington's disease and the symptoms associated with each stage.

Stage of Huntington's disease	Clinical presentation
Prodromal/ preclinical stage	Psychiatric manifestations such as depression, apathy, OCD, suicide ideations, anxiety, mild cognitive impairments, systemic manifestations
Early stage	Mild chorea (athetosis), saccadic eye movements, psychiatric manifestations, weight loss, advancing cognitive impairments, dysphagia/dysarthria
Middle stage	Advanced whole-body chorea and further progression in the cognitive, oral, and psychiatric symptoms
Late stage	Bradykinesia, akinesia, dystonia, muscle rigidity, subcortical dementia, psychiatric and oral manifestations become more severe leading to aphasia

Both psychiatric and cognitive symptoms can present prodromal, before the development of the disease and initiation of motor symptoms. Depression is the most common psychiatric condition associated with HD, seen in up to 75% of individuals with HD, followed by anxiety [19, 23]. Other psychiatric conditions such as OCD, apathy, and bipolar disorder are seen in HD patients but less commonly. Apathy, an absence of motivation or interest, has been the only psychiatric symptom shown to progress with HD in severity [17, 24]. Psychosis can also be present but is more commonly associated with early-onset HD. Psychiatric symptoms are often risk factors in the development of suicide ideations in HD patients, which is one of the leading causes of death for these individuals [7, 19, 24]. Cognitive deterioration is also a key feature that is present in HD. Mild cognitive impairments start to present in the prodromal and early phases of HD, with progression to subcortical dementia further in the disease stage [25, 26].

Studies have also suggested that systemic comorbidities can be associated with HD. These systemic presentations can often develop at least a decade before motor impairments [19]. Cardiovascular symptoms seen in individuals with HD can include bradycardia and heart failure, shown in the literature to be associated with about 30% of patients with HD. Cachexia associated with weight loss is often presented in HD patients before diagnosis, and individuals often find it difficult to maintain weight with an unchanged diet [19, 27]. Other reported systemic

manifestations include diabetes mellitus, osteoporosis, and testicular atrophy [16, 28]. With regards to JHD, epileptic seizures are commonly presented, seen in 30%–50% of these patients.

1.3 | Oral Manifestations

The most common oral presentation associated with HD is dysphagia. Dysphagia is a disorder in the swallowing process that can lead to potential aspiration of contents into the airway [29]. In the case of HD, the underlying chorea is often the cause of dysphagia. Recent studies demonstrated that dysphagia occurs in more than 50% of patients with HD and can be seen in as many as 77.6% of patients [30, 31]. Individuals often find it difficult to eat which can also affect their diet. As motor symptoms progress, tube feeding is often necessary for survival and weight maintenance. Dysphagia presentations can also vary between patients, and some presentations can include failure to clear solid particles in the pharynx, alterations of tongue movement, and/or lip incompetence [32]. Dysphagia is found in various stages with progression following disease severity [30]. As such, it can be useful as an indicator of the severity of HD [31]. A major complication requiring hospitalization following dysphagia is aspiration pneumonia [33].

Impairment of muscular tone and movement can also affect speaking and often cause dysarthria. Like dysphagia, dysarthria can be widely present in various stages of HD. Variations of dysarthria have been noted in recent studies, but patients most commonly vary by speech speed [34]. Of importance is the difficulty in communication between the patient and dentist due to dysarthria, which can have a negative consequence on successful treatment outcomes [32]. Chorea associated with the tongue and weakness of key facial muscles in maintaining oral prosthetics can present difficulty in removable prosthodontic treatment planning for patients with HD [35].

Drug-induced oral manifestations can also present in HD patients taking medications for symptomatic care. Xerostomia, or dry mouth is often a common culprit in patients taking antipsychotics and antidepressants, making it difficult to maintain proper oral hygiene (Table 2). Other noted oral adverse effects of these drugs include sialorrhea, increased fungal infections, and orthostatic hypotension, which increases the difficulty of providing proper dental care [36]. The disease ultimately leaves the patient with poor oral health. Chorea especially in later stages creates difficulty in maintaining oral hygiene practices such as brushing, flossing, and access to care. Cognitive and psychiatric impairments can also contribute to poor prioritization of oral health. This leads to common presentations in the dental office including caries, gingivitis, and periodontitis. These conditions often get worse as the disease progresses [32]. As well, the clinical presentations of HD also provide challenges in providing basic dental care to these patients.

1.4 | Medical Management

As it stands, there is currently no treatment or cure for HD. However, options exist to manage the symptoms associated with HD. Symptomatic care can involve pharmacotherapy, nonsurgical, and/or surgical care. In terms of surgical care, the use of deep brain stimulation has been shown by studies to reduce Huntington's chorea [37]. Stress and anxiety can often be associated with increasing chorea in patients with HD. Nonsurgical care can involve behavioral management and stress reduction strategies, such as exercise or psychological therapy. Environmental modifications and protective appliances can help to prevent injury from chorea [38]. Pharmacological therapy for HD is currently approved only for chorea. This involves the use of vesicular monoamine transporter 2 (VMAT-2) inhibitors, most notably Tetrabenazine. Other drug categories that have been reported to alleviate the clinical symptoms of HD include antipsychotics and antidepressants.

1.5 | Pharmacotherapy

1.5.1 | VMAT2 Inhibitors

Vesicular monoamine transporter 2 (VMAT2) inhibitors are used as the first-line agent in the symptomatic treatment of Huntington's chorea. VMAT2, primarily found in the CNS, functions to uptake monoamines, more commonly dopamine, into vesicles of presynaptic neurons for release and functioning in the synaptic cleft. There are three main VMAT2 inhibitors approved for HD chorea in the United States: Tetrabenazine (Xenazine), Deutetrabenazine (Austedo), and Valbenazine (Ingrezza) [39]. Some side effects of this class of drug reported in the literature include parkinsonism, sedation, akathisia, and anxiety (Table 2). An important adverse effect is depression, as it can lead to increased ideations of suicide in HD patients already experiencing mood disorders [39, 40]. While VMAT2 inhibitors are most effective for chorea treatment in HD, for patients with depression, antipsychotics or neuroleptics are generally recommended for managing chorea [40, 41].

1.5.2 | Antipsychotics and Antidepressants

In instances where patients do not respond well to VMAT2 inhibitors or also experience parkinsonism/depression, antipsychotics have been stated in the literature to be used as a second-line agent for reducing chorea but are primarily used for managing psychosis [40]. However, their role as a second-line agent for HD chorea can vary as there are few studies with protocols for this specific use in North America [42]. Drugs primarily used in HD chorea include Olanzapine (Zyprexa) and Risperidone (Risperdal). Some common side effects of these antipsychotics include xerostomia and orthostatic hypotension listed in Table 2 [40].

The literature also reports the use of antidepressants by some patients with HD as supportive care to reduce their symptoms of depression [43]. Classes of antidepressants can include selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine (Prozac) and citalopram (Celexa), and Tricyclic antidepressants (TCAs) such as amitriptyline (Elavil) and imipramine (Tofranil). Serotonin and noradrenaline reuptake inhibitors (SNRIs) can also be used such as venlafaxine (Effexor). SSRIs are commonly the first-choice antidepressant for initial treatment due to less adverse effects. SSRIs work to selectively inhibit the reuptake of serotonin

TABLE 2 | Common drugs used in the symptomatic treatment of Huntington's disease.

Drug classification	Class examples	Mechanism of action	Common adverse reactions relevant to dental care	Drug interactions relevant to dentistry
VMAT2 Inhibitors	Tetrabenazine Deutetrabenazine Valbenazine	Inhibits vesicular monoamine transporter type 2 (VMAT-2)	nausea, depression, sedation, bradykinesia, anxiety	Benzodiazepines Opioids
Antipsychotics	Haloperidol, Olanzapine, Risperidone	Blocks dopamine and/or serotonin receptors	tardive dyskinesia, QT prolongation, diabetes, xerostomia, orthostatic hypotension	Benzodiazepines Opioids Macrolides Quinolones Metronidazole
Antidepressants	TCAs, SSRIs, SNRIs	target certain neurotransmitters such as serotonin to regulate mood and behavior	Orthostatic hypotension, xerostomia, urinary retention	Benzodiazepines NSAIDs Opioids

*Pharmacology data was obtained from Lexi-comp and Compendium of Pharmaceuticals and Specialties (CPS)

from the synaptic cleft into presynaptic terminals. ADRs can include xerostomia, weight loss, nausea & vomiting, and sexual dysfunction. TCAs block the presynaptic reuptake transporters for norepinephrine and serotonin and the postsynaptic receptors for histamine, acetylcholine, and norepinephrine. Some AEs include xerostomia, urinary retention, tachycardia, and orthostatic hypotension. SNRIs work similarly to TCAs with less severe AEs. AEs seen with the use of this class can include hypertension, manic reactions, and vertigo [45, 46].

1.6 | Access to Dental Care

Access to dental care is one of the most important steps for patients to maintain oral health. However, there are situations where it may be difficult for patients especially with HD to receive care from a dentist. Common reasons why barriers persist include finances, location, lack of interest/knowledge, and fear [44]. For patients with HD, these barriers can be especially exacerbated. Due to the presenting chorea, maintaining a job can be very difficult leading to financial hardships, especially as HD can run in families. Later stages of HD can also make mobility difficult, making it harder to reach dental practitioners. The cognitive decline and mental health conditions associated with HD such as apathy and depression reduce the accountability one has on maintaining a good oral health regime. From the perspective of dental practitioners, there are a few reasons as to why they do not see many patients with movement disorders, such as HD. In a recent paper, many dentists report not having the knowledge and training to provide care for these patients, suggesting the need for a protocol for managing patients with HD [45].

1.7 | Dental Management of Huntington's Disease

The dental management for patients presenting with HD at various stages is summarized in Table 3. It is first important to obtain a thorough medical history, and a printed list of the patient's medications should be brought to the appointment if available. Consultation with the patient's neurologist and family physician is important to help guide the dental management and determine the patient's disease severity. Case selection is key for managing patients with HD. Complex specialty care such as endodontic procedures or extractions may be difficult to provide by a general practitioner as HD symptoms can increase the difficulty of the treatment. Referral to a specialty office or hospital care may be necessary for the management of patients in the later stages of HD. Severe cognitive decline and speech impairments can make it difficult for patients with HD to provide consent for dental treatment and the presence of a caregiver is required. It is important to establish a long-term treatment plan at the time of diagnosis in collaboration with the patient and caregivers when cognition has not declined. It should address the patient's concerns, caregiver role, and progressive impacts of HD on oral health. The treatment plan should be taken into consideration and be guided by the patient's wishes and cultural attitudes, such as deciding whether to provide restorative therapy to the dentition versus extractions of poor prognosis teeth versus noninvasive treatment. Biological factors should be addressed, including pain and oral disease management, while accounting for the difficulties faced by poor motivation due to coinciding depression. Alongside this, preventive therapy should be emphasized as later stages make complex operative procedures difficult to undergo. HD can also be associated with psychiatric manifestations such as depression. It is difficult enough to motivate depressive patients to maintain oral health, so it is important to build good rapport, provide emotional support, and involve caregivers to improve accountability and promote oral hygiene habits in these patients. Studies have suggested that local anesthesia becomes more difficult with anxious patients, and they tend to experience more pain during dental procedures [46]. Taking this into account, an intimidating dental environment can exacerbate the patient's anxiety which may make the success of local anesthesia difficult. As such, preparing a relaxing environment is beneficial for reducing operative pain in anxious patients.

When prescribing dental medications, it is important to be aware of drug interactions with the patient's current medication regime. The activity of Tetrabenazine and other VMAT2 inhibitors can be enhanced by other CNS depressants prescribed such as

TABLE 3 | Specific recommendations for managing patients presenting with Huntington's disease at various stages.

Stage	Dental concerns	Management strategies
Early-stage HD	- Patient may be able to give informed consent in early stages, but cognitive function can decline over time - High caries risk - Addressing the biopsychosocial needs of patients and caregivers - Mild chorea - Depressive symptoms may reduce oral hygiene motivation - Oral manifestations: dysphagia, xerostomia, bruxism, traumatic ulcers - Orthostatic hypotension	- **Long-term treatment planning** such as preventive, restorative therapy considering patient concerns/wishes, dental needs, diet, caregiver role - **Preventive**: OHI, consider 5000 ppm fluoride and varnish, xylitol, toothbrush and flossing alternatives (water floss, floss picks) - Good rapport, emotional support, and caregiver involvement can be **especially helpful for depressive patients** who may be less motivated to pursue oral hygiene - **Restorations**: consider GI over compositive for fluoride benefit and caries prevention in poor oral hygiene patients - A **mouthguard** prescription may be needed for bruxism management - Avoid reclining dental chair > 45° or up righting too fast - **Recalls**: 3–6 months and based on caries risk and patient preferences - **Do not discriminate** against these patients
Middle stage HD	- Procedure difficulty due to advancing chorea - Advancing cognitive and motor impairment may reduce the ability to undergo oral hygiene - May need to involve caregiver in oral care	- Consider the use of **minimal conscious sedation** to control chorea for long appointments - **Preventive**: In addition, SDF if the patient presents with many lesions to minimize invasive therapy - Positive stabilization, stabilizing pillows and bite block as needed for procedures
Late-stage HD	- Rigidity makes it almost impossible to provide self-care - Severe cognitive decline can reduce accountability - Care is primarily palliative	- Consider **avoiding complex procedures** or referring to a specialist/hospital - Emphasis on **caregiver education** for oral hygiene - Consider **more frequent recalls** and cleanings (every 3 months)

opioids found in pain management medications or benzodiazepines. Avoid the prescription of macrolides and quinolones as antibiotic therapy in patients taking antipsychotics due to the adverse effect of QT prolongation [47]. The choice of other antibiotics when necessary is recommended while considering the patient's allergies. The prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs) for dental pain management can increase the risk of gastrointestinal bleeding in patients taking selective antidepressants such as SSRIs [48]. The short-term use of acetaminophen for dental pain in this situation is the analgesic of choice. When NSAIDs are to be prescribed, use the lowest effective dose for the shortest duration possible. In cases where the chorea may interfere with procedures, the use of IV conscious sedation is generally recommended for patients with movement disorders, when possible, to avoid the complications associated with general anesthesia (GA). However, severe chorea presented in the later stages of the disease may require the use of GA in a hospital setting [49].

When undergoing procedures, dental chair positioning is important for HD patients. Orthostatic hypotension can be experienced from symptomatic pharmacotherapy in HD patients, especially in the elderly [50, 51]. The slow up-righting of the dental chair following long procedures is important for prevention. Also, it is not recommended to recline the dental chair beyond 45° to prevent severe outcomes of dysphagia such as aspiration pneumonia. A bite block, also called a mouth prop, can be a useful tool for patients in the middle to late stages of HD with advancing chorea and dystonia who find it difficult to keep their jaw open during dental procedures. This wedge-shaped tool is applied to the side opposite to which the dentist is working and can help to keep the mouth open, control unwanted movements, and reduce fatigue during the appointment. This tool has been shown to improve practitioner visibility and safety while being tolerated by patients with various disabilities, suggesting its benefit in patients with HD [52]. Positive stabilization can also be used in combination with bite blocks to control the unwanted movements experienced by the patient during the appointment. Positive stabilization is the act of restraining a patient's movement either physically or with a medical device to stabilize uncontrollable movements. HD patients may require frequent repositioning during dental appointments. To stabilize these patients, methods can be used

as physical restraint such as head holds by the healthcare team or family member, or the use of a blanket or stabilizing pillows, especially with HD patients who suffer from neck rigidity [53]. It is important to provide patient/family education early on and discuss the reasons protective stabilization can be used to provide an approach that is best suited based on the patient's wishes.

High-volume suctioning and using a rubber dam can also help prevent secretions and dental instruments such as burs and endodontic files from reaching the airway. For patients presenting with bruxism, the prescription of a mouth guard can help manage attrition and traumatic lip ulcers [54, 55]. Patients with HD have also been noted to be at a higher risk of sleep disorders and obstructive sleep apnea (OSA). It is important to be aware of the symptoms and when necessary, refer to the appropriate health practitioner for diagnosis and management. Mandibular advancement devices can also be used as a treatment modality for OSA in the dental office [56].

The choice of restorative material is also important to consider when managing patients with complex medical conditions. Currently, the most popular choice of restorative material is composite resin due to its aesthetics and patient acceptability. However, for patients with high caries risk such as HD, the risk of failure for these restorations is higher [57]. Frequent replacement of restorations may not be beneficial in HD patients, considering the challenges the practitioner may face in placing these restorations. Also, since HD patients often present with depression, the patient may lack oral hygiene motivation, or the chorea may impair their ability to undergo daily oral hygiene practices. The use of glass ionomer-based restorative materials may be useful in these patients due to their fluoride release mechanism and increased moisture tolerance where isolation is difficult. Although no significant differences between composite resin and glass ionomer in recurrent caries incidence, the literature suggests glass ionomer is beneficial in the prevention of new caries [50]. However, in cases where strength is required, such as posterior or large restorations, the practitioner should have discussions with the patient regarding the drawbacks of composite in high-risk patients and alternative treatment options such as extractions, taking into consideration the patient's wishes and priorities. High-viscosity glass ionomer restorations have also been suggested as an alternative atraumatic treatment modality for posterior restorations [58, 59]. However, its use may be best limited to posterior teeth lacking opposing occlusal stops and nonbruxer patients due to its poor mechanical properties.

Routine recall appointments are crucial for hygiene and caries prevention; however, a case study reported that significant plaque accumulation can still be seen in HD patients in as little as 3-month recalls [60]. Ideally, in earlier stages, these patients should have frequent 3–6 month recalls for hygiene, monitoring caries, and other oral complications. As the disease progresses and the oral cavity worsens, more frequent recalls/cleanings may be needed such as four appointments per year. The importance of prevention and oral hygiene instruction at every dental visit cannot be undermined, considering the fast progression of the disease and the difficulties faced by the patient. Dietary consultation is important as patients with HD are also put on a sugar-rich diet involving highly cariogenic foods such as honey, syrup, and sweet snacks to help maintain their weight [61]. Xylitol and other salivary substitutes are useful in reducing the symptoms of xerostomia that may be caused by the patient's medications. Pharmacological therapy for managing xerostomia can also be indicated when local salivary stimulation is not sufficient, such as the use of Pilocarpine [62]. The prescription of 5000 ppm high-fluoride toothpaste and fluoride varnish at each appointment is recommended for the prevention of caries in high-risk patients. In the middle to late stages where restorative therapy may be difficult and multiple lesions are present, 38% silver diamine fluoride (SDF) should be considered to arrest numerous carious lesions. When applied, isolation is crucial as permanent staining of the gingiva, skin, and margins of restorations can occur from SDF use. SDF application should be repeated twice per year for a prolonged benefit [63]. Electric toothbrushes with modified handles and water flossers with wide grips may improve hygiene outcomes for these patients experiencing moderate to severe chorea. In the earlier stages of HD, the patient may still have enough dexterity to undergo flossing. Customized floss handles may be beneficial in helping the patient maintain grip and access interproximal areas although not much research has been shown for this. However, the literature suggests that caregiver education remains critical in maintaining oral health as the disease progresses [64].

2 | Conclusion

Medical management for HD has been advancing in the past few years. In the future, it may be possible for a treatment to be designed to prevent the disease's pathogenesis. One therapy currently focusing on methods to reduce the body's mHTT protein levels is antisense oligonucleotide therapies (ASO) [40]. As patients are expected to have an increased life expectancy, it is of great importance to provide emphasis on the care for their oral health. It is also well known that poor oral health can exacerbate systemic medical conditions. Dentists should focus on preventive care to reduce the risk of caries and gingivitis while having modalities to provide dental care amongst the challenges of HD's clinical symptoms.

Conflicts of Interest

The authors declare no conflicts of interest.

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