



The Pain Paradox: A Narrative Review of Patient Misconceptions Conflating Dentine Hypersensitivity with the Silent Progression of Gingivitis and Periodontitis in the Absence of Dental Caries

By

Althaaf Khan

BDS, Assoc FCG Dent Private Practice, South Africa



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Abstract

Objective: Gingivitis and periodontitis are frequently misunderstood by patients as harmless because, unlike dental caries, they rarely produce overt pain until late in their course. When patients do experience oral discomfort, they commonly attribute it entirely to dentine hypersensitivity (DH) and, finding no cavity on visual inspection, conclude that nothing is clinically wrong. This narrative review examines the origins and clinical consequences of this misunderstanding and proposes a framework for patient communication that distinguishes the painless, plaque-driven inflammatory destruction of periodontitis from the short, sharp, stimulus-evoked pain of dentine hypersensitivity.

Methods: A narrative literature review was conducted using PubMed, Google Scholar and the Cochrane Library, together with consensus reports of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions and the World Health Organization Global Oral Health Status Report. Search terms combined 'periodontitis', 'gingivitis', 'dentine hypersensitivity', 'patient perception', 'self-awareness', 'health belief model' and 'oral health literacy'. English-language epidemiological, clinical and patient-perception studies published up to 2026 were included.

Results: The literature consistently shows that periodontitis behaves as a 'silent disease': self-awareness among affected patients ranges from approximately 25% to 50%, and awareness declines with age even as disease prevalence rises. Concurrently, dentine hypersensitivity affects an estimated 3-98% of the population depending on diagnostic criteria, with especially high rates (60-98%) among patients who have, or have been treated for, periodontitis, because gingival recession is the leading predisposing factor for dentine exposure. Because dentine hypersensitivity produces a recognisable pain response and caries does not, patients tend to use 'no cavity, no pain apart from sensitivity' as an informal, but clinically unreliable, health check, overlooking bleeding, halitosis, recession and mobility as meaningful disease markers.

Conclusion: The absence of caries and the presence of a plausible, familiar explanation for discomfort (sensitivity) together mask the silent progression of periodontal disease. Structured chairside communication that explicitly separates the mechanisms, symptoms and risks of gingivitis/periodontitis from those of dentine hypersensitivity is required to correct this misunderstanding and to improve timely presentation for periodontal care.

Keywords: Periodontitis; Gingivitis; Dentine hypersensitivity; Patient perception; Oral health literacy; Silent disease

1. Introduction

Dental caries has historically served as the lay public's mental model of oral disease: a visible cavity that hurts and that a

dentist repairs. Periodontal disease does not fit this model. Gingivitis, the reversible inflammation of the gingival tissues in response to bacterial biofilm, and periodontitis, the irreversible, progressive destruction of the periodontal



ligament and alveolar bone that can follow it, are described in the literature as predominantly slow, low-grade and painless conditions, frequently termed a 'silent disease' [1,2]. Substantial tissue and bone loss can therefore occur before a patient perceives that anything is wrong.

This silence creates a diagnostic vacuum that patients fill with the most available and most concrete evidence at hand: the absence of a cavity and the presence, or absence, of a sharp, stimulus-evoked pain that they recognise as 'sensitivity'. Dentine hypersensitivity (DH), defined as short, sharp pain arising from exposed dentine in response to thermal, evaporative, tactile, osmotic or chemical stimuli that cannot be ascribed to any other dental defect or pathology [3], is common, familiar, and — critically — episodic and stimulus-dependent rather than constant. When a patient with early or even moderate periodontitis notices cold sensitivity but no visible hole in a tooth, the everyday inference is reassuring but incorrect: 'there is no cavity, so the sensitivity I feel must be the whole problem, and since it comes and goes, my gums are basically fine.'

This conflation matters clinically. Gingival recession, whether from periodontal attachment loss, traumatic toothbrushing or periodontal therapy itself, is the leading predisposing factor for dentine hypersensitivity, with 39% to 96% of DH sufferers demonstrating recession [4], and DH prevalence rising to 60-98% among patients who have, or have been treated for, periodontitis [4]. Sensitivity is therefore frequently not an alternative to periodontal disease but a downstream symptom of it. Yet because sensitivity is the symptom patients can name, it becomes the explanatory anchor, while bleeding gums, halitosis, gingival recession and tooth mobility — the actual clinical markers of gingivitis and periodontitis — are dismissed as unremarkable or unnoticed altogether.

The objective of this narrative review is threefold: (i) to summarise the epidemiological and mechanistic basis for the painless progression of gingivitis and periodontitis relative to the pain-driven presentation of dentine hypersensitivity; (ii) to synthesise the evidence on patient self-awareness and health beliefs that underlie the 'no cavity, no problem' misconception; and (iii) to propose a practical communication framework that clinicians can use chairside to correct this misunderstanding and encourage appropriate periodontal care-seeking behaviour.

2. Materials and Methods

This is a narrative review; no original human or animal data were generated, and no ethical approval or informed consent was therefore required.

2.1 Search strategy

A structured literature search was performed in PubMed, Google Scholar and the Cochrane Library for articles published in English, with no lower date restriction and a search window extending to September 2026. Search terms were combined using Boolean operators and included: 'periodontitis' OR 'gingivitis' AND 'patient perception' OR 'self-awareness' OR 'health literacy'; 'dentine hypersensitivity'

OR 'dentin hypersensitivity' AND 'prevalence' OR 'mechanism' OR 'hydrodynamic theory'; 'periodontitis' AND 'dentine hypersensitivity'; and 'gingival recession' AND 'dentine hypersensitivity'. Reference lists of retrieved articles and relevant consensus reports were hand-searched for additional sources.

2.2 Source selection

Priority was given to systematic reviews and meta-analyses, epidemiological surveys with defined denominators, consensus classification reports from the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions [1,2,5], and reports from the World Health Organization and the Global Burden of Disease study. Narrative reviews and clinical guidance documents were included where they provided mechanistic or practice-relevant detail not available in primary studies. Case reports, non-peer-reviewed opinion pieces, and sources without verifiable bibliographic detail were excluded.

2.3 Analytical approach

Findings were grouped thematically into three domains: (i) the natural history and epidemiology of gingivitis/periodontitis as a largely painless disease process; (ii) the epidemiology, mechanism and clinical associations of dentine hypersensitivity, particularly its overlap with periodontal disease and recession; and (iii) the psychological and health-literacy literature describing how patients interpret the presence or absence of pain and visible cavities as a proxy for oral health. These themes were then synthesised into a discussion of the clinical misunderstanding and a proposed chairside communication framework.

3. Results and Discussion

3.1 Gingivitis and periodontitis: a painless disease process

The 2017 World Workshop classification defines periodontal health, gingivitis and periodontitis on the basis of clinical attachment loss, probing depth, bleeding on probing and radiographic bone loss — none of which requires the presence of pain [1,2,5]. Globally, the Global Burden of Disease studies estimate that severe periodontitis affects more than one billion people, corresponding to a global age-standardised prevalence of approximately 12.5%, making it one of the most prevalent chronic conditions worldwide [6,7], and a leading cause of tooth loss in adults. Despite this scale, periodontitis is repeatedly described in the literature as a 'silent' disease because pathological changes accumulate over years before pain, discomfort or functional disability become apparent [8]. This asymmetry between objective disease burden and subjective urgency is the central epidemiological fact underlying the misunderstanding addressed in this review.

Self-awareness studies corroborate this asymmetry directly. In a nationally representative United States sample, only 27% of adults with clinically diagnosed periodontitis correctly identified themselves as having gum disease, and awareness fell further, rather than rising, with increasing age — precisely the group in whom prevalence is highest [9]. A population-based Italian study similarly found that only about half of

adults correctly self-assessed their gingival bleeding, oral odour and tooth mobility status, with older patients and smokers significantly less likely to perceive their true condition [10]. A university-based survey found that almost 20% of patients did not know whether they had ever received a diagnosis of periodontitis, and patients without a periodontal diagnosis were substantially less likely to understand basic concepts such as the periodontal pocket than those who had been treated [11]. Awareness of the wider health implications of periodontal disease is similarly poor: a Saudi Arabian survey found generally low awareness of the association between periodontal disease and systemic conditions such as diabetes and cardiovascular disease [12], and a Chinese cross-sectional study of periodontitis patients found persistent gaps in disease-management knowledge even among those already receiving care [13].

Taken together, these findings show that patients cannot be relied upon to notice periodontal disease through pain, because pain is largely absent until advanced stages, and cannot reliably infer disease status from other cues either, because bleeding, recession and mobility are underrecognised or normalised as unremarkable.

3.2 Dentine hypersensitivity: a familiar, pain-driven symptom

Dentine hypersensitivity stands in direct contrast to this picture. It is, by definition, a pain condition: a short, sharp response to an identifiable stimulus [3]. The most widely accepted explanation, the hydrodynamic theory first proposed by Gysi and later developed by Brännström, holds that thermal, tactile, osmotic or chemical stimuli provoke fluid movement within exposed, patent dentinal tubules, which in turn activates mechanoreceptors associated with odontoblast processes near the pulp-dentine complex, producing pain [14,15]. Because this mechanism depends on a discrete external trigger, the pain of dentine hypersensitivity is intermittent and reproducible, unlike the constant, low-grade discomfort — or complete absence of discomfort — typical of gingival inflammation.

Reported prevalence of dentine hypersensitivity varies widely (approximately 3-98%) depending on diagnostic method, population sampled and clinical setting [16], but the association with periodontal disease and its treatment is consistent across studies. Gingival recession is the periodontal finding with the strongest predisposition to dentine hypersensitivity, present in 39-96% of DH sufferers, and DH prevalence rises to between 60% and 98% among patients with periodontitis, increasing further, from roughly 53% to over 80%, following periodontal surgical therapy such as scaling and root planing or flap surgery [4,17]. A large multicentre Chinese survey similarly identified gingival recession and periodontal treatment history among the principal risk indicators for dentine hypersensitivity [18], and systematic review evidence indicates dentine hypersensitivity is more prevalent in younger adults, an age group with lower background awareness of periodontal risk [16].

Mechanistically, then, dentine hypersensitivity in the adult population is frequently not an independent, isolated complaint but a clinical marker of underlying attachment loss and root surface exposure. However, because DH presents as a discrete, explainable, and often self-manageable symptom — reduced with a desensitising toothpaste, for example — it is experienced by patients as a resolvable nuisance rather than as a warning sign of an ongoing destructive periodontal process.

3.3 The 'no cavity, no problem' heuristic

The clinical misunderstanding at the centre of this review arises from the collision of these two disease patterns with an everyday, caries-centred mental model of dental disease. In the absence of a caries lesion, and in the presence of an intermittent, explicable sensitivity, many patients conclude — reasonably, given their frame of reference, but incorrectly — that no meaningful oral disease is present. Qualitative and survey-based research on patient knowledge repeatedly documents this pattern: patients with periodontal disease report that its impact and even its existence were unclear to them prior to diagnosis, and that pain, or the lack of it, was their primary yardstick for perceived urgency [9-11,13]. This is consistent with health-belief-model accounts of care-seeking behaviour more broadly, in which perceived susceptibility and perceived severity of a condition are strongly influenced by the presence of noticeable symptoms; a condition without pain, mobility or aesthetic change is systematically under-rated as a health threat, regardless of its true biological severity.

This heuristic has three clinically significant consequences. First, it delays presentation: patients wait for pain, mobility or aesthetic concern before seeking periodontal care, by which point attachment and bone loss may already be substantial and less reversible [8-10]. Second, it misattributes symptoms: when discomfort is present, it is filed under 'sensitivity' — a condition patients regard as manageable and non-threatening — even when the underlying cause is active periodontal attachment loss or post-surgical root exposure [4,17]. Third, it undermines adherence: patients who do not believe they have a disease, because it does not hurt and there is no visible cavity, are less likely to engage consistently with the repeated, long-term maintenance that periodontal therapy requires, since periodontitis, unlike a filled cavity, cannot be 'cured' in a single visit and instead requires ongoing management [13].

3.4 Toward a corrective communication framework

The literature reviewed here suggests that closing this gap requires clinicians to explicitly decouple the two conditions in patient communication rather than assuming that an absence of caries and pain equates to periodontal health. Practical elements supported by the evidence above include: (i) actively screening for and naming gingival bleeding, recession and mobility at every visit, since these are the markers patients are least likely to notice or value themselves [9,10]; (ii) explaining, in plain terms, that periodontitis is usually painless until late stages precisely because it is not a nerve-mediated process in the way that DH or caries-related pulpitis is, so the absence of pain is not evidence of health [1,2,8]; (iii)

explicitly reframing any reported sensitivity as a possible sign of gingival recession or attachment loss rather than a stand-alone nuisance, particularly where recession is present [4,17]; and (iv) linking periodontal status to systemic and functional outcomes (tooth mobility, tooth loss, and associations with systemic disease) to increase perceived severity in the absence of pain as a motivating cue [6,7,12]. Educational interventions of this kind align with calls in the literature for improved oral-health-literacy communication, given the consistent finding that patients with greater disease knowledge report better understanding of periodontal pockets, risk factors and the rationale for maintenance therapy [11].

4. Conclusion

Gingivitis and periodontitis progress largely without pain, while dentine hypersensitivity is, by definition, a pain-driven condition; because patients use the caries model of 'a cavity that hurts' as their default reference point for oral disease, the combination of an intact-looking tooth and an explicable, intermittent sensitivity is frequently misread as reassurance that nothing is clinically wrong. The evidence reviewed here shows that self-awareness of periodontal disease remains low across populations and age groups, that dentine hypersensitivity is strongly and mechanistically linked to gingival recession and periodontal disease and its treatment rather than being an unrelated complaint, and that this combination predisposes patients to delayed presentation, symptom misattribution and reduced engagement with long-term periodontal maintenance. Correcting this misunderstanding does not require new diagnostic technology but a deliberate shift in chairside communication: explicitly separating the painless biological process of periodontal destruction from the pain-driven symptom of dentine hypersensitivity, and actively surfacing bleeding, recession and mobility as the true clinical indicators the patient should be attending to, rather than allowing the absence of a cavity to stand in for the absence of disease.

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Conflict of Interest

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