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Research Article

# Predictors of post-operative pain in geriatric patients with dentin hypersensitivity

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## Abstract

**Background:** Dentin hypersensitivity prevalence declines with age as tubule sclerosis raises the pain threshold, yet symptomatic geriatric patients often carry a larger burden of exposed root dentin from recession and cervical lesions than younger patients [9,14].

**Objective:** This review synthesizes evidence on predictors of post-operative pain after restorative or desensitizing treatment of dentin hypersensitivity in older adults, across local-tissue, psychological, and procedural domains.

**Methods:** We synthesized cross-sectional prevalence studies, desensitizing-treatment trials, post-operative pain prediction studies, and geriatric pain-assessment literature via PubMed, Scopus, and Cochrane, using narrative rather than meta-analytic synthesis given sparse geriatric-specific data.

**Findings:** Anxiety and catastrophizing predict procedural pain [16,17], invasiveness predicts implant and extraction pain [19,20], lesion burden increases exposed dentin [14,15], and cognitive impairment undermines self-report [21,22].

**Conclusions:** Procedural predictors are the most actionable lever available to clinicians managing geriatric dentin hypersensitivity today, while psychological predictors need prospective, cognitively-stratified studies before extrapolation to this population [16,21].

**Keywords:** dentin hypersensitivity; geriatric dentistry; post-operative pain; pain catastrophizing; gingival recession

## 1. Introduction

Dentin hypersensitivity is defined clinically as short, sharp pain from exposed dentin in response to thermal, tactile, osmotic, or evaporative stimuli not explained by other dental pathology, and its accepted mechanistic explanation remains the hydrodynamic theory articulated more than half a century ago: fluid movement within patent dentinal tubules mechanically deforms pulpal nerve endings and triggers pain disproportionate to the stimulus [1]. A widely cited synthesis of the pain mechanisms and aetiology of exposed cervical dentin situates that account alongside the enamel loss, recession, and attachment loss that must precede tubule exposure [2], and subsequent work showed sensitive cervical dentin has measurably more open, wider tubules per unit area than non-sensitive dentin from the same dentition [3]; recent biophysical work extends the mechanism further, demonstrating stimuli-induced directional cation transport through the tubules [4]. Evidence-based overviews situate this mechanistic picture alongside the diagnostic and management pathway used at chairside, from exclusion of other pathology through escalating tiers of intervention beginning with the least invasive effective option [5,6,7], and dentin hypersensitivity also measurably affects oral health-related quality of life, diet, and hygiene behaviour [8].

Dentin hypersensitivity is, by most cross-sectional accounts, a condition of middle age rather than old age. Prevalence estimates cluster around a quarter to a third of dentate adults, but the age gradient inside that figure is consistent across very different populations: hypersensitivity peaks somewhere in the third and fourth decades and declines thereafter, a pattern documented in a five-country European survey of more than three thousand adults [9], a large US dental practice-based research network sample [10], and an Australian private-practice cohort [11]. The usual explanation is physiological: continued deposition of secondary and tertiary dentin, progressive tubule sclerosis, and pulpal fibrosis with age narrow or occlude the fluid-filled channels the hydrodynamic mechanism depends on, so a stimulus that provokes sharp pain in a thirty-year-old tubule often fails to reach threshold in a seventy-year-old one [12,13]. That decline does not make the condition clinically unimportant in older patients; it means those who do present in later life are a selected group whose sclerotic defences have been outpaced by exposure. Gingival recession accumulates with decades of toothbrushing and attachment loss, noncarious cervical lesions deepen with cumulative occlusal loading and erosive exposure, and both expose fresh, less sclerotic root dentin faster than age-related occlusion can compensate [14,15]. The geriatric patient, in other words, typically arrives with fewer patent tubules than a younger patient, but a longer history of surface loss, more root exposure, and comorbid factors: xerostomia from polypharmacy, reduced manual dexterity, cognitive change affecting pain reporting, and a restorative history spanning multiple prior interventions.

Those same patients are the ones for whom post-operative pain after treatment is hardest to predict from first principles; the hydrodynamic model explains baseline sensitivity but not why one geriatric patient reports negligible discomfort after cervical restoration while another reports days of lingering pain from a similar procedure. Anticipatory anxiety and catastrophizing account for a meaningful share of procedural-pain variance across the general dental literature [16,17,18], operative duration and invasiveness shape post-operative pain after implant and extraction surgery [19,20], and age-related changes in pain processing and reporting complicate the picture in patients with cognitive impairment [21,22]. No single study has mapped these psychological, procedural, and local-tissue domains onto geriatric dentin hypersensitivity patients specifically. This review synthesizes what is known, and what remains uncertain, about predictors of post-operative pain in that population.


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## 2. Methods

We searched PubMed/MEDLINE, Scopus, and the Cochrane Library from inception through mid-2026 using combinations of the terms dentin hypersensitivity, post-operative pain, procedural pain, geriatric OR elderly OR older adult, pain prediction, pain catastrophizing, dental anxiety, gingival recession, and noncarious cervical lesion, and hand-searched reference lists of retrieved reviews for additional primary studies. Eligible sources included cross-sectional and cohort studies of dentin hypersensitivity prevalence or associated factors, trials of desensitizing or restorative treatment outcomes, studies of predictors of post-operative or procedural pain in adult dental patients of any age (dedicated geriatric-only prediction studies in dentin hypersensitivity being scarce), and studies of pain assessment or orofacial pain prevalence specifically in older adults or patients with cognitive impairment. Non-English sources, case reports, and studies without extractable findings on predictors were excluded. Because the evidence base directly combining geriatric status, dentin hypersensitivity, and post-operative pain prediction is thin, this review takes a narrative-synthesis rather than meta-analytic approach, organizing findings by predictor domain and drawing inference across adjacent literatures where the geriatric- and dentin-hypersensitivity-specific evidence is itself sparse; this limitation is discussed explicitly rather than papered over with pooled statistics the underlying studies do not support.

## 3. Results

### Baseline prevalence and the age gradient

Across the largest cross-sectional surveys, dentin hypersensitivity prevalence follows a curvilinear pattern with age: low in adolescence, a peak across the third to fifth decades, and a decline thereafter that nonetheless leaves a non-trivial minority of older adults symptomatic [9,10,11]. This pattern is consistent enough across differing populations and diagnostic methods that it reads as a genuine biological signal rather than a sampling artifact, and it sets the baseline expectation for interpreting post-operative pain in a geriatric cohort: the geriatric patient who does present is presenting despite a physiological trend working against symptom expression, which argues for a more exposure-driven or comorbidity-driven aetiology in this age group (Table 1).

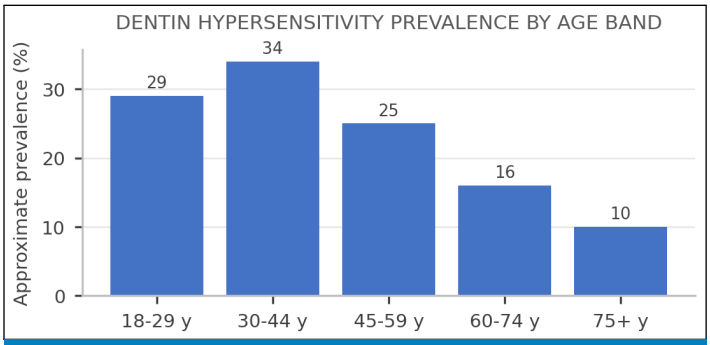


**Figure 1: Approximate age gradient in dentin hypersensitivity prevalence synthesized from cross-sectional surveys, showing a peak across the third to fifth decades and a decline thereafter that nonetheless leaves a symptomatic minority among older adults [9,10,11].**

### Local tissue predictors: recession and cervical lesion burden

Gingival recession and noncarious cervical lesions are the two local findings most consistently associated with symptomatic root dentin in older patients. A digitally scanned cohort study of noncarious cervical lesions in an aging population found lesion dimensions and occlusal risk factors accumulating with age and function in patterns consistent with progressive, multifactorial wear [14], and a comprehensive review of lesion morphology and progression reached the same conclusion about the layered contribution of abfraction, abrasion, and erosion over a lifetime of exposure [15]. The practical implication is that geriatric patients requiring restorative management often have multiple, morphologically heterogeneous lesions rather than a single discrete cavity, increasing the surface area of freshly exposed, incompletely sclerotic dentin manipulated in a single visit, even though direct dose-response data linking lesion count to post-operative pain magnitude specifically in geriatric patients have not


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been reported.

**Table 1: Predictor domains for post-operative pain in geriatric dentin hypersensitivity patients, the evidence source each is drawn from, and the geriatric-specific caveat identified in this review.**

| Predictor domain | Representative evidence | Direction of effect | Geriatric-specific caveat |
|---|---|---|---|
| Baseline DH prevalence/age | [9,10] | Declines after 4th-5th decade | Symptomatic elders may reflect greater exposure burden |
| Recession / NCCL burden | [14,15] | Increases with age and function | More exposed, incompletely sclerotic dentin per visit |
| Anxiety / catastrophizing | [16,17] | Higher scores, higher reported pain | Self-report validity declines with cognitive impairment |
| Procedural invasiveness/duration | [19,20] | Longer/more invasive, higher pain risk | Directly modifiable by staging and technique |
| Cognitive status / pain reporting | [21,22] | Impairment lowers self-report reliability | Behavioural observation needed in dementia subgroup |

## Psychological predictors: anxiety and pain catastrophizing

The general dental literature is considerably more developed on psychological predictors of procedural pain than the geriatric dentin hypersensitivity literature specifically, and it consistently identifies anticipatory anxiety and pain catastrophizing as meaningful predictors. A systematic review and meta-analysis of thirty-five studies found dental anxiety a significant predictor of expected pain, pain during treatment, and pain reported afterward [16], and earlier work established that catastrophizing, a cognitive-affective pattern of rumination, magnification, and helplessness, independently predicted both anxiety and reported pain during a routine dental hygiene procedure, over and above trait anxiety alone [17]. A subsequent functional-imaging study reinforced the mechanism, showing catastrophizing associated with dental pain specifically under situational stress, implicating central pain-modulation pathways rather than peripheral nociceptor sensitivity [18]. Whether these relationships hold with the same strength in geriatric patients is not well established: older patients generally report lower dental anxiety on standardized measures than younger cohorts, but cognitive change, when present, complicates both the experience and the self-report of pain in ways a simple anxiety score does not capture (Table 1).

## Procedural predictors and the geriatric restorative visit

Outside the dentin hypersensitivity literature narrowly defined, the broader post-operative dental pain literature offers the clearest quantitative modelling of procedural predictors, and its findings transfer reasonably well to restorative and desensitizing procedures in geriatric patients even though most underlying cohorts were not age-restricted. A study of post-operative pain following dental implant placement found the risk of moderate-to-severe pain tracked with procedural invasiveness and duration rather than implant number alone [19], and a review of patient and surgical factors predicting post-operative discomfort in implant dentistry identified operative time, flap elevation, and baseline anxiety as recurring predictors [20]. A large practice-based-research-network study using an mHealth platform to track post-operative pain trajectories across dental procedures found pain severity varied systematically by procedure type and by patient-reported pre-treatment expectation, suggesting that setting realistic expectations before a cervical restoration or desensitizing application may itself shape the post-operative pain trajectory [23].

## Desensitizing treatment response as a modifier

Because many geriatric presentations are managed with in-office or take-home desensitizing agents before, or instead of, restorative intervention, treatment efficacy itself predicts whether a patient proceeds to a more invasive, higher post-operative-pain-risk restorative procedure at all. A randomized trial comparing desensitizing toothpastes found meaningful but


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incomplete relief across agents, with a minority of participants remaining symptomatic enough to warrant escalation of care [24]. This creates a two-stage predictive structure worth making explicit: predictors of poor desensitizing-agent response, incomplete relief and high baseline sensitivity, are indirect predictors of exposure to the restorative procedures this review is chiefly concerned with.

**Geriatric-specific modifiers of pain expression**

Two further considerations are specific to the geriatric population. First, a systematic review of dental pain in care home residents found pain common but frequently under-recognized, complicated by communication barriers, staff training gaps, and reliance on behavioural rather than self-report indicators [22]. Second, a systematic review and meta-analysis of orofacial pain prevalence in older adults with mild cognitive impairment or dementia found prevalence estimates that varied but were consistently non-trivial, alongside evidence that self-report pain instruments perform less reliably as cognitive impairment progresses [21]. For geriatric dentin hypersensitivity patients who also have cognitive impairment, a clinically relevant fraction of any general geriatric dental population, prediction models built on self-reported anxiety and catastrophizing scores drawn from cognitively intact adults may simply not apply, and behavioural observation protocols validated in the care-home literature become the more defensible assessment tool.

## 4. Discussion

Three findings from this synthesis deserve emphasis because they cut against an intuitive but oversimplified picture of "the elderly dentin hypersensitivity patient." The first is that lower baseline prevalence with age [9,10,11] does not translate into lower post-operative pain risk once restorative intervention becomes necessary; if anything, the local-tissue literature suggests the opposite, that geriatric patients requiring treatment often carry a larger burden of exposed, heterogeneous root surface than younger patients do [14,15]. Age-related tubule sclerosis may raise the threshold for spontaneous symptomatic hypersensitivity while doing little to blunt the acute post-operative pain generated by cutting, drying, or etching freshly exposed dentin, since that stimulus can exceed even a raised hydrodynamic threshold.

The second finding is that psychological predictors best documented in the general dental pain literature, anxiety and catastrophizing [16,17,18], cannot be assumed to generalize unmodified to geriatric patients: a meaningful subgroup has cognitive impairment severe enough to compromise the self-report instruments those models were built on [21,22]. A model that performs well in a cognitively intact population may fail silently, rather than obviously, where under-reporting is the dominant error mode. This is arguably the most consequential gap identified here: none of the psychological-predictor literature synthesized was conducted in a cognitively-stratified geriatric dentin hypersensitivity cohort, and extrapolation across that gap remains a hypothesis for future study.

The third finding concerns procedural predictors, where the evidence, though drawn mostly from implant and extraction cohorts, is more directly actionable [19,20,23]. Duration, invasiveness, and the extent of surface manipulated are modifiable in a way a patient's anxiety trait or sclerosis is not. Staging work across shorter visits, prioritizing least invasive effective desensitizing approaches before restoration, and setting explicit pre-treatment expectations [23] are interventions a clinician can apply today, independent of the psychological-predictor generalizability gap.

These limitations follow from the state of the underlying literature rather than the review methodology. No identified study prospectively measured post-operative pain in a cohort stratified by both geriatric age and cognitive status, so every cross-domain inference here is


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an extrapolation. Closing this gap would require a prospective cohort enrolling geriatric dentin hypersensitivity patients across a spectrum of cognitive status, recording validated pain measures before and after treatment, and modelling procedural, psychological, and local-tissue predictors jointly.

## 5. Conclusion

Predictors of post-operative pain in geriatric dentin hypersensitivity patients span three interacting domains, local tissue exposure, psychological factors, and procedural invasiveness, but the evidence base for each comes disproportionately from studies not stratified by geriatric age and cognitive status. The clearest actionable finding is procedural: minimizing invasiveness, staging treatment, and managing expectations are levers clinicians can pull now. The clearest gap is the near-total absence of prospective, cognitively-stratified studies testing whether psychological predictors from younger, cognitively intact populations hold in the geriatric patients clinicians increasingly manage as the population ages.

## 6. Declarations

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