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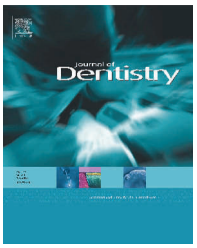

Dentine sensitivity: Past, present and future

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KEYWORDS

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Mouthrinse

ABSTRACT

Objectives: This review defines dentine sensitivity (DS), its prevalence, its aetiology, the mechanism(s) responsible for DS, its diagnosis and its treatment. The review then examines the modes of action of various treatments for DS including potassium salts, strontium salts, bioglasses, arginine/calcium carbonate and professional treatments such as adhesives and oxalates. The methods used to evaluate the various treatment modalities are discussed, including laboratory studies and randomised controlled clinical trials.

Data sources and study selection: A literature search was conducted using PubMed, Ovid Medline and Cochrane reviews for information on DS and its treatments, as well as laboratory and clinical studies used to evaluate the efficacy of various DS treatments. With regard to efficacy of treatments for DS only reports of clinical studies that were randomised, controlled and blinded were reviewed. The authors offer new insights into the shortcomings of the recent systematic review of the use of oxalates for DS.

Conclusions: The authors introduce the concept of a novel desensitising mouthrinse containing 1.4% potassium oxalate: Listerine® Advanced Defence Sensitive mouthrinse. Readers of this supplement issue of the Journal of Dentistry are invited to review the significance of managing the clinical problem of DS. They are also invited to assess data from laboratory and randomised controlled clinical studies in order to understand the advantages offered by regular use of 1.4% potassium oxalate-containing mouthrinse, Listerine Advanced Defence Sensitive, in particular its resistance to daily erosive and/or abrasive challenges.

1. Introduction

Dentine sensitivity (DS) is a global clinical oral health problem in the adult population. It is defined as "pain arising from exposed dentine in response to stimuli, typically thermal, evaporative, tactile, osmotic or chemical, which cannot be ascribed to any other form of dental defect or pathology"[1-4] and satisfies all the criteria to be classified as a true pain syndrome.[5] It is clinically described as a brief, sharp, "bright" type of pain with a rapid onset, although it may also be followed by a dull, aching pain. The pain may be localised or generalised, affecting one or multiple tooth surfaces simultaneously.[6] The definition of DS therefore has two aspects: one describing the clinical presentation and the second identifying the condition by exclusion of other pathologies, highlighting the need for correct differential diagnosis.[7]

Considerable research effort has been invested and expended on understanding the processes leading to DS and on developing effective treatments to alleviate and prevent this painful condition. This article will describe the current understanding of the prevalance and aetiology of DS, and will provide an overview of various management options.

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2. Data sources and study selection

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3. Prevalence: How common is this clinical problem?

It is generally accepted that screening for this clinical condition is not routinely conducted except when prompted by patients, and DS is therefore under-diagnosed and under-treated.[8] Many patients suffer in silence and it is the dental professional's role to identify the problem, make the patient aware of it, address it and manage it. Based on a survey conducted in 2007 by Martin Akel & Associates, Strassler and colleagues reported that 78.7% of dentists and hygienists who responded thought that the prevalence of DS was increasing.[9] When questioned about the single most common cause of DS, 47% thought it was gingival recession, 25% attributed it to abrasion and just 3% thought it was erosion. Seventy-nine per cent thought that erosion and toothwear were increasing in prevalence, while 74.5% thought that the prevalence of gingival recession was increasing. When asked if they thought DS was a challenge to long-term oral health, 78.2% agreed. Over half of the respondents (52.8%) thought that DS was a challenge to general health, 59% thought it was of public health importance and 88% thought that it affected their patients' quality of life.[9]

Studies have demonstrated huge variations in prevalence of DS, ranging from 1% to 98%.[10-13] This broad range does not help in understanding this clinical problem and poses many questions regarding the validity of the methods used. However, if one looks more closely at these studies, it is apparent that they fall into three distinct categories: (a) self-reported assessments, (b) professional/clinical examinations and (c) professional examinations of periodontally involved patient groups.

Self-reported assessments are based on self-administered questionnaires that aim to collect information on demography; consumption of, for example, carbonated drinks; management of DS; and other similar questions.[14,15] Self-report methods have the advantage of providing the patient's individual perspective, however there are a number of limitations associated with this approach, including response distortions;[16-18] variations in the reliability and validity of the instruments used in the surveys;[19,20] and the design and reliable analysis and interpretation of the data derived using these methods.[21,22] These limitations explain in part the large variation (9–84%) in DS prevalence in self-reported assessments.[10-13,15,23-30]

The second category of studies of DS prevalence – clinical examination studies – has reported DS prevalence as ranging from 1% to 34%.[10,24,25,31–36]

Interestingly, the prevalence of DS is found to be much higher in patients with periodontal conditions, ranging from 60% to 98%.[12,31,37] It can peak in the first few days after scaling and root planing or periodontal surgery, and is usually substantially reduced by 8 weeks after the procedure, although the duration can vary from months to more than 30 years.[4] In many ways, this observation is to be expected as patients with periodontal problems have more exposed dentine due to gingival recession, and studies have confirmed that scaling and root-planing procedures in periodontal therapy result in an increase in the number of teeth that respond to painful stimuli,[38,39] as does periodontal surgery.[40–42] Studies have also confirmed that meticulous plaque control reduced DS[39] and that post-operative DS gradually decreased approximately 6 weeks after periodontal surgery.[42]

Peak incidence for DS occurs between the third and fourth decades of life, with subsequent reductions in incidence[34] due to the natural processes of ageing.[43] In general, there appears to be a higher prevalence of DS in women than men,[10,25,44] which may reflect better female oral hygiene awareness.[2] Intra-orally, DS is mostly reported on the buccal cervical surfaces of permanent teeth, with canines and first premolars being the most affected sites and molars the least affected.[45–47]

4. Subjective nature of pain

Another factor that may explain the wide range in self-reported prevalence assessments is the subjective nature of pain. The experience of a sensory event is highly subjective and can vary substantially between individuals.[48–50] In the case of pain, positive expectations can reduce the subjective experience of pain evoked by a consistently noxious stimulus, whereas negative expectations may result in the amplification of pain.[51–54]

Another major disadvantage of self-reported assessments of DS is incorrect diagnosis of the pain by the respondent (patient). One cannot emphasise enough that all other dental diseases with a similar pain should be excluded before confirming the diagnosis of DS, and this can only be done by a clinician. Self-reported assessments of the prevalence of DS need to be interpreted with caution by clinicians and researchers.

Clinical examination studies usually evaluate DS using various quantitative probes. The Yeaple probe and the scratchometer[55] measure tactile sensitivity. Subjective probes, such as the Schiff Cold Air Scale, measure perception of pain from an air-blast stimulus. Subjective sensitivity measurements are often recorded using a visual analogue scale. However, even those studies may be compromised by the patient's subjective perception of pain, which appears to be altered by sensory factors, prompting a heightened pain response.[56]

5. Aetiology and risk factors

Several theories have been proposed in order to explain the biological mechanism of DS, with the hydrodynamic theory[57,58] being the most widely accepted. This states that dentinal fluid flow induced by any perturbation of dentinal fluid within the dentinal tubules activates pulpal nociceptors, resulting in pain.[57,59-61] More specifically, most pain-inducing stimuli (cold, evaporative and osmotic) increase outward fluid flow within the dentinal tubules, causing fluid shear forces over mechanoreceptor nerves in the central end of tubules. This, in turn, activates the intradentinal Aδ nerves at the pulp–dentinal interface, thereby generating pain. So,