Dentine Hypersensitivity and its Significance in the Dental Practice

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Summary

Dentine hypersensitivity is not a new disease. More than a hundred years ago Gysi, 40 years ago Brannström and since then many others have dealt with the problems of its symptoms, pathomechanism, differential diagnosis and therapy. The revived interest today may be attributed to improving oral health and to the presence of more teeth in older age, as well as decreasing caries prevalence. The exposure of dentine surfaces due to gingival recession, erosion and abrasion may cause serious complaints. It occurs in about 40% of the adult population, although, few of them turn to the dentist. Therefore, practising dentists should be aware of the possibilities of treatment, managing strategies and prevention. New aspects include the appropriate timing of toothbrushing after the consumption of acidogenic, erosive foods and beverages, as well as non-invasive (desensitising toothpastes with potassium-nitrate/fluoride) and invasive (reconstruction with fillings, coverage of the exposed roots) treatment options. Continuous care of patients with dentine hypersensitivity is advisable in order to prevent more serious consequences (irritation of the pulp).

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Dentine hypersensitivity, manifested in sensitive and painful teeth, is relatively common. It is not a new disease; over 100 years ago Gysi (1) discussed dentine hypersensitivity in the dental literature, describing the fluid movement in the dentinal tubules. Sixty years later, Brannström's (2,3) investigations in the development of dentine hypersensitivity confirmed the hydrodynamic theory.

Throughout history the ever-changing profile of diseases has also made its mark in dentistry. The improving oral hygiene of populations benefited by reducing such extensively prevalent diseases as caries and periodontal diseases, but at the same time highlighted other, lesser-known problems. The revived interest in dentine hypersensitivity may be attributed to improving oral health and to the presence of more teeth in the middle-aged and older population.

Definition, terminology

The typical symptom of dentine hypersensitivity is a short, sharp pain, caused by stimuli

at the exposed dentine. At macroscopic level, dentine exhibiting hypersensitivity appears no different from non-sensitive dentine; the histopathological state of the pulp has not been clearly established either (4,5). The literature uses various names for this phenomenon. Some argue whether dentine sensitivity or dentine hypersensitivity should be used, later site descriptors were also included in the terminology, distinguishing between root-, cervical- and cemental sensitivity or hypersensitivity (Table 1). Although sensitivity can occur at any area on a tooth, the most common is the exposure of the cervical dentine and that of the root surface (Figure 1). Root sensitivity was adopted at the 2002 workshop of the European Federation of Periodontology to describe the sensitivity of teeth following periodontal surgical procedures. Terminology also disputes ambiguity in the origin and pathomechanism of pain and sensitivity. However, the most accepted term among experts is *"dentine hypersensitivity"*(5).

The definition of dentine hypersensitivity was suggested in 1983 by Dowell and Addy (6). An international team lead by Holland et al accepted this definition and developed the directives of clinical studies in 1977 (7). The definition was also accepted with minor changes by the Canadian Advisory Board, resulting in the following: *"Dentine hypersensitivity is a short, sharp pain arising from exposed dentine in response to stimuli typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other form of dental defect or pathology."*

Symptoms, diagnosis, differential diagnosis

The consensus-based definition of dentine hypersensitivity comprises a first part, which describes the most common forms of the disease and a second part, which refers to the necessity for differential diagnosis. Pain, which is similar to dentine hypersensitivity, can be caused by other pathological conditions, such as (crown or root) caries, chipped teeth or filling, fractured restorations, marginal leakage around restorations or broken cusps. These can all be macroscopically identified, ruled out, diagnosed and easily treated. Dentine hypersensitivity, however, can be caused by other problems, such as gingival recession (Figure 2), which quickly leads to the loss of the cement or dentine "smear-layer," as well as the enamel. The exposure of dentinal tubules to abrasion, erosion, attrition, abfraction or their combination is also manifested in sensitivity and pain (8). During the discussion of the patient's history and the examination, practising dentists should identify pain and sensitivity, caused by dentine exposure.

The first step in diagnosing dentine hypersensitivity is to discuss the patient's history by asking the following questions (9):

- How long has the patient had the pain.
- What kind of pain is it (sharp, dull, throbbing).
- Number and location of sensitive teeth.
- The intensity of pain.
- The nature of pain stimulus.
- Frequency and duration of pain-attacks.

Other factors in the patient's history, which may trigger the pain (previous oral hygiene-, periodontal-, or conservative dental treatment, change in diet or oral hygiene, whitening procedures).

The second step in diagnosing dentine hypersensitivity is to examine the patient and search for certain factors that the differential diagnosis may involve. These (9) may have nothing to do with dentine hypersensitivity or the objective dental pathology may include symptoms of dentine hypersensitivity. The above mentioned factors are the following:

- Non-vital pulp, periapical lesions of the tooth.
- Broken tooth: vertical fracture or fractured cusp.
- Caries.
- Gingival recession, often following periodontal surgery. Old age, mechanical trauma, short frenulum and occlusal trauma can also result in sensitive dentine exposure.
- genetic predisposition to sensitivity – probable family history.
- temporary sensitivity after filling, marginal leakage around restorations.

- conditions manifested in all types of loss of tooth structure – apart from caries –, such as erosion, attrition, abrasion and abfraction, which can be defined as follows (4,5,9,10):

Erosion: the chemical dissolution of the tooth surface caused by acids and not by bacteria, leading to the sensitivity of the exposed dentine and loss of the tooth structure. It can be caused by internal (acid reflux, hiatus hernia, bulimia) and external (acidic diet) factors, the former on the palatal, the latter on the buccal surface of the teeth. Brushing the teeth immediately after consuming carbonated drinks, low-pH coke and fruit juices, as well as acidic food and sweets may destroy the softened enamel or dentine, causing loss of tooth structure and erosion.

Attrition: caused by tooth-to-tooth contact. It may result from parafunctional tooth contact (bruxism), when it would be considered pathological and leads to occlusal dentine hypersensitivity.

Abrasion: the abnormal wear of teeth from extrinsic physical sources, other than the opposing teeth. It is most common around the cervical areas of maxillary canines and premolars. It can be caused by hard toothbrushes or abrasive particles in toothpaste, and usually appears on the left side of right-handed persons and vice versa. Due to the interrelation between abrasion and gingival recession, one may enhance the occurrence of the other.

Abfraction: the concentration of occlusal trauma from parafunction in the cervical region of teeth bends the anatomic crown, causing loss of structure and cracks in the enamel (Figure 3.) These lesions may be extremely sensitive and can even reach the pulp. Combined with abrasion and erosion, it can cause the loss of surface tooth structure.

After confirming or ruling out the above factors, we should check if the problematic tooth (teeth) reacts to stimuli and to what extent. This can be followed by the consideration of treatment.

Prevalence

It is not easy to study the prevalence and incidence of dentine hypersensitivity by using classical epidemiological methods. The abundance of data on prevalence shows extreme variations in figures, depending on the composition of the sample population, the survey location and methods, and so on.

According to the literature on cross-section studies, the prevalence of dentine hypersensitivity is between 3 and 57% (5,11). Prevalence among hospitalised periodontal patients ranges between 72 and 98%. This, however, stems from differences in etiological factors: due to periodontal interventions, it does not match the definition completely and should rather be identified as "root-sensitivity".

On average, dentine hypersensitivity occurs in 40% of the adult population. The age distribution is considerably large: between 20-50 years, peaking between 30-40 years.

Prevalence by gender shows that dentine hypersensitivity is significantly higher among women (Table 2). The teeth most often affected are the buccal surfaces of (upper, followed by lower) canines, first premolars, incisors and molars.

In spite of the above data, only a small proportion – 50% of people with dentine hypersensitivity –actually visit the dentist or dental hygiene specialist (5,11) and only 50% of them receive appropriate or any other treatment.

Mechanisms of the development of dentine hypersensitivity

The hydrodynamic theory– suggested around 100 years ago (1) and confirmed by evidence during the 1950's and 60's (2,3) – postulates that most pain-evoking stimuli, especially cold, increase the outward flow of fluid in the tubules (Figure 4). Through mechanoreceptors the pressure change across the dentine activates intradentinal nerves at the pulp-dentine border. Hot stimuli have an opposite effect: they facilitate the slow, inward flow of dentinal fluid (Figure 5.). The generated flow potentials are in proportion with the pressure change and may activate the A-delta nerve fibres of the dentine (5).

According to the hydrodynamic theory, the surface of the dentine contains open dentinal

tubules. Scanning electron microscopic studies suggest that dentinal tubules on the hypersensitive dentine surface are greater in number and wider in diameter, compared to non-sensitive dentine (Table 3). The tubule numbers and diameters increase from the outer dentine towards the pulp. Fluid flow is proportional to the fourth power of the radius; therefore doubling the diameter induces a 16-fold fluid flow (12).

Based on the above, tooth-wear processes result in the exposure of more and wider dentinal tubules, increasing the pain sensation – assuming that reparative processes are not triggered in the dentine, leading to its sclerotisation.

Etiology

The occurrence of two factors leads to dentine hypersensitivity: dentine exposure and the opening of dentinal tubules.

Dentine may be exposed through loss of enamel or periodontal tissues, the latter termed as gingival recession. Both phenomena may result from the combination of physical and chemical impacts.

For a long time dentine exposure was thought to be the result of inadequate oral hygiene techniques: namely toothbrushes. Today the focus has shifted to the abrasive effect of toothpastes; this is insignificant on its own, but may be included in the etiology when combined with other factors. Erosion is likely to cause buccal cervical lesions, intrinsic and extrinsic acids may enhance the abrasive impact of toothpastes and open the dentinal tubules by removing the "smear-layer."

Dentine sensitivity caused by inadequate techniques or abrasive toothpastes usually occurs on the left teeth [opposite the person's dominant side] and is more common among women, since they are known to have better oral hygiene. According to the literature, plaque-index figures are inversely proportional to dentine sensitivity (5).

Abfraction can also damage the tooth. Due to stress on gingival edges, apatite crystals at the cervical area become more susceptible to chemical (erosion) and mechanical (abrasion) forces, resulting in wedge-shaped defects, especially on canines and premolars (5).

Tooth wear caused by erosion, abrasion and abfraction is a slow process, cumulating and usually undetected throughout many years. In time, however, all these impacts may lead to the opening of dentinal tubules, which is the key factor of dentine hypersensitivity (8).

Periodontal disease and related therapies contribute to gingival recession and dentine exposure, especially in the cervical area. Forceful subgingival depuration may provoke the opening of the tubules or even bacterial infection, leading to root sensitivity in the pulp, which is manifested in inflammation.

Even after the loss of the enamel and the root's thin cement layer, the dentine is still protected by a "smear-layer," covering the dentinal tubules, and the opening of the tubules is protected by calcium phosphate deposits from the saliva. During dentine exposure, toothpastes with abrasive components or detergents (sodium lauril sulphate) destroy this protective layer, thus opening the dentinal tubules. Erosion can also trigger dentine exposure and pain.

Therefore, the definition suggests that dentine hypersensitivity can be diagnosed, but internal and external causes and impacts need to be explored thoroughly.

Therapeutic options

The treatment of dentine hypersensitivity has been heavily treatment based for decades. Our present knowledge suggests that – similarly to other diseases – management strategies together with treatment are more successful. The management-treatment strategy (Addy: "management strategies") (5) comprises the following steps:

- Diagnosis, which is compatible with the disease definition.
- Differential diagnosis: identify and treat other conditions that induce symptoms similar to dentine hypersensitivity.

- Identify and eliminate etological and predisposing factors, particularly with respect to erosion and abrasion (dietary history and oral hygiene habits).
- Recommend treatment appropriate to the patient's individual needs.

Treatment: There are two treatment options for dentine hypersensitivity, to occlude dentinal tubules, thereby blocking the hydrodynamic mechanism and/or to block neural transmission at the pulp. Management methods, agents and materials can be reversible and non-reversible (Table 4.).

Reversible methods

Desensitising toothpastes. Potassium and strontium oxalates, as well as fluorides have been used for decades to reduce the sensitivity of exposed dentine. Potassium- and ferric oxalates and also strontium-chloride block open dentinal tubules, thus reducing the fluid flow. Potassium nitrate, which has been used for a long time to treat dentine sensitivity, passes easily through dentine, all the way into the dentinal tubules and depolarises the nerve surrounding the odontoblast process. In Hungarian literature, Sugár's "Oral diseases" (13), published in 1959, recommends potassium salts as part of the Gottlieb brushing treatment for cervical sensitivity. Although, according to a meta-analytical study of the prevailing "evidence-based medicine", the effectiveness of potassium nitrate is yet to be proven by clinical studies universally (14), toothpastes with 5% potassium nitrate and fluoride are successfully used to treat dentine sensitivity in the USA and in Europe.

As mentioned above, desensitising toothpastes contain fluoride. In the USA, toothpastes with very high fluoride content (5000 ppm) are available on prescription and used for treatment effectively (9).

Some dentine bonding materials, such as HEMA/glutaraldehide products, act by blocking the tubules through protein precipitation.

Jerome (15) and Haywood (16) recently described a new method for using desensitising toothpastes by placing the 5% potassium nitrate toothpaste in a custom-made tray. They determined that 10-30 minutes of wear every day for several weeks alleviates symptoms. This method is recommended before and after bleaching treatments to prevent or reduce dentine sensitivity.

Non-reversible methods include grinding in order to restore occlusion in occlusal trauma. When filling is used to restore the loss of cervical structures, presenting dentine sensitivity, abfraction forces must also be eliminated. The incomplete cervical root surface may be covered with glass ionomer or composite restorations, paying particular attention to avoid marginal leakage and plaque formation, which may cause periodontal complications and gingival recession in the future. American authors describe surgical methods to cover the exposed cervical areas, but in such cases previous cervical filling is contraindicated (11). Long-term results of these surgical procedures, however, are yet to be proven.

As a final resort, we can opt for pulp extirpation, root canal filling or the removal of the tooth.

Prevention

As any other disease, dentine hypersensitivity can also be prevented. We should focus on three areas to avoid dentine exposure and hypersensitivity: oral hygiene, periodontal interventions and bleaching.

Gingival recession, which is prevalent in the adult population, is a result of poor dietary habits and oral hygiene. Tooth enamel is softened and eroded by acidogenic, low pH food and beverages, and the enamel is further damaged by forceful brushing immediately after eating. Some authors recommend (5,11) that patients should avoid brushing their teeth immediately after consuming acidic (especially sour and carbonated) drinks.

Gingival recession, leading to exposed dentine, can be the result of oral hygiene and

periodontal interventions, such as supra- and subgingival depuration, root polishing and periodontal surgery. Particular attention must be paid during these procedures not to damage gingival tissue margins and the cervical areas.

Currently used popular bleaching techniques can also cause dentine exposure and sensitivity. As mentioned previously, it is recommended to use desensitising toothpastes before, during and after bleaching treatments.

All of the above indicates that primary prevention of dentine hypersensitivity is possible and must be used in dental practice in order to avoid tiresome, long-term treatment and care.