Dentine hypersensitivity
From diagnosis to treatment

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Dentine hypersensitivity is defined as pain arising from exposed dentine in response to thermal, chemical, tactile or osmotic stimuli, which cannot be explained as arising from any other form of dental defect or pathology (Addy & Unrquart 1995). Such hypersensitivity is common: in a 1987 survey of a group of patients in the UK, Orchardson found that 74 percent suffered from some form of hypersensitivity. He also noted a very early peak of prevalence between 20 and 25 years. However, Addy (1992) reported a peak between 20 and 50 years, and Fisher (1992) reported a peak between 40 and 49. The teeth most commonly affected were the canines and upper premolars, followed by the upper first molars. It was also reported that in patients with periodontal disease, molars were more frequently affected.

It has been suggested that the incidence of hypersensitivity will increase as humans’ longevity increases and they retain their teeth until even more advanced ages; however, this is not supported by epidemiological studies. It is true that gingival recession and loss of cementum are uncommon in older adults, but aged dentine is also less permeable, owing to the deposition of sclerotic and secondary dentine. Dentine hypersensitivity, while not a serious dental problem, can be an uncomfortable and unpleasant experience for the patients and can lead to modification of behaviour, such as avoiding brushing of the affected areas, which in turn has a negative impact on oral health.

Dentine is a very permeable tissue, it contains a dense network of dentinal tubules, which are essentially highways connecting the external environment to the pulp. In a young person, odontoblast send processes deep into tubules (Fig. 1), with the remaining space filled with extracellular fluid. A prerequisite of dentine hypersensitivity is the exposure of dentinal tubules, with their patent orifices (Fig. 2), to the oral environment.

Dentine is normally covered by enamel or cementum, which can be removed by attrition, abrasion or erosion. It can also be removed by aggressive tooth brushing or root planning. One can explain the high incidence of sensitivity after periodontal treatment with exposed dentine due to both gingival recession and aggressive root planning. Another reason for exposed dentine is a developmental anomaly in the cervical region when enamel and cementum do not meet during the development stage. However, dentine hypersensitivity is often the result of a combination of the above factors.

It is a common belief that enamel is an impermeable tissue; however, a study in a microstructure showed that it contains space around the crystals and prisms (Fig. 3), which is normal.

Neural theory

Nerve endings are present in the dentinal tubules, some mechanical and chemical stimuli trigger the pulpal nerve fibres directly.

Hydrodynamic theory

This theory was proposed by Brannstrom and co-workers and is the most supported theory. The dentinal tubules are fluid filled and the fluid is disturbed by thermal, mechanical and osmotic changes on the surface of dentine, the hard receptors are stimulated, which leads to discharge of nerve endings. Accordingly, dentine hypersensitivity tends to stand out in poor oral hygiene and subsequent plaque accumulation on root surfaces. It could be possible that stagnant plaque leads to demineralisation with a smear layer and opening of dentinal tubules. These early demineralised areas tend to be softer and more discoulored than the surrounding areas (Fig. 4).

Treatment options

The treatment options for dentine hypersensitivity can be broadly grouped into the desensitisation of nerve endings and the plugging or covering of the dentinal tubules. These options with their relevant active ingredients are listed in Table 1. The high prevalence of dentine hypersensitivity led to the development of a surprisingly large number of products designed to alleviate this clinical problem. All available treatments appear to work; however, in recommending a treatment to patients, dental professionals should consider the needs of the individual, in order to maximise compliance.

Nerve desensitisation

There is much evidence to indicate that products containing potassium nitrate are effective in controlling dentine hypersensitivity. Tarbet et al. (1980; 1981; 1982) demonstrated in well-conducted clinical trials that, with daily use, a toothpaste containing 5 percent potassium nitrate is effective in desensitising for up to four weeks and that potassium nitrate does not induce changes in the pulp. It was suggested that the potassium (K+) ions block nerve impulses by interfering with the sodium (Na+) pump and depolarisation of nerve cells.

In the case of potassium oxalate, it is postulated that in addition to the effect of K+ there is also some plugging of the dentine tubules with

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Nerve desensitisation Potassium nitrate Self-application, daily use

Minerals/Salts

TCP Self-application, daily use

CPP-ACP Self-application, daily use

Fluoride varnish Professional application

Dentine bonding agent Professional application

Nerve desensitisation

Potassium nitrate Self-application, daily use

Minerals/Salts

TCP Self-application, daily use

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calcium oxalate salt, which decreases dentine’s permeability.

Nerve desensitisation works best in patients with generalised, mild to severe dentine hypersensitivity. An active ingredient is built into a toothpaste; compliance is normally high because no extra step is added to the daily oral-care routine.

Dentine surface cover

When dentine hypersensitivity is severe and localised, patients may not be able to brush the affected areas well. It is important to protect these areas temporarily with a physical barrier, such as a varnish or thin glass ionomer, in order to desensitise and allow better cleaning. This method should also be combined with the use of a product containing potassium nitrate.

The use of a dentine bonding agent has been advocated, as it can provide short-term relief, but the seal provided by a dentine bonding agent, especially the single-bottle version, does not last long. Dentine bonding agents are not designed to be exposed to the oral environment and should not be utilised in an off-label fashion.

CPP-ACP-containing products, such as Tooth Mousse or MI Paste, are also good desensitisers. These products work instantly after direct application to the affected areas. The mechanism of action has not been elucidated, but it has been suggested that the protein component of CPP-ACP, casein phospho-peptide, forms a protective physical barrier over exposed dentine.

Plugging of dentinal tubules

There is evidence to support the use of the various active ingredients listed under the minerals/salts sub-heading. Most of the products containing these ingredients are for daily home use, and compliance is thus an issue that clinicians will need to manage.

The application of formaldehyde or glutaraldehyde should be done with caution, as these are strong tissue fixatives and much safer alternatives for the treatment of dentine hypersensitivity are available.

Conclusion

Dentine hypersensitivity is a common dental problem that can be managed successfully, using a wide range of in-office procedures and at-home products. When it is mild and generalised, the condition can be treated using toothpaste containing potassium nitrate or potassium oxalate as the active ingredient. Potassium nitrate, stannous fluoride and strontium chloride are active ingredients specifically designed for dentine hypersensitivity. Only in severe and localised cases should in-office procedures be used in complement at-home treatment.

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