A product containing casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) has also been used with little efficacy[2,13]. Most desensitizing agents act by blocking open dentine tubules while potassium nitrate has a depolarising effect causing disruption of pain transmission[2,5,6]. A Cochrane review, however, suggested that there was not sufficient evidence to corroborate the effectiveness of potassium-containing toothpastes for management of dentine hypersenisitivity[14]. More recently, new desensitizing toothpaste containing arginine and calcium carbonate as active ingredients has been claimed to occlude dentine tubules offering relatively fast and effective treatment for dentine hypersensitivity[11,15,16]. It has also been demonstrated that the arginine-containing toothpaste provides an instant relief when applied directly to the sensitive teeth and that its efficacy is superior to desensitizing toothpastes containing potassium ion as the active ingredient[15,16].

Control measures

Dental practitioners can advocate the following measures, which may be effective in the prevention and control of dentine hypersensitivity in their patients.

- Avoid vigorous toothbrushing techniques that damage the teeth and supporting structures.
- Avoid using a toothbrush with hard filaments. Always use a toothbrush with soft filaments.
- Reduce the frequency of taking acidic foods and drinks by confining to main meals.
- Use a straw to drink acidic beverages.
- Rinse the mouth with water and avoid brushing for at least 30 minutes after any acidic challenge to teeth.
- Maintain good oral hygiene and initiate periodontal treatment where necessary.
- Wear night-time splints to minimise tooth wear associated with parafunctional habits like bruxism.

Conclusion

As a consequence of increased life expectancy and a foreseeable decline in tooth mortality, people would be more likely to retain their teeth for longer, which would be vulnerable for tooth wear, and hence it would not be unrealistic to expect that dentine hypersensitivity would become a more common clinical entity in the future than what we experience/d present or past[2,6]. Studies suggest that dentine hypersensitivity is regarded as an enigma because it commonly occurs yet is inadequately understood[1,11]. While there may be much left to comprehend, the growing body of our scientific knowledge on this problem has enabled us to develop comprehensive management strategies for it[11,15,16]. As long as dental health care professionals are updated, confident and competent with such management strategies, dentine hypersensitivity will no longer be an enigma.

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Sensitive Teeth

Dentine hypersensitivity – is it an erstwhile problem or a modern-day enigma?

Tooth sensitivity has been one of the key topics among dental researchers in the recent past[1–8] even though one of the earliest reports on dentine hypersensitivity goes as far back as the 16th century[6,8]. Despite being extensively studied, dentine hypersensitivity can be considered as a frequently encountered clinical entity, which has not yet been clearly understood by dental practitioners[1,3,7,8]. This information sheet attempts to address some of these issues.

What is dentine hypersensitivity?

Dentine hypersensitivity is defined as a distinctive short sharp pain arising from exposed dentine characteristically in response to an array of stimuli including thermal, tactile, evaporative, osmotic or chemical, which cannot be attributed to any other form of dental defect, disease or pathology[3].

- Pain should come from exposed dentine.
- Pain should not be ascribed to any other dental disease. Hence, all other dental diseases with a similar pain should be excluded before confirming the diagnosis of dentine hypersensitivity.

Terminology

The term "dentine/dentin hypersensitivity" has been widely used and accepted for a long time both by clinicians and researchers alike. Other commonly used terms include dentine/dentin sensitivity, dentinal hypersensitivity/sensitivity, cervical hypersensitivity/sensitivity, root hypersensitivity/sensitivity and cemental hypersensitivity/sensitivity[1].

Epidemiology

It has been estimated that well over 40 million people in the US and up to 30% of adults at sometime during their life are affected with dentine hypersensitivity[4]. A huge variation in the reported prevalence of dentine hypersensitivity ranging from 1.1% to 98% has been ascribed to various methodologies adopted by different studies[7,8]. However, according to general scientific consensus, between 10% and 30% of the global population are affected with this condition[2,5,6,9,10]. Latest reports indicate that dentine hypersensitivity represented 9.1% of the weekly patient load of private dental practitioners in Australia with 2.3 teeth per person and 1.2 surfaces per tooth affected on average[8]. Dentine hypersensitivity has been shown to affect more females than males while peaking between the third and fourth decades of life with a subsequent reduction in its occurrence thereafter[8,10]. With people retaining teeth longer, the elderly would expect to have higher rates of gingival recession as well as loss of enamel and cementum and, consequently, more dentine hypersensitivity. However, they would be less likely to complain of hypersensitive teeth because of reduction in neural sensations and dentine permeability as well as sclerosis and occlusion of dentinal tubules, which might be associated with the natural ageing process[2].

Aetiology

Dentine hypersensitivity has a multi-factorial aetiology – interaction between many factors including predisposing factors and triggers (stimuli) play an important role in establishing it[1,2,5,6]. Gingival recession, abrasion, erosion and attrition are among the main predisposing factors whereas cold, as well as air stimuli, and dietary acid are considered to be important triggers. While tooth whitening, periodontal surgery and restorative treatment are some of the less common predisposing factors, touch and hot stimuli are regarded as occasional triggers. Latest findings suggest that dietary acid was the only trigger whereas gingival recession and erosion were the predisposing factors that were significantly associated with greater dentine hypersensitivity levels in a private practice patient population in Australia[8]. Gingival recession can occur in both healthy gingiva and periodontal disease – the former would be most often seen in buccal surfaces of dentine hypersensitivity patients who have overenthusiastic brushing habits

while the latter could be linked to hypersensitivity anywhere around the root (also known as root sensitivity) in patients with periodontal disease and those who have undergone periodontal treatment[5,6]. More recently, periodontal attachment loss per se has been suggested as an early indicator of dentine hypersensitivity[10]. Two phases have been proposed to be involved in dentine hypersensitivity: loss of enamel or gingival recession causes dentine exposure (lesion localization), which should be followed up by opening of dentine tubules (lesion initiation) mainly via erosion and abrasion[1,3,11].

Biological mechanism

The currently accepted mechanism for pain from dentine hypersensitivity is the hydrodynamic theory[12]. According to this theory, whenever the exposed dentine comes into contact with a stimulus, there will be an increased fluid flow in the dentine tubules. This in turn causes an alteration in pressure across the dentine and excites a pressure-sensitive nerve receptor. Thereafter, activation of intradental nerves at the pulp-dentine border or within the dentine tubules transmits the stimulus evoking pain. This theory suggests that dentine tubules should be open at both the dentine surface and pulpal surface of the tooth to exhibit a response to the stimuli. Accordingly, the number of open tubules and their diameter are considered important factors in initiating pain from dentine hypersensitivity[1,2,5,6]. In other words, the higher the number and greater the diameter of the open dentine tubules the more intense will be the pain from dentine hypersensitivity. It has been postulated that triggers such as cold stimulate fluid to flow away from the pulp creating more rapid and rigorous neural responses than stimuli like heat, which cause somewhat sluggish fluid flow towards the pulp[1,3,11]. This is in line with the observation that dentine hypersensitivity patients more frequently complain of pain in response to cold stimuli than heat[1–6, 8–11].

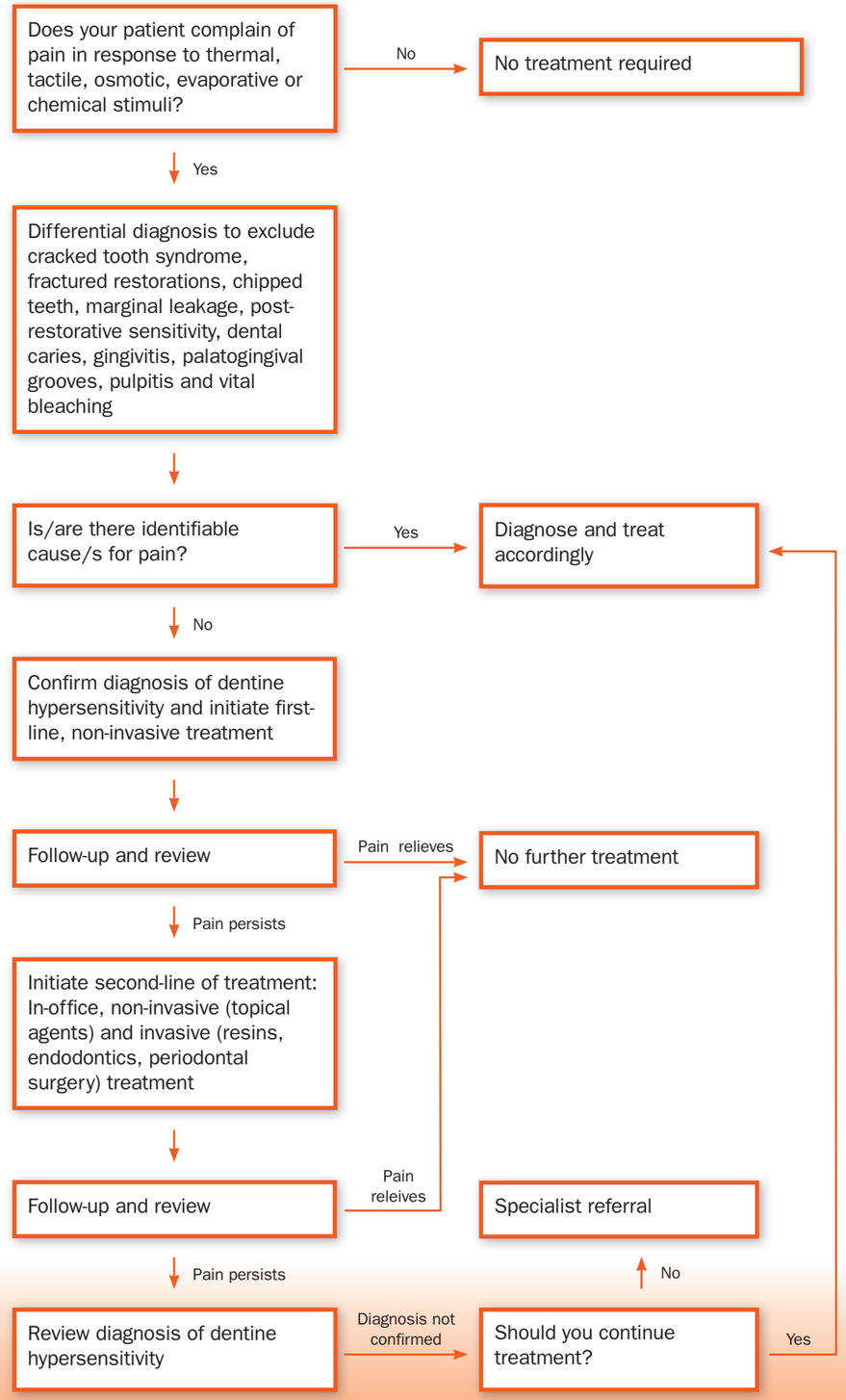
Clinical presentation

Dentine hypersensitivity patients usually complain of and present with discomfort, pain and inability to brush their teeth on receiving stimuli including cold, air, acid and touch[7,8]. Difficulties in eating and sleeping are some of the less frequent symptoms of dentine hypersensitivity[8]. While dentine hypersensitivity is most prevalent in maxillary premolars and molars, buccal surfaces are the worst affected sites[1–6,8–10]. Intra oral distribution of dentine hypersensitivity may resemble that of gingival recession in right-handed individuals where premolars as well as teeth on left and buccal surfaces are commonly affected in comparison to other teeth and sites in the mouth[1,5,6,8].

Diagnosis

The diagnosis of dentine hypersensitivity should be based on detailed history taking and clinical examination. The most commonly used diagnostic tools are blasting air or water using an air-water syringe (thermal method) and scratching the tooth surface with a sharp dental explorer (tactile method)[4,9,10]. Air blast, which includes both thermal and evaporative elements of stimuli, may simulate a real-life situation experienced by a dentine hypersensitivity patient rather than probing with a dental explorer[10] whereas thermal stimulation would be considered more effective than tactile in detecting dentine hypersensitivity[9]. It is very important to consider a differential diagnosis to exclude other conditions

Figure 1: **Flowchart for the diagnosis and management of dentine hypersensitivity**



such as cracked tooth, fractured restorations and chipped teeth that mimic dentine hypersensitivity[1–3,5–7] (Table 1). It would be also relevant to take a detailed dietary history and information on oral hygiene practices including toothbrushing technique, frequency, duration and timing of brushing as well as frequency of toothbrush change and appearance of brush at change[1]. A comprehensive oral examination, sometimes, coupled with a radiographic investigation may be necessary to confirm the diagnosis[3].

Table 1: **Conditions to be excluded in the diagnosis of dentine hypersensitivity**

• Cracked tooth syndrome	• Dental caries
• Fractured restorations	• Gingival inflammation
• Chipped teeth	• Palatogingival grooves
• Marginal leakage	• Pulpitis
• Post-restorative sensitivity	• Vital bleaching

Screening

Given that screening plays a critical part in the establishment of dentine hypersensitivity diagnosis, dental practitioners are encouraged to employ screening for dentine hypersensitivity as a routine measure in clinical practice. This would minimize under-diagnosis and under-treatment of the condition[3].

Management strategy

Management strategies of dentine hypersensitivity are broadly classified as non-invasive and invasive. The currently accepted guidelines suggest that the first-line treatment of dentine hypersensitivity comprises the non-invasive strategy where recommendation of desensitizing agents for home use should be coupled with removing or modifying predisposing factors for dentine hypersensitivity. To maximize the benefits of this management strategy, regular brushing, twice daily, with desensitizing toothpaste is necessary until the symptoms of dentine hypersensitivity alleviate[3,5]. Accordingly, invasive management strategies such as periodontal surgery and endodontic treatment should be confined to the minority of dentine hypersensitivity patients who do not favourably respond to first-line treatment[1–3,5,6]. Follow-up of dentine hypersensitivity patients is an essential component of the management strategy to review the diagnosis and/or for specialist referral where appropriate[3]. Figure 1 illustrates a flowchart for diagnosis and management of dentine hypersensitivity.

Desensitizing agents

Dentifrices containing different desensitizing agents as active ingredients are widely used these days for management of dentine hypersensitivity and consequently are popular among both dental practitioners and dentine hypersensitivity patients alike. Potassium nitrate is considered to be the most extensively available active ingredient in desensitizing toothpastes followed by stannous fluoride[5–7]. Other less commonly available active ingredients include sodium fluoride, stannous fluoride, strontium chloride and sodium monofluorophosphate. In addition to toothpastes, these ingredients are incorporated in products such as mouthrinses, varnishes and solutions[5].