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The rise of **Dentine hypersensitivity and tooth wear in an ageing population**

Abstract

Our understanding of the aetiology of **Dentine Hypersensitivity (DH)** has changed dramatically over the past few decades. It is no longer an enigma, but other problems exist. The prevalence of DH in the **world** and in particular in the UK is increasing, **predominately** due to **increases in** tooth wear and the erosive dietary intake in the younger population. **DH is increasingly reported in all age groups and is shown to provide clinical indication of an active erosive tooth wear. As the population ages and possibly retain teeth for longer, the likelihood of tooth wear and DH could increase.** This paper describes the prevalence, aetiology, diagnoses **and management** of DH **in relation to tooth wear.** The aim is to raise awareness and to help **inform a prevention strategy in an ageing population, which starts from younger age groups.**

Introduction

The presentation of patients with sensitive teeth (or dentine hypersensitivity DH), anecdotally, appears to be increasingly common and this is similarly backed up in the literature.¹⁻³ **DH** has been defined as 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 dental defect or disease.⁴ DH has been shown to significantly affect the quality of patients' lives.⁵

Prevalence

In a recent observational study of DH, the largest of its kind in Europe, seven nations (including the UK) discussed how to record the prevalence of DH and tooth wear. After calibrations, a group of 3187 patients aged 18-35 years attending general dental practice across Europe were assessed for the prevalence and risk factors of DH between 2011 and 2013.² A combination of clinical examination and patient questionnaires were used. This study showed a DH prevalence of 42% across Europe, when applying a stimulus to teeth in a clinical setting. Worryingly, subjects with DH were far more prevalent in the UK compared to continental Europe.² In a UK study conducted around the same time in the South East of England, 55% of subjects attending for a regular dental examination had DH on at least one tooth surface.³ In contrast, in an earlier study, Rees and Addy 2004 reported a considerably lower prevalence of DH (2.8%) in a group of 5,477 patients attending for a regular dental examination at General Dental Practices across the UK.⁶ Diagnosis of DH was similarly confirmed via a clinician applying cold air to patients' teeth, and then patients were given a questionnaire. Looking at this data, it appears the proportion of subjects with DH in the UK has risen dramatically and this can be seen anecdotally in our daily practices.

The reason why there are differences in the reported prevalence of DH can be due to the method of reporting. However, the dramatic increase in prevalence can be explained from the changes in lifestyle and increase in certain risk factors linked with DH. The European study investigated these risk factors.² It was concluded that there is a relationship between the prevalence of DH and erosive tooth wear in particular. There were also associations with loss of **gingival** attachment, medications that reduce salivary flow rate and smoking.² Currently it is not known specifically which of the risk factors plays the greatest role in DH.² In regards to smoking, tobacco control has come under a lot of focus in Europe and the UK during recent decades, but needs more attention **and input from the dental care profession**^{7,8}. **Another major cause was erosive wear due to dietary acid intake.**² **Indeed, tooth wear has been shown to be very prevalent in Europe recently in subjects aged 18-35 year olds.**⁹ In this study, the prevalence of tooth wear was 57%, with 29% of tooth surfaces having a tooth wear defect.⁹ In similarity to DH, there were strong associations between dietary erosive beverages and tooth wear.⁹ More worryingly, the proportion of tooth wear in the UK was higher compared to continental Europe⁹, in similarity to DH. In particular, research conducted in the South East of England at the same time as the European studies^{2,9}, showed that the prevalence of tooth wear was 91%, with 46% of tooth surfaces having a tooth wear defect.¹⁰ In the UK, the increase in tooth wear was previously demonstrated in the 2009 Adult Dental Health Survey (ADHS).¹¹ Compared to the prior ADHS conducted in 1998¹², the prevalence of tooth wear in the UK had increased from 66% to 76% in just ten years and moderate tooth wear had increased from 11% to 15% particularly in younger age groups.¹¹ The latter is more worrying for the UK **and predicts further increases in tooth wear as the population ages.**

It appears from the literature¹³⁻¹⁵ that the increase in frequency of erosive beverage consumption in the UK has contributed to a rise in both tooth wear and DH **over the years. Indeed, the consumption of more than just three acidic food or drinks per day have been shown to contribute to an increased risk of developing erosive tooth wear and frequency.**¹⁶ **Looking to the future, these changes must be considered together with the fact we have an ageing population, who might retain teeth for longer**^{11,17} **and the likelihood of DH affecting more teeth throughout the patient's lifetime. The possibility of individuals retaining more teeth should be balanced together with the fact that extractions are higher in some younger individuals, due to a rise in caries prevalence in high risk groups.**¹⁸ **Nonetheless, recent studies, conducted outside Europe, support the increase in DH in older populations.**¹⁹ **In a 2017 study from China, the prevalence of DH recorded in individuals aged 20-69 years was 33.7% from questionnaires and 25.5% from clinical examination. The prevalence was highest in females than males and was highest in the age group 50-59 year olds (39.3%).**¹⁹ **These findings have been shown previously, again amongst older individuals, when individuals of all age groups were assessed for DH.**²⁰

In addition to tooth wear, patients with periodontal disease and gingival recession will likely have more DH. In a group of 277 patients attending General Dental Practices, the prevalence of DH has been reported via the use of questionnaires as 52%.²¹ It was also noted that DH peaked in the 3rd decade and was reported more in females.²¹ The high prevalence was possibly due to the fact the patients had seen a hygienist and received tooth debridement. Other studies show DH even may be up to 98% in patients referred to a specialist periodontology department for treatment²².

Aetiology

Surface phenomenon

For the purposes of explaining the aetiological basis of DH, we must consider that in order for DH to occur, dentine has to be exposed. The terms Lesion Localisation and Lesion Initiation have been proposed when considering this. Lesion localisation involves the loss of either enamel or gingival recession, which exposes dentine.²³ The loss of enamel is generally associated with the condition of tooth wear. The exposure of dentine leads to the second stage in the pathophysiology of DH – lesion initiation.²³ This involves the exposure of dentine tubules that must be patent from the surface of the dentine to the pulp.

Classic laboratory studies have examined the surface features of sensitive and non-sensitive dentine surfaces (from teeth recently extracted for orthodontic purposes).²⁴ Compared to non-sensitive cervical dentine, sensitive cervical dentine has **been** shown to have wider and more numerous dentine tubules where 0.83µm has been suggested as the minimum diameter necessary of a dentine tubule to be involved with DH clinically.²⁴ The presence of reactionary or tertiary dentine will also play a role, depending on the rate of progression of the tooth wear, size of the lesion, age of the patient etc. **However, as mentioned, DH is frequently reported in older populations and although numbers of tubules may reduce with age, patency from the surface to the pulp is maintained given the huge number of dentine tubules traversing dentine, in particular closer to the pulp. An important aspect, for both aetiology and management, is therefore the smear layer covering the dentine surface.**²³ On a compositional basis, the smear layer is comprised of a thin and loose layer.²⁵ The matrix of this layer is composed of organic collagen and glycosaminoglycans that forms an adherent phase for mineralised tissue arising from saliva and dentine particles that might occlude dentine tubules.^{25,26} The thickness of the smear layer has been suggested as 2-5µm²⁷ and it can form when tooth structure is cut by either hand or rotary instruments.²⁸ The smear layer (on dentine recently brushed with toothpaste) can be seen in Figure 1. **Olley et al. 2015** showed that the presence of a smear layer and **particulate** deposits (following brushing with various toothpaste formulations) occurs near to and within 5µm ± 2µm of the surface of dentine.²⁹ **Taken together, the aetiology of DH is therefore often referred to as a surface phenomenon.**²⁹

Tooth wear and DH

DH has been reported on all worn tooth surfaces; occlusal/**incisal**, as well as buccal and **lingual**.^{15,13} **Tooth wear has been defined in 2017 as pathological as opposed to physiological when it is atypical for the age of the patient, causing pain or discomfort, functional problems or deterioration of aesthetic appearance, which, if it progresses, may give rise to undesirable complications of increasing complexity.**³⁰ **Pathological tooth wear, due to its speed and inability of the pulp to lay down sufficient dentine, most commonly causes DH in contrast to physiological tooth wear.** It must be remembered that tooth wear is generally a multifactorial condition that can be subdivided into erosion, attrition and abrasion. Erosion has been defined as the chemical dissolution of tooth substance without the presence of plaque, attrition as wear that occurs from tooth-to-tooth contact

without the presence of food and abrasion as wear that occurs by the friction of exogenous material that is forced over the surfaces of the tooth.³¹

Erosion and DH

Erosion of dentine with acidic beverages **leads** to the loss of the smear layer (Lesion initiation), opening of the dentinal tubules and initiation of a DH lesion, **visualised microscopically on dentine samples**, very readily.³² This has been shown clinically by Olley *et al.* 2015 in which there were **statistically significant associations** seen between tooth wear lesions and the presence of DH, **and between these diseases and the frequency of consumption of acidic beverages.**¹³ **The presence of tooth wear in a patient made them 40% more likely of having DH and tooth surfaces associated with DH also had tooth wear.**¹³ Interestingly, amongst patients who consumed an acidic beverage within 60 minutes of their appointment, 87.2% (n=130) had DH. In contrast, amongst subjects who had consumed the acidic beverage more than 1 hour previously, the prevalence of DH was 12.8% (n=19). Thus, **reducing the frequency at which a patient consumes an acidic beverage appears to reduce DH (and tooth wear)**, by preserving the smear layer on the teeth. This clinically supports the episodic nature of DH (which is also affected by patients own varying coping mechanisms²). **It also shows that if dentine surfaces are exposed, then the only clinical indicator of active erosion is the loss of smear layer and subsequent pain resulting from DH.**

Tooth brushing abrasion and DH

Another important aspect of the smear layer is the role of tooth brushing. In practice, we often advise patients with DH to brush more gently using a soft toothbrush head. Sehmi and Olley, investigated the role of brushing force on dentine using manual toothbrushes (with soft tooth brush heads) and 1450ppm NaF toothpaste.³³ It was found that brushing at lighter brushing forces (100g) could create a smear layer after the equivalent of 4-6 weeks brushing. However heavier brushing forces (400g) brush away the smear layer and exposed patent dentine tubules after weeks of brushing.³³ This shows that the force of brushing in particular, over time, is important and can affect the presence or absence of the smear layer. **This occurs as more of the toothbrush filament comes into contact with the tooth surface at heavier brushing forces.** It is therefore important to advise patients to brush using lighter forces in order to prevent DH. Many electric toothbrushes have pressure indicator heads to help warn against overzealous brushing force.

Mullan *et al.* went on to investigate the effect of a desensitising toothpaste (designed to occlude the dentine tubules) at these various brushing forces on the dentine.³⁴ **The toothpaste occluded the dentine tubules at 100g and 400g, but at 400g there was also a significant increase in surface roughness. In contrast, brushing at low forces (100g) using a tubule occluding desensitising toothpaste results in tubule occlusion with minimal increases in surface roughness and is therefore recommended for patients with DH.**³⁴

Gingival recession, oral hygiene and DH

DH is often seen in patients with good oral hygiene and limited bleeding on probing, but gingival recession and this has been reported by Olley *et al.*³. Gingival recession can result in such patients with good, but otherwise overzealous tooth brushing, which will expose dentine to further abrasive wear and increase the risk of developing DH. Assuming all other diseases and conditions have been excluded for DH as per the definition of the condition⁴, DH might therefore be described, perhaps incorrectly, as a disease of the healthy mouth.

Other causes of attachment loss, which like tooth wear, is often described as a multifactorial condition³⁵, include periodontitis and its management, thin alveolar bone, trauma, orthodontic or prosthodontic treatment.³⁶ Following placement of indirect restorations, consequent gingival recession and DH has been observed in patients with good oral hygiene and regular recall of up to 50 years in dental practice³⁷.

Mechanism of pain in DH

The hydrodynamic theory is one of three suggested mechanisms of how pain occurs in DH; the other two include the neural and the odontoblast theories.³⁸ The hydrodynamic theory appears to be the most accepted. Gysi **first** suggested that dentinal pain occurred with fluid movement within the dentine³⁹ and Brännström later demonstrated that noxious stimuli such as cold, application of pressure and sugary liquids causes a change in pressure of the fluid within dentine.⁴⁰ This change in fluid pressure causes a triggering of nerve fibres within the pulp.

The neural theory in contrast, suggests that nerves located within dentine are directly triggered by noxious stimuli and this leads to activation of pulpal nerve fibres. However nerve endings in dentine do not extend to the enamel dentine junction.⁴¹ Another theory, the odontoblast transducer/receptor theory⁴², suggests that the observation of neurotransmitter substances within the whole length of the odontoblast process (which extends into dentine) allows the odontoblast to act as a transducer mechanism. Noxious stimuli are transmitted to synapses between odontoblasts and free nerve endings within the dentine. Interestingly, current research is focusing on the odontoblast in animal models. This has investigated how the odontoblast may be triggered in response to a noxious stimulus via mechanical fluid movement and, secondly how it may then interact with nerve endings. The term “odontoblast hydrodynamic receptor theory” has been used.⁴³

An interesting recent finding from animal models suggests that ‘chronic mild stress induced depression’ exacerbates the nociceptive response associated with DH.⁴⁴ Indeed, many would agree anecdotally that as well as impairing quality of life; depressive-like behaviour might worsen the severity of DH.

Diagnosis of DH in the lab and on clinic

Laboratory or better still *in-situ* experiments (in which the sample can be removed from the surrogate oral environment after exposure to an experimental variable and analysed in the laboratory⁴⁵), frequently rely on microscopic technology. Microscopes are used to visualise the number and size of dentine tubules at the surface of dentine samples, which is indirectly linked to DH via the hydrodynamic mechanism and the concepts of Lesion localisation and Lesion initiation. The importance of a validated method and measurement cannot be underestimated, to accurately investigate the aetiologies and assess the efficacy of desensitising products. Methods of measurement range from manually counting all visible patent dentine tubules in an image, validated visual ordinal scales or sensitive computer based systems⁴⁶. **The *in-vivo* measurement of patent dentine tubules might also be assessed using microscopic images taken of silicone impressions from the surface of sensitive dentine.**⁴⁷

For the purpose of a clinical trial **investigating DH, variations in the prevalence of DH between clinical pain studies are also due to the heterogeneity of studies looking at DH, for example how a study defines and diagnoses DH. Data could be collected from a clinician applying a noxious stimulus and recording the response or patient based questionnaires could be used. In these situations, differences may arise due to patient coping mechanisms¹⁵ or alternatively, as a result of the transient nature of DH¹⁵ as previously discussed.** It is advisable that two stimuli (**e.g. tactile or evaporative stimuli or repeats of the same stimuli**) are used to test if dentine is sensitive.⁴⁸ The two methods of assessing the response are stimulus and response based. The first can be assessed with electronic force probes such as the Yeaple force probes and Jay sensitivity probe, which vary the amount of tactile force applied to the tooth. Response based provides an indication of the pain as assessed by the patient, which is then measured as a visual analogue scale or verbal descriptor scale.⁴⁹ Taking this a step further, the Cumulative Hypersensitivity Score (CHI) score⁵⁰ has been validated for use as a standard measure of the severity of DH per patient (rather than per tooth). It **was developed from the earlier 'Schiff' score per tooth surface.** A blast of air from the 3 in 1 tip may be applied across all teeth quickly and the highest score recorded in each **intra-oral** sextant is added together. Therefore it avoids laborious measurement on individual tooth surfaces and provides a valid representation of the severity of sensitivity occurring on all **tooth** surfaces. The scores **per sextant** are as follows:

- 0 tooth/subject does not respond to air stimulus;
- 1 tooth/subject responds to air stimulus but does not request discontinuation of stimulus;
- 2 tooth/subject responds to air stimulus and requests discontinuation or moves from stimulus;
- **3 tooth/subject responds to air stimulus and has time to consider the stimulus. The pain is exaggerated and the patient requests discontinuation of the stimulus.**

The CHI score is similar to the scoring system used in the Basic Periodontal Examination and the Basic Erosive Wear Examination.¹⁰ **It provides another useful tool for the practitioner's armamentarium to show changes in DH severity over time and in relation to tooth wear.**

Management and the future ageing dentition

An important aspect of DH measurement and subsequent management focuses on the aetiology of DH, which as a surface phenomenon²⁹, as described above. Therefore, a large proportion of the management involves prevention (in particular reduction of erosive dietary intake and overzealous tooth brushing) and then treatments that will reduce dentine patency and protect against further tooth wear at the dentine surface. Recent studies have provided good evidence to support tooth paste formulations (that often act by tubule occlusion) using strontium acetate⁴⁵, calcium sodium phosphosilicate³⁴, arginine^{29,45}, stannous fluoride⁵¹ and nano-hydroxyapatite toothpastes⁵². The pastes were applied using a tooth brush mechanically and create tubule occlusion/smear plugs. Some of these have been shown as resistant to acid wear in varying degrees including strontium acetate⁵³, arginine²⁹, calcium sodium phosphosilicate³⁴. Various desensitising mouth rinses are also available and a systematic review shows there are significant reductions in patient self-reported DH when these products are used, as opposed to tactile or evaporative stimuli⁵⁴. There is little evidence to support professionally applied formulations in the management of DH.⁵¹ It is also important to note that the pathophysiology of bleaching sensitivity is different to classic DH described in this article and tubule occluding toothpaste formulations will prove ineffective.

Looking to the future, the UK population **might continue to** retain their teeth for longer, tooth wear is on the rise especially in the young and as in most of the developed world, the population is ageing.¹ Therefore, tooth wear and DH will be a problem in all age groups in the future. This will require better understanding of the condition, diagnostic skill and methods to prevent as well as manage the condition. The presence of DH on a tooth surface provides the only clinical indication of an active tooth wear and the management of DH and tooth wear go hand in hand. **Regular examinations and prevention of tooth wear and DH, as opposed to restorations, will therefore be important in future to help maintain the teeth into old age.**^{30,55} **Such an approach, using regular recall and strict preventive programs has been demonstrated in long term clinical studies conducted in practice, in order to conserve a functional dentition, in an ageing population.**¹⁷

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