WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Aetiology of Dentin Hypersensitivity: A Short Review

Gaurav Rawat [a], Harpreet Singh [b], Urvashi Saggar [c], Navneet Kaur [d*]

[a,c,d] Post Graduate Student, Conservative Dentistry and Endodontics, Baba Jaswant Singh Dental College, Hospital and Research Institute
[b] Professor and Head, Conservative Dentistry and Endodontics, Baba Jaswant Singh Dental College, Hospital and Research Institute
Email – hsgentledental@gmail.com

A B S T R A C T

Dentin hypersensitivity (DH) is a prevalent clinical issue characterized by pain and/or discomfort triggered by various stimuli, including thermal, tactile, osmotic, or chemical factors, which cannot be specifically attributed to any identifiable cause. Dentin hypersensitivity is an extreme form of dentinal sensitivity that results from localized pulpal inflammation, pulpal nerve sprouting, and the formation of inflammatory sodium channels, even though the terms are used interchangeably. It is characterized by short sharp pain emerging from exposed dentinal tubules in reaction to various stimuli. Dentin exposure may result from either enamel loss or cementum loss. After that, the smear layer is removed by either mechanical or chemical means. Currently, the most widely accepted explanation for dentin hypersensitivity is the hydrodynamic theory, which explains fluid movement in exposed dentinal tubules in response to stimuli. It affects the cervical aspect of the facial surfaces of teeth and is more prevalent in premolars and canines. Periodontal therapy and hygienic professional procedures may also induce mild to moderate discomfort that require the use of desensitizing products to reduce and remove symptoms. The present article outlines the aetiology of dentin hypersensitivity.

Keywords: Abfraction, abrasion, attrition, erosion, dentin hypersensitivity, recession.

Introduction

Dentin Hypersensitivity (DH) is defined as 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. [1,2]

According to American Association of Endodontists, DH is described as the short, exaggerated, sharp painful response elicited when exposed dentin is subjected to thermal, mechanical or chemical stimuli.

1. Aetiology of Dentin Hypersensitivity

DH is linked to various etiologies, including both non-surgical and post-surgical treatments for periodontal disease, as well as tooth wear and gingival recession. It is commonly noted that DH is linked to the exposure of dentin, particularly the opening of dentinal tubules, which in turn increases the sensitivity of dental pulp nerves to external stimuli. Dentin exposure can occur due to physical, chemical, pathological, or biological factors, as well as developmental abnormalities, all of which can lead to dental and/or periodontal damage or defects.[3] The common aetiologies are discussed below:

1.1 Postsurgical Periodontal Disease (Recession Induced by Management)

DH may occur iatrogenically, and Irwin et al. has reported that it affects up to 57 % of the general population following scaling or root planning.[4] Chabanski et al. reported a prevalence of DH of 85–95 % in periodontal patients. [5] In an in vitro study, it was reported that after root surface debridement, the dentin surface tubules were exposed following the removal of the surface covering or smear layer (in part). In a healthy condition, the smear layer re-forms, effectively blocking about 86% of fluid movement across the dentin. [6] The smear layer may be defined as a thin 'loose' layer consisting of organic collagen and glycosaminoglycans that form an adherent matrix over mineralized tissue arising from saliva and dentin particles that might occlude the dentinal tubules. [7,8] Moreover, it has been proposed that bacteria may colonize, dissolve the smear layer, and enter the root dentin in cases of periodontal disease or any treatment that may expose dentin [9] which in turn may initiate inflammation in the pulp.

1.2 Tooth Whitening Procedures

Tooth whitening procedures frequently utilize carbamide peroxide, a compound that decomposes into hydrogen peroxide and urea, effectively bleaching the teeth. This causes dehydration within the teeth and symptoms of DH. However, the symptoms are often temporary and for the duration of treatment.

[10] Bleaching may, however, involve a different mechanism to that of DH [11] and therefore is not strictly an aetiologic factor for DH and may therefore require alternative management strategies.

1.3 Developmental Disorders

These can often affect the primary and later permanent dentition and can cause symptoms DH. These include amelogenesis imperfecta, dentinogenesis imperfecta as well as other hypomineralized and hypoplastic lesions of enamel and/or dentine. These developmental lesions affects the growth and architecture of hard dental tissues(enamel or dentin) leads to exposure of dentinal tubules thus causing Dentin Hypersensitivity.

1.4 Dietary Factors (Erosive Diets)

DH is most commonly associated with erosive dietary factors (such as carbonated beverages , sports drinks, vinegar or candies). In a study by West et al. it was observed that there was a solid developing relationship between clinically elicited DH and erosive tooth wear caused by dietary factors. [12] In another study Bartlett et al. found 29 % of the subjects had signs of tooth wear (irrespective of any aetiology) which was recorded using the Basic Erosive Wear Examination. [13]

1.5. Gingival Recession

The various factors causing gingival recession are overzealous tooth brushing, improper brushing technique, or hard toothbrushes. On the other hand, lack of tooth brushing, with consequent accumulation of dental plaque on root surfaces in patients with inadequate oral hygiene, may lead to periodontal complications and migration of the gingiva in the apical direction. [14] Gingival recession leads to exposure of root surface which in turn leads to DH.

1.6. Tooth Wear

Tooth wear is the irreversible, non-traumatic loss of hard dental tissues due to aetiological processes classified as erosion, attrition, abrasion [15] and abfraction. [16. In the field of dentistry, Smith and Knight were the first to differentiate between physiological tooth wear and active or pathological tooth wear. Tooth wear can be considered pathological rather than physiological when it occurs in relatively younger patients and progresses at a rapid rate. In contrast, physiological tooth wear allows the pulp sufficient time to produce secondary or reparative dentin, which can reduce DH and help halt fluid flow within the dentinal tubules. In contrast, pathological tooth wear may cause DH as well as other aesthetic and functional concerns. [17]

1.7. Erosion and Dentin Hypersensitivity

Erosion is presently regarded as the predominant and significant causal factor contributing to tooth wear. There is an increasing body of literature indicating that acid erosion caused by relatively small acidic challenges will lead to loss of enamel and dentine and expose the dentinal tubules and initiate DH. [12] The superficial demineralization of hard tissue and the chemical dissolution of the apatite crystals in enamel by an acid that is not produced by the oral flora but from intrinsic or extrinsic sources. Intrinsic acid sources typically include regurgitated stomach acid, which contains hydrochloric acid (HCl), due to vomiting or gastroesophageal reflux causes erosive lesions as shown in Figure 1. Extrinsic sources of acid include:

- **Diet**: Acidic foods and beverages such as citrus fruits, carbonated drinks, sports drinks, beer, herbal teas, vinegars, pickles, and candies.
- **Medications**: Non-encapsulated HCl replacement, acetylsalicylic acid (aspirin) tablets , chewing vitamin C (ascorbic acid) tablets, iron supplements, and salivary stimulants.
- **Occupation**: Jobs that involve wine tasting or exposure to acidic industrial vapors.
- **Sports**: Activities such as swimming in improperly chlorinated pools.

These factors can contribute to tooth wear, which, in turn, may lead to DH.

Figure 1. Severe Erosion in cervical region

1.8. Abrasion and Dentin Hypersensitivity

Abrasion is a physical phenomenon that takes place due to the mechanical wear of dental tissues caused by external substances. Toothbrushing and toothpaste formulations are common forms of dental abrasion. [18] Toothpaste formulations of higher abrasivity or overzealous brushing or use of a toothpaste may initiate dentin wear. DH lesion initiation occurs due to removal of the smear layer and establishment of patent dentine tubules. [19] It has been proposed that, first, dentin exposure occurs as a result of enamel loss and/or soft tissue loss associated with gingival recession (including the loss of cementum). Second, once the dentin is exposed, the open dentinal tubules become accessible to the oral environment. This allows external stimuli (such as cold) to trigger small fluid movements within the dentinal tubules, which in turn activate the mechano-receptors in the dentin/pulp complex. [20] Buccal cervical abrasion on lower premolar can be seen in Figure 2.

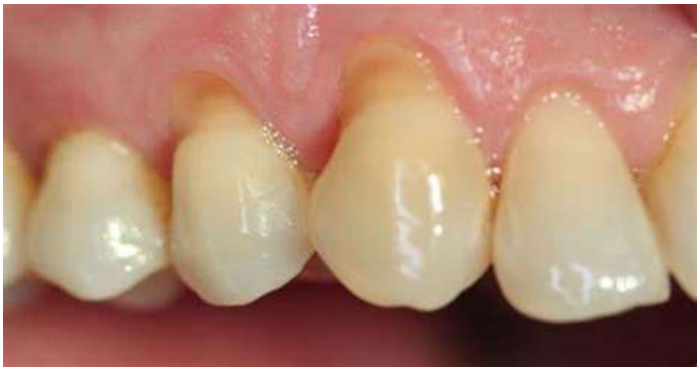

Figure 2. An illustration of a buccal cervical abrasion, often known as a non-carious cervical lesion (NCCL)

1.9. Attrition and Dentin Hypersensitivity

Attrition is the physical wear of hard dental tissues due to tooth-to-tooth contact on occlusal or incisal tooth surfaces. During normal function, the teeth make contact only for short period of time, primarily during eating or swallowing. However, when this contact occurs at other times, it is termed parafunction or bruxism can be seen in Figure 4. DH is more commonly observed on buccal tooth surfaces associated with gingival recession, however some studies have also reported DH involvement on occlusal surfaces. [21]

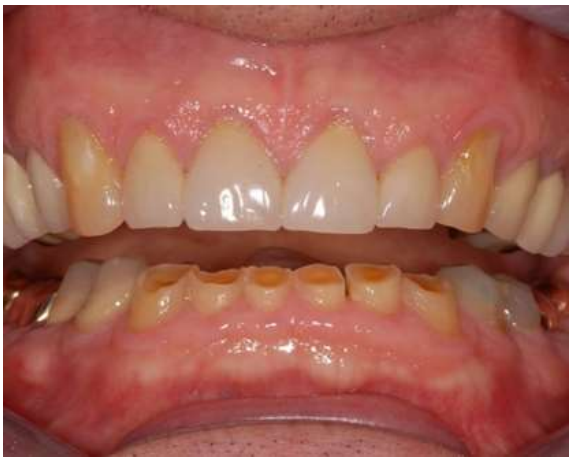

Figure 3. . An example of severe attrition associated with bruxism. Note that the incisal surfaces are flattened

1.10. Abfraction and Dentin Hypersensitivity

Abfraction was ascribed to those tooth wear lesions that could not be described due to erosion and/or abrasion, and which occurred due to occlusal stress often occurring near the cervical margin of teeth. Non-Carious Cervical Lesions (NCCLs) have been linked to the development of DH. There is limited data however to support the correlation between occlusal stress and NCCLs and the important aetiologies are therefore more likely to be erosion and abrasion. [22]

2. Conclusion

Dentin Hypersensitivity is a multifactorial disease and proper diagnosis of each and every factor is important to alleviate sensitivity response. Acknowledging the significance of etiology of DH is crucial for developing future treatment strategies that consider the underlying causes of this condition.

References

1. Holland GR, Nahri MN, Addy M, Gangarosa L, Orchardson R: Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. Journal of Clinical Periodontology. 1997 Nov; 24:808–13
2. Canadian Advisory Board on Dentine Hypersensitivity: Consensus-based recommendations for the diagnosis and management of dentine hypersensitivity. Journal of the Canadian Dental Association. 2003 Apr; 69:221–28
3. Liu XX, Tenenbaum HC, Wilder RS, Quock R, Hewlett ER, Ren YF: Pathogenesis, diagnosis and management of dentin hypersensitivity: an evidence-based overview for dental practitioners. BMC Oral Health. 2020 Aug 6;20(1):220.
4. Irwin CR, McCusker P: Prevalence of dentine hypersensitivity in a general dental population. The journal of the American Dental Association. 1997;43(1):7–9
5. Chabanski MB, Gillam DG, Bulman JS, Newman HN: Prevalence of cervical dentine sensitivity in a population of patients referred to a specialist Periodontology Department. Journal of Clinical Periodontology. 1996 Nov;23(11):989–92
6. Pashley DH, Livingston MJ, Reeder OW, Horner J: Effects of the degree of tubule occlusion on the permeability of human dentine in vitro. Archives of Oral Biology. 1978;23(12):1127–33
7. Brannstrom M: Sensitivity of dentine. Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology. 1966 April;21(4):517–26
8. Pashley DH: Smear layer: physiological considerations. Journal of Operative Dentistry. 1984; 3:13–29
9. Adriaens PA: Bacterial invasion in periodontitis, is it important in periodontal treatment? Revue Belge de Medecine Dentaire. 1989;44(2):9–30
10. Ferrari M, Cagidiaco MC, Monticelli F, Kugel G, Barker ML, Gerlach RW: Daytime use of a custom bleaching tray or whitening strips: initial and sustained color improvement. Am J Dent. 2007 Sep 20;A:19A–22A
11. Gillam DG: Current diagnosis of dentin hypersensitivity in the dental office: an overview. Clinical Oral Investigations. 2013 March;17(Suppl 1): S21-9
12. West NX, Sanz M, Lussi A, Bartlett D, Bouchard P, Bourgeois D: Prevalence of dentine hypersensitivity and study of associated factors: a European population-based cross-sectional. Journal of Dentistry. 2013 Oct;41(10):841-51
13. Bartlett DW, Lussi A, West NX, Bouchard P, Sanz M, Bourgeois D: Prevalence of tooth wear on buccal and lingual surfaces and possible risk factors in young European adults. Journal of Dentistry. 2013 Nov; 41(11):1007–13
14. Arua, SO, Fadare AS, Adamu, VE: The etiology and management of dentinal hypersensitivity. Orapuh Journal. 2021 Aug 11; 2(2) e815.
15. Ganss C, Klimek J, Lussi A: Accuracy and consistency of the visual diagnosis of exposed dentine on worn occlusal/incisal surfaces. Caries Research. 2006; 40(3):208–212.
16. Lee WC, Eakle WS: Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. Journal of Prosthetic Dentistry. 1984 Sep;52(3):374–80
17. Smith BG, Knight JK: A comparison of patterns of tooth wear with aetiological factors. British Dental Journal. 1984 Jul 7;157(1):16–19
18. Addy M, West NX: The role of toothpaste in the aetiology and treatment of dentine hypersensitivity. Monographs in Oral Science. 2013 Jun 28;23:75–87
19. Addy M, Hunter ML: Can toothbrushing damage your health? Effects on oral and dental tissues. International Journal of Dentistry. 2003;53(Suppl 3):177–-86
20. Gillam DG.: A New Perspective on Dentine Hypersensitivity – Guidelines for General Dental Practice. Dental Update. 2017 Jan;44(1):33-6, 39-42.
21. Olley RC, Wilson R, Moazzez R, Bartlett D: Validation of a Cumulative Hypersensitivity Index (CHI) for dentine hypersensitivity severity. Journal of Clinical Periodontology. 2013 Oct;40(10):942–47
22. Smith WA, Marchan S, Rafeek RN: The prevalence and severity of non-carious cervical lesions in a group of patients attending a university hospital in Trinidad. Journal of Oral Rehabilitation. 2008 Feb;35(2):128–34.