Review Article

Online ISSN: 2349-6940
DOI: 10.17354/cr/2017/282

Etiological and Predisposing Factors for Dentin Hypersensitivity: An Overview

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There are potentially numerous and varied etiological and predisposing factors to dentin hypersensitivity (DH). Certainly, no prime cause can be identified. By definition, DH may arise as a result of the loss of enamel and or root surface denudation with exposure of underlying dentin. Enamel loss as a part of tooth wear may result from attrition, abrasion or erosion followed by the constant action of acids, which keep the tubules open on the dentin surface, or because the root surface has been denuded due to loss of structures such as cementum, which is easily removed by brushing or periodontal treatment, or more commonly, by the association of two or more of these factors. This article aims to overview the various etiological and predisposing factors of DH.

Keywords: Dentin, Enamel, Hypersensitivity, Periodontal ligament, Tooth wear

INTRODUCTION

Tooth wear has usually been divided into attrition, erosion and abrasion, in reality, dentin hypersensitivity (DH) is due to a combination of these but often with differing proportional effects. The various factors involved either act as predisposing or as etiological factors. One problem of DH surveys is the definition of the condition and parameter of measure. The value of surveys comes into question when one wants to know the progress of DH since the data are a cumulative record. It is here where longitudinal studies play a key role in describing the disease process. Unfortunately, these studies are rare largely because wear occurs slowly.[1]

Gingival Recession

Gingival recession and subsequent root surface exposure allow more rapid and extensive exposure of dentinal tubules because the cementum layer overlying the root surface is thin and easily removed. Gingival recession, as with DH, has been described as an enigma, having what appears to be a multifactorial etiology. Tooth brushing has long been implicated in gingival recession on buccal surfaces and is a frequent finding in subjects with a high standard of oral hygiene, or with a history of hard toothbrush use. Furthermore, recession increases with increasing brushing frequency. Gingival recession is frequently cited to result from periodontal treatment particularly surgery as is DH.[1]

Access this article online

www.ijsscr.com

Month of Submission : 03-2017
Month of Peer Review : 04-2017
Month of Acceptance : 05-2017
Month of Publishing : 05-2017

Corrosion

Tooth surface loss caused by chemical and electrochemical action is termed "corrosion." There is both endogenous and exogenous source of corrosion. Endogenous source of corrosion is bulimia which produces a unique pattern of enamel loss. The corrosion called "perimolysis" is most marked on the palatal surface of maxillary anterior teeth and in most severe cases buccal surfaces of posterior teeth. The pattern is consistent with the head's position while vomiting. The forcefully directed movement of the vomitus, which has a mean pH of 3.8 determines the site and extent of dental corrosion. A special pattern of surface loss also is observed in patients with gastroesophageal reflux disease, or GERD. However, the movement of gastric acid juice in GERD as compared with that in bulimia is slower, less forced, more prolonged, more pervasive and more likely to intermingle the acid with food especially when the condition is "silent" and unknown to the patient. The enamel appears thin and translucent, enamel is lost on the posterior occlusal and anterior palatal surfaces; depression or concavities occur at the cervical areas of upper anterior teeth, "cupped" or invaginated areas develop where dentin has been exposed on the occlusal surfaces of posterior teeth because of wear. This dentinal cupping results from the joint digestive action of hydrochloric acid and the proteolysis enzyme pepsin that is contained in gastric juices. Atypical

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sites of corrosion may occur at locations where the gastric reflux fluid pools, especially while the patient is asleep. When the dentist finds evidence of gastric reflux referral to a gastroenterologist for evaluation and control is indicated.[2]

Gingival crevicular fluid has been shown to be acidic and may be corrosive when in contact with the teeth in the cervical region. Any food substance caused with a critical pH <5.5 can become a corrodent and demineralize teeth. Acidulated carbonate soft drinks have become a major component of many diets, particularly among adolescents and young children. The corrosive potential of an acidic drink does not depend exclusively on its pH value but also is strongly influenced by its buffering capacity, the chelating properties of the acid and by the frequency and duration of the acid.[2]

Alcohol abuse has been reported to cause a high incidence of corrosion, owing to the chronic regurgitation and vomiting that stems from the gastritis associated with the alcohol abuse. Occupational tooth corrosion can occur during exposure to industrial gases that contain hydrochloric or sulfuric acids as well as acids used in plating and galvanizing and in the manufacture of batteries and soft drinks.[3]

Attrition

The term attrition is derived from the Latin verb atrium which describes the action of rubbing against something. Dental attrition is defined as the physiologic wearing of teeth resulting from tooth to tooth contact as in mastication. This is an age-related process that can occur at the incisal or occlusal surfaces and sometimes on the proximal surfaces. Clinically, the first manifestation is the appearance of a small polished facet on a cusp tip or ridge or an incisal edge. Severe attrition may lead to dentinal exposure, which may increase the rate of wear. Tooth wear occurs at an ultrastructural level and can be caused by direct contact between surfaces or the action of an intervening. Attrition may be hastened by a coarse diet and abrasive dust. Silica particles to be abrasive in tobacco chewing and some para-functional habits (bruxism and clenching) may also contribute to attrition. The prevalence of dental attrition was not associated with the presence or absence of temporomandibular joint symptomatology. While a certain amount of attrition is physiologic, excessive destruction of tooth structure is not physiologic.[4]

Abrasion

Abrasion is defined as the wear of teeth caused by objects other than another tooth, examples include toothbrush/toothpaste abrasion and the variety of facets which can be caused by pipe smoking or other similar habits.

Traumatic brushing due to the poor position of vestibularized teeth makes them more subject to brushing trauma, or by excessive force or even lack of brushing, with consequent accumulation of dental plaque, causing gingival inflammation which may lead to periodontal complications and migration of the gingiva in the apical direction, exposing the cementum and then the root dentin.

In the 6000-year history of "oral hygiene products," today's toothpaste and toothbrushes are relatively recent introductions and date back to the early 20th century. Previous toothpaste/toothpowders could variably be described as revolting, highly abrasive, erosive, and even potentially toxic.[4] Modern toothbrushes and toothpaste formulations have in place, or in development, national and, more importantly, international standards, which primarily relate to potential safety issues. Most relevant here is the abrasivity of toothpaste.[5] By definition, toothpaste contains abrasive agents, the role of which is to remove stains and other superficial deposits from the tooth surface. Different formulations contain different abrasive agents, some being more abrasive than others. Relative dentin abrasivity (RDA) is a numeric scale, which indicates the degree of abrasivity, and is useful for comparison between different paste. A higher RDA value indicates a greater abrasive formula. The allowed pH range for toothpaste (pH 4-10) could be a cause for concern for tooth wear due to acid erosion, but virtually all products worldwide are above a pH which might cause demineralization (pH 5.5 for enamel, pH 6.5 for dentin), or the contained fluoride balances the low pH effect.

A study by Frandsen explains excessive zeal in performing oral hygiene procedures is also pointed out as being responsible for the appearance of pain. Tooth brushing with toothpaste has been stated as the most common oral hygiene habit practiced by persons living in developed countries.[6]

A study by Adam *et al*. explains typical toothbrush abrasion lesions are side dependent, for example being greater on the left-side in right-handed people. The buccal cervical areas of the teeth are the sites of predilection. Furthermore, canines and premolars are most affected because of their position within the dental arch where they receive the most attention during tooth cleaning. The toothbrush itself has little or no effects on dental hard tissues. Even toothpaste on a toothbrush alone causes almost no enamel abrasion and only clinically insignificant effects on dentin. However, when combined with erosive agents tissue loss from toothbrushing with toothpaste is increased enormously. Little is known about abrasion from chewing: The crushing of bones between the teeth and chewing tobacco were believed to lead to abrasion of teeth. It may also be caused by the gingival recession which occurs with aging, chronic periodontal disease and patients deleterious habits. The

dentin exposure may result in anatomic characteristics in the area of the enamel/cementum junction and/or enamel, or cementum loss due to one or more of the following processes.[7]

A study by Addy *et al.* explains toothbrushes alone produce essentially no wear of enamel. Toothbrushes alone over extended periods of use, measured in years, cause minute amounts of dentin wear, which may be restricted to the smear layer. The smear layer is an artificial surface structure that is formed when dentin is abraded or cut. The layer is about one micron thick and made up of collagen and hydroxyapatite from the native dentin. The smear layer covers the underlying dentin and occludes the tubules. Tooth brushing with toothpaste in the absence of acid causes little or no wear of enamel because, with the exception of the rarely used non-hydrated alumina, contained abrasives are softer than enamel.[8]

To conclude, toothbrushing with toothpaste will still have little or no effect on enamel, but abrasion of dentin can reach pathological proportions. It must be emphasized that these conclusions are at best drawn from studies *in vitro*, there are a few *in situ*, and at worst from the case or anecdotal reports. Unfortunately, studies *in vivo* that investigate toothpaste abrasion alone would be difficult, perhaps impossible, to design. The overall conclusion, therefore, must be that if toothpaste abrasion was the only wear process ongoing in the mouth, in normal use, it would have no clinical significance, except potentially to open dentinal tubules. Abrasion by toothpaste, however, is not the only wear process and teeth are exposed to wear by attrition and erosion. It is unlikely that attrition and toothbrushing with toothpaste would co-operate to cause tooth wear except where attrition has exposed dentin, which is subsequently brushed. Abrasion, on the other hand, does have the potential to enhance tooth wear due to acid erosion.

Erosion

The term erosion is derived from the Latin verb erosum (to corrode) which describes the process of gradual destruction of a surface, usually by a chemical or electrolytic process. Dental erosion is defined as the loss of tooth structure by a nonbacterial chemical process. Erosion may be by either extrinsic or intrinsic acids. Extrinsic erosion can be subdivided into dietary and environmental, while intrinsic erosion is the result of exposure of teeth to gastric juice. Dietary erosion may result from foods or drinks containing acids such as citrus fruits, pickled food, fruit juices, carbonated drinks, wines, and ciders. A recent publication showed that a raw food diet bears an increased risk of dental erosion compared to conventional nutrition. Beverages such as red and white wine, citrus fruit juices, apple juice, and yoghurt were capable of rapidly dissolving the dentin smear layer within a few minutes. Perhaps surprisingly, a carbonated cola drink was considerably less erosive.[4]

Vitamin C (ascorbic acid), which is considered a healthy additive in many drinks, has been implicated in dental erosion. In addition citric acid, found in many drinks, has the potential to both demineralized dental hard tissues and chelate calcium. Industrial erosion results from occupational exposure to acids or acidic vapor, such as workers in battery manufacture and wine tasters. Other extrinsic sources of erosion have been reported, including swimmers who trained in poorly maintained pools with water at pH 2.7. Improper use of bleaching agents particularly delivered in night guards is another risk factor for the erosion of enamel and dentin and has been implicated in the development of DH. Some mouth rinses have low pH values and have the potential to dissolve the smear layer and thereby expose dentinal tubules; an effect enhanced by post-treatment brushing. Intrinsic erosion may result from gastric reflux as in patients with hiatus hernia, chronic alcoholism and eating disorders. When erosion is caused by gastric regurgitation, the palatal aspects of the upper incisors and the occlusal and buccal aspects of lower posterior teeth are primarily affected.[4]

There are two types of erosive lesions found using the scanning electron microscope. The active lesion shows distinctly etched enamel prisms resembling a honeycomb, while latent or inactive erosions are faint with unrecognizable characteristics. Further ultrastructural studies have demonstrated irregular patterns of enamel dissolution. As the lesion progresses to dentin, the first area to be affected is peritubular dentin. Dentinal tubules then become enlarged, affecting intertubular areas as well. Rapid processes may lead to sensitive teeth, while slower progression may be asymptomatic.[4]

The solubility of enamel is pH dependent, and the rat eat which apatite precipitates depends on certain factors, such as calcium binding in saliva. Saliva contains calcium and phosphate ions and exists in a supersaturated state at neutral pH with respect to enamel hydroxyapatite. As the pH of saliva decreases, it crosses the saturation line at a point known as the critical pH. Since the critical pH of enamel is approximately 5.5, any solution with a lower pH may cause erosion, particularly if the attack is lengthy and intermittent overtime.[4]

In advanced cases, restorations may project above occlusal surfaces and cusps of posterior teeth (and incisal edges of anterior teeth) exhibiting concavities known as "cupping." Erosion associated with diet may be evident on labial surfaces of maxillary anterior teeth and present as' scooped-out "depressions."[9]

Abfraction

The cervical stress lesions have been hypothesized as an etiological factor in tooth wear. The process is thought to involve eccentric occlusal loading leading to cusp flexure. This in turn leads to compressive and tensile stresses at the cervical fulcrum area of the tooth with the resultant weakening of the cervical tooth structure. The process may be co-destructive rather than directly causal whereby abrasion and or erosive processes are potentiated. It is difficult to diagnose such lesions properly, but generally, in cases where a deep V-shaped cervical notch is present or when cervical restorations are repeatedly lost, the practitioner should look for wear facets or other signs of occlusal trauma.[4]

In 1991, Grippo coined the term "abfraction" as a new classification of cervical lesions caused by biomechanical loading forces, to distinguish it from erosion and abrasion. During eccentric loading, flexing stresses throughout the tooth produce tension on one side and compression on the other in the area of the fulcra, generally located at or near the cementoenamel junction. Non-carious cervical lesions are more commonly associated with the loss of enamel; exposure of dentin occurs much less frequently, ranging between 2% and 6%.[10]

Combined Mechanism of Tooth Wear

Attrition-abfraction

Attrition-abfraction is the joint action of stress and friction when teeth are in tooth to tooth contact, as in bruxism or repetitive clenching.[11]

Abrasion-abfraction

Abrasion-abfraction is the loss of tooth substance caused by friction from an external material on an area in which stress concentration due to loading forces may cause tooth substance to break away. Such a synergistic tooth destructive effect may be observed cervically when toothbrushing abrasion exacerbates abfraction to produce wedge-shaped lesions. The critical roles of both toothbrushing abrasion and occlusal loading of an anatomically vulnerable zone may be one reason why such lesions are limited almost exclusively to the buccal and labial cervical areas of teeth.[11]

Corrosion-abfraction

Corrosion-abfraction is the loss of tooth substance due to the synergistic action of a chemical corrodent on areas of stress concentration. This physiochemical mechanism may occur as a result of either sustained or cyclic loading or leads to static stress corrosion or cyclic stress corrosion.[11]

Static stress corrosion

Static stress corrosion is the loss of tooth structure due to the action of a corrodent on an area of sustained stress. This may occur during clenching. Static stress corrosion may be observed as demineralization that occurs around orthodontic appliances in the presence of corrodent.[11]

Cyclic (fatigue) stress corrosion

Cyclic stress corrosion is the loss of tooth substance due to the action of corrodent in an area of concentrated stress during cyclic loading. This combination of mechanism could occur during mastication, as seen among patients who engage in fruit mulling as dentinal invaginations, but is seen most strikingly among patients who brux in the presence of endogenous (for example, GERD) or exogenous(carbonated soft drinks) corrodents. In such situations, tooth substance may be lost rapidly and extensively.[11]

Attrition-corrosion

Attrition-corrosion is the loss of tooth substance due to the action of a corrodent in areas in which tooth to tooth wear occurs. This process may lead to a loss of vertical dimensions, especially in patients with GERD or gastric irritation.[12]

Abrasion-corrosion

Abrasion-corrosion is the synergistic activity of corrosion and friction from an external material. This could occur from the frictional effects of a toothbrush on the superficially softened surface of a tooth that has been demineralized by a corrosive agent. Teeth that are out of occlusion could be affected by this mechanism and develop cervical lesions, since they frequently extrude, thus exposing the vulnerable dentin. Similarly, gingival recession may expose the cementum and dentin to this oncolytic process.[11]

Low Level of Oral Hygiene

Patients with a low level of oral hygiene have a high degree of periodontal tissue destruction, loss of supporting bone tissue and root exposure. Root exposure is related to DH, and it can be aggravated by the action of acids secreted by bacteria capable of opening the dentinal tubules even further.[11]

Periodontal Therapy

Periodontal therapy has been associated with DH due to the exposure of dentinal tubules after the removal of supra and/or subgingival calculi. Another factor is the removal of dental cementum which covers the root or the root dentin itself during periodontal scraping. Scaling and root planning removes the outer layer of hypermineralized dentin and thus leaves the surface expose to the effect of hydrodynamic phenomena. Surgical periodontal treatment, similarly, usually involves complete debridement of the root surface. Post-operative recession of soft tissue further exposes the dentinal tubules. Patient inability to maintain plaque control in the healing phase further complicates

the problem, as plaque and acid production due to plaque accumulation could act as a noxious stimuli and cause dehydration and lead to fluid movement across the dentinal tubule. Instrumentation of the root creates an outer contiguous smear layer covering the instrumented surface as well as pushing debris into the dentin tubules for several micrometers. The smear layer thus is a natural iatrogenic yet transient treatment to DH. Removal of the outer smear layer and smear plugs with acids permits an increased in outward fluid flow and thus increase the patient post-operative DH.[12]

Physiological Causes

The increase in the number of teeth with root exposure is evident, as age advances. Dental extrusion, in the absence of an antagonist tooth, results in root exposure, which may lead to DH. It mostly affects individuals at the end of their third decade of life, causing patients great discomfort. In some cases, it may lead to emotional alterations and behavior changes.[13]

Role of Dental Plaque

Plaque control plays a key role in reducing the patency of dentinal tubules and may therefore actually promote the natural repair of DH. It is this removal of plaque, by either mechanical (tooth brushing) or chemical means, that has been found to reduce the diameter of dentinal tubules and therefore helps to alleviate DH. Dental professionals must therefore promote this message of maintaining good oral hygiene to patients with DH. Plaque accumulation on root surfaces can lead to demineralization of the root surface, which in turn leads to the opening of the dentinal tubules, and therefore to pain. Patients with poor plaque control on the root surfaces report more problems with DH.[13]

Bleaching Sensitivity

Another chemical factor that can trigger tooth sensitivity similar to dental sensitivity is the effect of bleaching agents, which cause bleaching sensitivity. Sensitivity often occurs during both in-office and at-home tooth whitening treatments, and has been considered to be the most common complication of at-home tray treatments. The problem will commonly manifest itself as generalized hypersensitivity to cold stimuli, but often also occurs as a spontaneous sharp pain, sometimes limited to one or a few teeth. The cause of the sensitivity experienced during whitening treatments is considered to be multifactorial - involving acidic pH, dentinal fluid outflow caused by osmotic stimuli and penetration of peroxides through enamel and dentin. The latter supposedly results in a reversible pulpal irritation. Pain needs to be prevented and treated to avoid negative effects on treatment compliance. Pre-existing DH is considered one of the best predictors of post-whitening hypersensitivity, and the use of classic desensitizing agents applied topically, has proven effective in managing the pain. Patients undergoing tooth-whitening procedures can be offered an in-office DH treatment, as well as preventive advice and instruction on the daily use of home DH products.[13]

Restorative Sensitivity

Restorative sensitivity is triggered following placement of a restoration for several possible reasons: Certain amalgams having a history of 24-48 h sensitivity due to shrinkage, rather than the usual expansion, during setting; contamination of composites during placement or improper etching of the tooth on composites, which results in microleakage; improper tooth-drying technique; incorrect preparation of glass ionomer or zinc phosphate cements; general pulpal insult from cavity preparation technique; thermal or occlusal causes; galvanic reaction to dissimilar metals that creates a sudden shock or "tin foil" taste in the mouth.[12]

Medication Sensitivity

Medication sensitivity is caused due to medications that dry the mouth (e.g. antihistamines, high blood pressure medication), thereby compromising the protective effects of saliva and aggravating diet-related trauma or proliferating plaque. Even a reduction in salivary flow due to ageing or medications can lower the pH of the saliva below the level at which carries occurs (6.0-6.8 for dentin caries; <5.5 for enamel caries) and increase erosive lesions to exposed dentin.[12]

SUMMARY

DH is a common problem among many dental patients. The patient plays a role in this process since their daily habits could be one the etiological or predisposing factors which is a part of levels of prevention. The initial etiology of DH, in the majority of cases is gingival recession with the exposure of dentinal tubules. Once the tubules are exposed the patient is susceptible to pain in response to thermal, tactile, or osmotic stimuli. Desensitizing treatments should be delivered systematically. Prevention of DH could be the initial approach which can be further followed by professional treatments.

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How to cite this article: Bhowmick GS. Etiological and Predisposing Factors for Dentin Hypersensitivity: An Overview. IJSS Case Reports & Reviews 2017;3(12):16-21.

Source of Support: Nil, **Conflict of Interest:** None declared.