Tooth Wear – Biology and Recent Management Strategies

Alsolaihim Abdulrahman Nasser[1]*, Alsolaihim Aljood Abdulrahman[2], Alowais Layla Omar[3]

1. Consultant in operative dentistry, King Abdulaziz Dental center, National guard health affairs, Riyadh.
2. General Dentist. PO box 260527, Riyadh 11342.
3. Dental Hygienist, Alyamamah Hospital, Ministry of Health, Riyadh.

Abstract

Tooth wear is group of non-carious destructive lesions of teeth wherein wear mechanism have numerous interactions and almost never have a single etiology. Although the severity of wear is higher in older individuals, even younger population has demonstrated a high incidence of wear. Wear mechanism have been traditionally classified as Attrition, Abrasion and Erosion.

The most prevalent technique to clinically quantify wear of teeth is to use the different tooth wear indices. Raising the patient's awareness of the causative factors of the tooth wear and minimising further damage are essential. The ideal management approach incorporates a treatment plan incorporating an accurate diagnosis of the tooth wear etiology, education and promotion of lifestyle modifications to help prevent further wear, restoration or rehabilitation to rebuild the worn dentition if required and monitoring and periodic review.

This paper attempts to elaborate the various mechanisms of tooth wear and their management strategies.

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Introduction

Regressive alterations of teeth are fundamentally wearing away of teeth occurring as a multifactorial process developing during aging. Wear is the term that represents gradual material removal from a surface due to a mechanical movement and/or a chemical process.[1] It is group of non-carious destructive lesions of teeth wherein wear mechanism have numerous interactions and almost never have a single etiology. Although the severity of wear is higher in older individuals, even younger population has demonstrated a high incidence of wear.[2] Tooth wear is usually classified into attrition, abrasion (mechanical wear)and erosion (chemical wear). Teeth undergoing wear exhibit both microscopic and macroscopic changes in the enamel and dentin – pulp complex. With the geriatric population increasing and along with the higher percentage of dentate individuals, the phenomenon of tooth wear gains additional significance. Traditionally, more research has been directed towards carious destruction of tooth substance and tooth wear as received less scrutiny. Therefore, it becomes crucial that sufficient prominence and thorough analysis is given to the phenomenon of tooth wear.

Corresponding author:
Dr. Abdulrahman N Alsolaihim
King Abdulaziz Dental center, King Abdulaziz medical city,
Ministry of National Guard Health Affairs,
Mail Code – 1243, PO box – 22490
Riyadh -11426. Kingdom of Saudi Arabia
E-mail: Soulaihim@hotmail.com

Microscopic Changes Due To Wear

Enamel

The microstructure of enamel is heterogenous. This aspect is due to the masticatory forces causing multi axial and non-uniform stresses on enamel. Unlike other human tissues, Enamel does not remodel or regenerate when it undergoes wear.[3]

a) Changes in Permeability

Enamel acts as semipermeable membrane and permits slow passage of water and molecular substances through pores between crystals. As age increases the crystals grow in size, the pores become obliterated resulting in reduced permeability of enamel.

b) Changes in Colour

The nitrogen content of enamel increases with age. This has been correlated with the increase in the amount and intensity of pigment,

which causes a darkening of teeth in older age. Changes in the refraction and absorption of light appear to be related to structural changes in the enamel and dentin.

(c) Formation of Cracks

Microcracks occur during mastication of abrasive substances. During the rubbing of these microparticles, friction occurs leading to formation of microcracks. This is a cumulative process of plasticity-induced microcrack generation in the surface and subsurface regions.[4]

Dentin

Dentin , unlike enamel, consists of approximately 70% inorganic matter and 30% organic matter and water. Dentin is a more ductile and plastic structure due to its increased organic content.

A) Dentinal sclerosis

The mineralisation and hardness of dentin increase while tubular diameter decreases with age. This is most commonly seen in apical third of the root that slowly progresses towards the cervical regions. This phenomenon may not be always associated with any external traumatic influences.[5]

B) Changes in secondary dentin

Secondary dentin comprises the circumpulpal portion in continuity with the primary dentin. The size of the dental pulp cavity is reduced with age, because of continuous secondary dentin deposition. In old age the secondary dentin deposition becomes irregular and an increasing loss of dentinal tubules occurs. Active and rapid primary dentin formation changes to slow secondary dentin formation due to factors related to aging rather than related to tooth function.

C)Formation of Reparative Dentin

Tertiary dentin is the irregular dentin that is formed in response to abnormal stimuli, such as excess tooth wear and other factors. Deposition of tertiary dentine leads to obliteration of the pulp cavity. In older persons tertiary dentine is laid down in response to chronic stimuli.

Changes in Pulp

A) Decrease in Cellular Components - Fibroblasts and odontoblasts undergo degeneration. More number of vacuoles are seen and there is also decrease in number of regenerable cells. As the age increases there is increase in the amount of collagen fibres in the pulp due to decrease in the number of cells. So, this prominence of fibres bundles in pulp addition to decrease in ratio of ground substance to collagen, causes atrophy of pulp.[6]

B) Dystrophic mineralization

Dystrophic mineralization is found in varying amounts and degrees in most of pulps. Ground substance alteration occurs in the dental pulp and results in decreased reactivity of less soluble macromolecules. Such changes can lead to dystrophic mineralization.

Macroscopic Changes on Teeth

Attrition

Attrition is the condition resulting mainly from tooth to tooth contact without any third substance in between the two substrates. Attrition is sourced from Latin verb “atterere”, which is defined as the action of a material chafing or scrubbing. Attrition affects the occluding surfaces and results in flattening of the cusps and in depressed facet formation.

Clinical Signs and Symptoms:

Blunting (flattening) of cusp causes increased masticatory force generation in order to grind the bolus into smaller ingestable size. These forces can cause hypertrophy of the masticatory muscles and increase the bite force of the teeth thereby leading to more attrition.[7]

Decay as a result of attrition of enamel can occur because the underlying dentin will be exposed and thereby become more susceptible to decay. However, this susceptibility is decreased, to some extent, by higher clensability as the grooves and fissures are flattened out.

Tooth sensitivity is a symptom that can be due to many factors precipitated by attrition process e.g., dentin exposure, pulpal reaction due to abnormal masticatory stresses, and injury to periodontal ligaments, microcracks, and pooling of food on the depreesed facet on the occlusal surface.

Abrasion

The term abrasion is derived from the Latin verb abrader (to scrape off). It is the pathological wear process of the teeth involving a third external substrate. Depending on the etiology, the patterns may vary from localized to diffuse. Most common variant form of abrasion is tooth brush abrasion. It occurs in the gingival aspect in the buccal surfaces of teeth. It is on left side of arch for right handed persons and vice versa. Other habits like chewing tobacco, forcing tooth pick can cause proximal abrasion.[8]

Clinical Signs and Symptoms:

The Surface of lesion smooth and polished seldom has any plaque /calculus and the Surrounding walls tend to make a v shape by meeting acute angle axially. Also, peripheries of lesion are angularly demarcated from adjacent tooth surface.[9]

Erosion

Dental erosion results when there is dissolution of enamel and dentin because of the effect of acid and/or chelation with no involvement of bacterial activity.

On the surface of enamel, the acid with its hydrogen ion (or a chelating agent) will start to dissolve the enamel crystal. First, the prism sheath area and then the prism core is dissolved, leaving the well-known honeycomb appearance.[8] Fresh, unionized acid will then eventually diffuse into the interprismatic areas of enamel and dissolve further mineral underneath the surface, in the subsurface region. [10,11]This will lead to an outflow of ions and subsequently to a local pH rise in the tooth substance and in the liquid surface layer in close proximity to the enamel surface.

In dentine, due to the high content of organic material, the diffusion of the demineralizing agent (i.e. acid) deeper into the region and the outward flux of ions and minerals are hindered by the organic dentine matrix. Thus, the organic dentine matrix has a sufficient buffering capacity to retard further demineralization.[12]

Abfraction

Abfraction was coined from Latin word “ab” away and “fraction” meaning breaking. It causes chronic loss of tooth structure due to masticatory forces resulting in wedge shaped defects in cervical areas.

This is due to breaking away of Enamel rods, cementum and dentin. Stress that concentrate to produce abfraction are transmitted by occlusal loading forces.[13] Abfraction lesion is located at in region of bending or pivoting area of the crown where stress is at its greatest concentration. It is a typically wedge shaped and displays a size proportional to the magnitude and frequency of tensile force application.

However, the theory of Abfraction is not yet proven. Overall, there is a weak association between Non-carious cervical lesions (NCCLs) and occlusal factors.[14]

Combined mechanisms of tooth wear

Although some of the aforementioned individual tooth wear systems may act singly, concurrent phenomenon of multiple wear forces acting together occur more often than single wear type.[15]

Attrition-Erosion.

Attrition-Erosion is the loss of tooth substance occurring due to the joint effect due to the action of erosive acids in areas in which tooth-to-tooth wear occurs. This phenomenon may commonly to a loss of vertical dimension, especially in patients with GERD or gastric regurgitation

Abrasion-Erosion.

Abrasion-Erosion is the synergistic activity of erosion and rubbing activity due to a third foreign substance. This could occur from the frictional effects of a toothbrush on the pliable exterior of a tooth that has been previously demineralized by an acidic substance. In supraerupted teeth, these cervical lesions are more common, as the exposed dentin is more at risk. Similarly, gingival recession may expose the vulnerable cementum and dentin leading to higher tooth loss.[16]

Tribology of wear

Tribology (from the Greek tribe in, meaning to rub) is the study of friction, wear and lubrication. Tooth wear is a term commonly used to describe the triad of attrition (proximal and occlusal inter-dental friction), abrasion (friction with the intervention of particles) and erosion (chemical dissolution).[17]

Abrasive wear

Abrasive wear is the most common type of wear. Depending on the number of materials in contact, tribology distinguishes two types of abrasive wear: two-body abrasion and three-body abrasion.

Two-body abrasion

This type of abrasion is the friction between two solid bodies in movement where the surfaces are in direct contact.

Three-body abrasion

Three-body abrasion is the displacement of two bodies, one across the other, with the interposition of abrasive particles that constitute the third body.[18,19]

Fatigue Wear:

When one surface under high pressure slides over another, a compression zone is created ahead of the movement whilst a tension

zone is created behind. In the oral cavity, fatigue wear can occur on certain enamel occlusal contact surfaces subjected to considerable pressure, besides during mastication.

Erosive wear

Erosion is caused by chemical dissolution of the tooth-mineralized tissue.[20] Tribochemical wear is caused when chemicals (acid, chelating agent) weaken the inter-molecular bonds of the surface and therefore potentiate the other mechanical wear process.[21] This situation can be particularly critical in cases of sleep bruxism where two-body abrasion following episodes of teeth clenching and/or grinding is often accompanied by intrabuccal acidity linked with gastroesophageal reflux disease (GERD).[22] Three-body abrasion linked with iatrogenic tooth brushing can also significantly aggravate occlusal and cervical lesions in an acid environment as surface molecules are driven away, only for the newly exposed surface to be immediately attacked by the erosive environment.

Measurement of Tooth Wear

The most prevalent technique to clinically quantify wear of teeth is to use the different tooth wear indices. Other methods measuring tooth wear are surface profilometry, polarized light microscopy, microhardness, microradiography, digital image analysis and scanning probe microscope.

Among the various indices, the commonly used index is the Smith and Knight Index.[23]The other indices commonly employed are Basic Erosive wear Examination,[24]

Exact Tooth Wear Index,[25] Simplified tooth wear index and many more.[26]

Larsen et al.[27] recommended a new clinical index based on a combination of clinical examination, photographs and study casts, with complicated qualitative and quantitative criteria There are too many indices proposed and used, with lack of standardization in terminology. Also, attempts to quantify the amount of tooth tissue loss due to tooth wear, have historically concentrated only on one aetiology, and these indices tend to be surface limited.

Preventive and Management Strategies of Tooth Wear

Lifestyle risk factors are most important in the majority of patients, and preventive focus must consider these risks.[28] Educational campaigns to increase awareness of the causes of tooth wear may be possible. Teenagers and young adults may be counseled to reduce the consumption of erosive soft drinks.

Frequent consumption of acidic foods and beverages

- Sports- or work-related dehydration reducing salivary protection
- Acidic beverage rehydration at times of dehydration
- Health Medications affecting salivary protection
- Work-related exposure to acids or abrasive
  - GERD
  - Alcoholism causing vomiting / regurgitation
  - Bulimia and anorexia
  - Chewable vit c supplements
  - Biting on pencils, sewing pins, pipe stem, seeds and nuts.
- Or any combination of the above[29]

The WATCH strategy

The WATCH strategy is a preventive approach encouraging a lifestyle that is healthy, irrespective of whether patients present with significant tooth wear or not. Individualised, patient-empowering erosion WATCH strategies; on Water, Acid, Taste, Calcium and Health in promoting healthy diet and lifestyle is promoted.[30]

Treatment Planning

The ideal management approach incorporates a treatment plan which includes the following:[31]

- Accurately diagnose the tooth wear etiology
- To educate and promote lifestyle modifications to help prevent further wear
- Restoration or rehabilitation to rebuild the worn dentition if required
- Monitoring and periodic review.

In order to manage the hypersensitivity arising due to tooth wear, adjuncts like topical fluoride gels and rinses,[32] artificial saliva solutions,[33] CPP-ACP[34] as mousse and chewing gum,[35] and a large variety of medicated toothpastes can also be prescribed.

Tooth surface loss (TSL) occurring due to wear may lead to a lack of axial height and thus insufficient retention and resistance for conventional extra-coronal restorations.[36, 37]

Dahl et al[38] proposed that relative axial tooth movement that is observed when a localised appliance or localised restorations are placed in supra-occlusion and will lead to

occlusion re-establishment of full arch contacts over a period of time. The occlusion tends to re-establish after about six months on average but it can take up to a period of 18-24 months.[39]

Wood et al. (2008)[40] concluded that the value in restoring noncarious cervical lesions is unclear and occlusal adjustments to prevent lesion progression or improve retention of restorations cannot be supported. Sakoolnamarka and Tyas and Burrow (2007)[41] considered bond strengths of various dentine-bonding agents and restoration of non-carious cervical lesions, as well as possible reasons for restorative failure of bonded restorations attributable to the differences compared to regions of normal dentine. They concluded that significant doubt exists as to the importance of the involvement of abfraction in restorative failure and instead considered non-carious cervical lesion surfaces to have a lower phosphate-to-silicon ratio compared with normal dentine and the dentine surfaces were more highly mineralised with greater proportions of obliterated dentinal tubules.

Raising the patient's awareness of the causative factors of the tooth wear and minimising further damage are essential in this pre-restorative phase of treatment.[42,43] Changing the balance from demineralisation to remineralisation not only prevents further tooth structure loss but may reduce sensitivity caused by an active erosion process.[44] This remineralisation is also critical in establishing a stable substrate for adhesive restorations. In the absence of a remineralisation potential, mineral from tooth structure is lost and the resultant demineralised surface is weak, unsupported and prone to breakdown. This hypo mineralised surface not only leads to adhesive failures with the restorative material, but it is also possible to obtain cohesive failures within the tooth surface related to weakness in the demineralised surface. To minimise the risk of failure, it is therefore necessary, prior to any restorative treatment, for the tooth surface to be remineralised and strengthened to optimise interface adhesion.[45]

Conclusions

In recent times, the diagnosis of tooth wear has increased manifold. Accurate diagnosis and a thorough understanding of the mechanisms of tooth wear will enhance the standard of care for such patients. Clinical measurement of the severity of wear must be undertaken. Long term monitoring and periodic motivation of the patients play an important role in the successful management of tooth wear.

Declaration of Interest

The authors report no conflict of interest.

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