

Distinguishing and diagnosing contemporary and conventional features of dental erosion

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The vast number and variety of erosion lesions encountered today require reconsideration of the traditional definition. Dental erosion associated with modern dietary habits can exhibit unique features that symbolize a departure from the decades-old conventional image known as *tooth surface loss*. The extent and diversity of contemporary erosion lesions often cause conflicting diagnoses. Specific examples of these features are presented in this article. The etiologies, genesis, course of development, and characteristics of these erosion lesions are discussed. Contemporary and conventional

erosion lesions are distinguished from similar defects, such as mechanically induced wear, carious lesions, and dental fluorosis, which affect the human dentition.

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The increased incidence of dental erosion associated with dietary sources has become a growing universal concern for dental health care providers.¹⁻⁷ The severity of this widespread issue has gained the attention of clinicians, researchers, the media, and the public in both the United States and worldwide.¹⁻⁷ Published literature has indicated that acidic constituents of dietary ingredients—especially acidulated carbonated beverages, considered to be the single most prominent etiology—have considerable impact on the development of dental erosion.⁷⁻¹⁴ The excessive consumption of these beverages among younger generations renders these consumers extremely vulnerable to this dental hard tissue disease.⁵⁻¹⁵ Likewise, excessive consumption of highly acidic citrus fruit and juices increases the potential for dental erosion in a large segment of the senior population.¹⁶⁻¹⁹

An overwhelming variety of dental erosion lesions are encountered by clinicians.^{16,17,20-22} The clinical features of these contemporary lesions transcend the definition of conventional tooth surface loss (TSL) that has been known for more than half a century.^{8,14,16,23} The range of these lesions is associated with numerous sources of acidic dental erosion, the most predominant of which are nutritional sources.^{8,14,16,17,23} Each type of erosion lesion possesses specific clinical signs and a unique appearance. These signs differ widely from each other, but their spectrum defines the general characteristics of contemporary dental erosion.

Recognizing the unique characteristics of each, and identifying their respective etiologies, could facilitate their differentiation from similar lesions of nonerosion origins. Examples of these are noncarious defects, carious lesions at various stages of development, and acquired anomalies such as dental fluorosis and tetracycline-stained hard tissues.²⁴⁻²⁷

Distinguishing and decoding contemporary erosion lesions is fundamental for achieving an accurate diagnosis and planning a proper course of management. The most clinically satisfactory, economical, and practical approach is prevention. However, proper identification of these lesions may present a clinical challenge. This is primarily due to an inadequate awareness of the specific characteristics of each type of lesion. It can also be due to the shortage of relevant information from patients' health and dietary histories, as well as their oral hygiene care. These factors collectively could negatively impact the diagnostic process and result in an impulsive and potentially incorrect conclusion. Arming dental professionals with the ability to recognize the wide spectrum of these newly emerged erosion lesions, and familiarizing them with the specific features of each, could expedite identification and alleviate confusion. The objectives of this article are to identify and precisely describe the assortment of contemporary erosion lesions observed today, and clearly differentiate between their clinical features and those of lesions with closely similar characteristics.

The commonly known definition and the classical description of conventional erosion lesions allude to TSL of enamel with or without dentin involvement. This enamel/dentin defect is presented clinically as a smooth surface with either partial or complete loss of topographic and anatomic configurations. The TSL could appear in the form of a flat or saucer-shaped defect that is usually limited to enamel, but may also involve dentin in the affected region.^{23,28,29} Progression of the enamel defect leads to removal of the entire enamel thickness, exposure of dentin, and further invasion into the dentin core.^{14,23,28-30} These defects are often seen on the cervical to middle segments of the tooth crown, although their vertical expansion may lead to either crown and/or root surface involvement. These erosions are commonly seen on the facial aspects of the dentition and should be differentiated from defects due to toothbrush abrasion and abfraction (Fig. 1). Their presence on the lingual aspect of a tooth is etiology-specific. In severe cases, they may also affect the biting surfaces of teeth.

Examples of erosion lesions that present features of TSL on the lingual aspects of dentition are those that develop as a result of either extrinsic or intrinsic sources. The former could be caused by excessive sipping of lemonade; sucking slices of lemon, orange, or grapefruit; or soaking the dentition in pooled soda for a while before swallowing.^{8,14,16} Erosion lesions from intrinsic sources that appear on the lingual aspects of the anterior maxillary incisors and canines may be an indicator



Fig 1. Anterior view of mandibular premolar/canine segment showing erosion defects associated with gastroesophageal reflux disease combined with aggressive toothbrushing and mastication of fibrous food.



Fig. 2. Lingual view of maxillary anterior segment showing tooth surface loss associated with pyloric sphincter obstruction.



Fig. 3. Occlusal view of the anterior maxillary region showing tooth surface loss associated with bulimic purging of stomach contents.



Fig. 4. Facial view of the lower dentition showing tooth surface wear that is characteristic of erosion caused by acidic drinks.

of involuntary purging of the stomach contents, as in the case of severe chronic gastroesophageal reflux disease (GERD) or pyloric sphincter stenosis obstruction (Fig. 2).^{23,28,29} Similar signs of dental erosion might be manifested by perimolysis (erosion from gastric acids due to regurgitation) associated with voluntary purging by bulimic persons (Fig. 3).²³

The patients' health and dietary histories in these cases, combined with clinical signs, often disclose the causative etiology. The typical amalgamated features of these erosion lesions are that they maintain normal tissue color, translucency, texture, and surface hardness. Accordingly, they may bear much resemblance to mechanical wear defects caused by tooth brushing, abfraction, and attrition, or occlusal and incisal wear due to mastication, and need to be differentiated from one

another.^{24-26,30} The radiographic image of the eroded enamel defect may display grades of radiolucency that correspond with the extent of the surface defect and the type of tissues lost. In general, the radiolucency corresponds with the lesion's location and dimension. This radiolucent image is frequently seen bordered by a definitive line of demarcation, characterizing dental erosion from tooth decay, although it is a common feature in toothbrush abrasion defects. The aforementioned manifestations of TSL of enamel and possibly dentin precisely fit the classical description of dental erosion defined by Hubert Stones in 1954 as:

*... a destructive process of enamel and dentin by the chemical action of acids from various sources that leads to tooth surface loss, without involvement of microbial organisms.*³¹

This definition falls short of encompassing the variety of erosion lesions associated with a modern day diet, known as *contemporary* lesions. The clinical features of contemporary erosion lesions are diverse. The variety of clinical signs epitomizes the uniqueness of contemporary erosion lesions. For identification and differentiation purposes, they may be categorized into 3 types that incorporate a select range of features.

Type I erosion lesion

This lesion correlates to the conventional dental erosion lesion. It is characterized by TSL involving enamel and/or dentin. This type of lesion maintains normal tissue color and surface texture (Fig. 1-4).

A Type 1 lesion is frequently observed in association with systemic, dietary, and environmental etiologies, and its location

is consistent with the causative etiologies.^{16,20,31} Individuals who are vulnerable to this type of dental erosion are those with systemic conditions that propel gastric juice into the oral cavity either by GERD or purging, avid consumers of regular or diet soda for prolonged durations, persons who consume excessive amounts of citrus fruits and/or juice on a daily basis, and individuals exposed to unmonitored chlorinated water in swimming pools.^{14,16,20,21}

TSL due to dietary acids may be seen even in the dentitions of individuals who practice good oral home care and are conscientious of their dental health and physical appearance. There is a strong possibility that these individuals may practice aggressive toothbrushing, possibly several times per day. They are also more likely to prefer a low carbohydrate and high fiber diet; hence their preference of citrus fruits and non-sweetened acidic beverages. Constant exposure of the dentition to these acids combined with the mechanical processes in the oral cavity accelerates the removal of newly decalcified, softened enamel and/or dentin.³⁰

Differential diagnosis

Because of the close resemblance of the general characteristics of these erosion defects compared to TSL due to aggressive toothbrushing, attrition, or mastication, it is critical to differentiate between these lesions. Unlike the aforementioned erosion defects, toothbrush abrasion often develops as a result of the differential wear resistance of coronal enamel and root dentin in the presence of an aggressive horizontal toothbrushing technique. This could result in gingival margin recession followed by the formation of defects located on the cervical third of the exposed facial aspect of the root surface (Fig. 5). Toothbrushing defects are often localized on a single tooth or a group of adjacent teeth; one or more arches may be affected, but to various degrees. The incisor-canine and premolar regions are common sites for these defects, and they often present as either a linear or V-shape, or as a shallow depression depending on the extent of tissue damage.

An occasional misnomer with subsequent confusion lies between the mechanically induced wear defects due



Fig. 5. Facial view showing white enamel spots associated with excessive daily intake of orange juice for 3 years.



Fig. 6. Facial view of mandibular incisors showing yellowish-brown stained cervical enamel associated with chronic intake of large amounts of regular soda. Note the remainder of the dentition is totally destroyed by erosion.

to toothbrushing and abfraction. These 2 defects may appear similar in configuration, but their initiation mechanisms and exact locations differ. Unlike toothbrushing defects, abfraction is a consolidation of 2 mechanisms, an abrasion process that is preceded by fracture or cracking of the cervical enamel. Cracking of the cervical enamel occurs as a result of flexion of an isolated tooth situated in a maloccluding position and subjected to occlusal strain. Microscopic compression cracks develop in the thin cervical enamel (often on the facial aspect) resulting from selective flexion of the tissues involved, causing the dentin to yield under the strain while the enamel stays intact. The ensuing microscopic cracks in the enamel progress both superficially and in depth, separating enamel prisms that then become readily dislodged by the aggressive and abrasive force of toothbrushing. Abfraction occurs at the cervical segment of coronal enamel, while toothbrush abrasion usually affects the cervical segment of root dentin. Both of these wear defects maintain natural tooth color and surface consistency.²⁴⁻²⁷

Attrition and demastication wear of the occlusal/incisal aspects are unlike erosion defects that may appear in the same locations. The 2 mechanical wear defects demonstrate flat, smooth (horizontal or slanted) planes that are separated from adjacent aspects of the tooth by a sharp demarcation line (Fig. 6). Erosion lesions display inverted cusps with incisal edges on a semiflat occlusal/incisal surface that has softened transitional curvature

between the worn biting surface and adjacent aspects, without a distinctive line of demarcation (Fig. 7). The natural tooth color and surface consistency are maintained for both the attrition/demastication wear defects and the occlusal/incisal TSLs due to erosion.

Type II erosion lesion

This lesion is characterized by discolored opaque white or tan enamel, with or without brown spots (Fig. 7-9). The dentin, if exposed, tends to display either a light or dark tan color. These lightly discolored dental tissues (LDDT) distinguish a Type II erosion lesion.

The uniqueness of this particular type of lesion is the generalized increased optical opacity of enamel that presents as a chalky white color throughout the dentition, due to a decalcification process (Fig. 8).¹⁴ Occasionally, the discolored enamel displays a dirty tan hue with or without brown spots (Fig. 9). These spots are likely to be the result of extrinsic stains from food and/or beverages deposited onto the porous decalcified surface, or they may be secondary to the intrinsic discoloration of the enamel and/or dentin. The enamel surface could have a chalky, easily scored texture. The excessive intake of acidic soft drinks, aggravated by the lack of routine oral home care, are common factors detected in individuals affected by this type of dental erosion. This combination, along with the consumption of soft food and lack of a high fiber diet, allows for the decalcification of the enamel surface to



Fig. 7. Facial view showing chalk-white cervical enamel associated with chronic daily intake of large amounts of diet soda.



Fig. 8. Facial view showing brown erosion lesions of dentin associated with excessive consumption of sugar-sweetened soda.



Fig. 9. Erosion lesions at various stages of development associated with intake of soda. *Left.* Buccal view of anterior erosion. *Right.* Buccal view of posterior erosion. Note that the maxillary incisors were restored with resin-based tooth-colored material.

continue undisturbed, becoming softened, permeable, and porous. This allows for more acid to come in contact with the subsurface layer, leading to decalcification and increased porosity. Repetitive assaults of acid increase the thickness of decalcified enamel and boost the intensity of its optical opacity. Although the erosive fluid inundates the entire dentition, reaching every surface, the highly opaque decalcified enamel is most often seen in the regions that are deprived from a constant bathing in salivary fluid. The lingual aspects of the dentition may therefore be selectively affected, depending on the amount of salivary flow present, the duration of acidic fluid contact with dentition surfaces, and the destructive progression of erosion lesions from adjacent proximal or biting surfaces.

Decalcification of the underlying dentin leads to the softening and consequential undermining of the unsupported brittle enamel that eventually crumbles (Fig. 10).⁸ The exposed decalcified softened dentin could display a range of light to dark

tan discoloration, and while the surface may retain its smooth integrity, it may be cartilaginous or have a soft or mushy consistency with an uneven topography. Sequential breakdown of the underlying dentin hastens the destruction of the tooth core. In turn, concomitant softening of surface and subsurface layers of dentin leads to partial or total loss of coronal segments.

Differential diagnosis

At first glance, the entire facial enamel of the affected teeth may appear to have homogenous opacity. The enamel surface that displays this increased opacity could also exhibit opaque white lines running across the coronal part of the tooth following the pattern of the perikymata. The opaque enamel and the opaque white lines mimic dental fluorosis. The patient's history of dental health and dietary patterns, the knowledge of the regional water fluoridation at the individual's birthplace and the clinical signs of these lesions may help to exclude dental fluorosis in the diagnosis. Another presentation of the

decalcified discolored enamel associated with the erosion process is that the facial enamel of the incisal or occlusal third of the affected teeth displays a light degree of opaque white discoloration, while the cervical two-thirds display an optical opacity that is more intense (Fig. 10). The decalcified, opaque white, cervical segment of the affected dentition could resemble early supragingival calculus. This initial perception may change when the exact nature of these lesions is disclosed by careful clinical examination and surface exploration. Furthermore, radiographic images of the lesions and calculus deposits in similar locations could verify the suspected clinical findings. The decalcified enamel image displays degrees of radiolucency, while calculus deposits could show degrees of radiopacity, subject to the level of maturation.

Type III erosion lesion

This lesion is identified by dark brown dentin discoloration (DBDD) with or without overlaying decalcified enamel islands (Fig. 11 and 12).^{8,14}



Fig. 10. Facial view of the maxillary incisors and canines showing tooth surface loss associated with excessive toothbrushing.



Fig. 11. Anterior view showing slanted orientations associated with attrition wear on teeth No. 5 and 28.



Fig. 12. Occlusal view of premolars showing erosion. Note the smooth transition of line angles as well as ditching into dentin.

The location, distribution, and progression of this type of lesion are strikingly identical to those of Type II. The discolored dentin could exhibit brown discoloration that ranges from dark brown with or without a bluish tint. This discoloration appears to intensify with deeper penetration of the lesion into the dentin. The affected dentin may maintain its topography, have a cartilaginous consistency, or may become a mushy structureless mass. The localized brown discolored dentin may also be surrounded by a halo of brittle, white, decalcified enamel margins that readily crumble (Fig. 12). This type of erosion lesion is frequently associated with the intake of excessively large volumes of regular sugar-sweetened soda for prolonged periods.¹⁴ The acids and sugar ingredients in these beverages act synchronously to impart the intense brown discoloration in the organic rich dentin matrix. This biochemical reaction causes a nonenzymatic browning known as the *Maillard reaction*, or nonenzymatic glycosylation that is primarily responsible for the browning of dentin in erosion lesions.^{32,33}

Differential diagnosis

The browning of dentin in Type III lesions could create a pseudo-image that can be mistaken for dental caries, dental fluorosis, and/or heavy tetracycline staining. A detailed review of the patient's systemic health history (including medications taken during formative years and the regional water fluoridation) may shed light

on whether dental fluorosis or tetracycline staining are the causative etiologies of the dentin staining. When considering the possibility of dental caries in lieu of dental erosion, an examiner should take into consideration that an acid/sugar reaction takes place in the genesis of both lesions, and although the sources of acid differ, those of sugar in both cases are the same. Therefore, for accurate identification of these lesions, a comprehensive protocol of investigations must be followed that should include the distinctive characteristics of the lesions, their location, relative surface to depth proportions, distribution, progression, extent, sequelae and symptoms, radiographic imaging, as well as the condition of the associated periodontal tissue and the presence or absence of plaque deposits. These efforts in tandem with obtaining a caries risk assessment and a review of the patient's oral hygiene and dietary history are essential to establishing an accurate diagnosis and identifying respective etiologies.

Discussion

The definition of dental erosion as "a tooth surface loss due to a chemical process involving acids and tooth structures without bacterial involvement" was stated 6 decades ago by Stone.³¹ This definition continues to be the guiding parameter used for diagnosing dental erosion.³¹ It has remained unaltered, modified, or updated to adjust to the changes in diet and dietary habits, as witnessed by the gradual but

consistent departure from nonprocessed food and beverages.³⁴⁻³⁶ During the past 50 years, the daily diet in industrialized modern societies has become inundated with processed, canned, and fast foods, and acidic beverages.³⁶ This regrettable shift in diet from water, milk, natural juice, and fresh produce to an increased consumption of food and drinks that are laden with saturated fat, carbohydrates, salt, and acids has influenced the features of the associated erosion lesions.^{34,37-39} This change in the clinical topographies of the traditionally known dental erosion have created a disparity between the features of the conventional lesions and today's contemporary erosion lesions. This shift poses a challenge to the existing definition of dental erosion lesions and has contributed to the quandary of diagnosing these lesions. Compounding this identification dilemma is the potential of insufficient information from the patient's systemic health profile, dental health and dietary history, and oral hygiene habits that could help to reveal a lesion's etiology.^{8,14}

The worldwide emergence of erosion as a serious dental hard tissue disease has been a source of great concern since the 1980s, eventually prompting the World Health Organization to declare erosion as a major dental health threat at the turn of the 21st century.^{1,2,9,40} In order to properly manage this dental hard tissue disease, proper identification and diagnosis of the diversified lesions are of paramount importance. Examples of the variety of features

of the contemporary erosion lesions associated with today's modern acidic diet have been reported in the literature.^{6-8,14,16,17,22}

Certain types of erosion lesions have been routinely observed among the large patient pool of a major US university dental clinic for more than 3 decades. Additionally, over the past 3 years, the unique features of Types I, II, and III lesions were repeatedly detected by the author during multiple field evaluations of the dental health of the Central Appalachian population through the clinics of the Remote Area Medical and Mission of Mercy. The observed lesions were associated with excessive intake of large volumes of acidic carbonated beverages, whose daily intake per person was estimated to be well above the recommended average.^{8,11,14} Furthermore, observations of morphological, topographical, and geometrical changes of disassociated human teeth that were subjected to various acidic fluids for prolonged duration were the subject of *in vitro* experiments.⁴⁰ The outcome of these experimental observations revealed the diverse topographic characteristics of Types II and III erosion lesions.⁴¹

Due to the potential pitfalls and possible misdiagnosis of contemporary erosion lesions and the subsequent selection of an incorrect approach for management, redefining dental erosion should be considered in order to include the information needed to diagnose these lesions.^{8,14,16,23} The revised definition should reflect the process of erosion and characterize the contemporary lesions/defects. This definition should be used as a universal tool and a guiding parameter to assist in the process of accurate diagnosis and to facilitate communication among dental professionals. It should also provide a platform and solid base for discussion among dental health care providers and their patients. The proposed definition may be stated as follows:

Dental erosion is a biochemical process in which acid from various sources reacts with the hydroxyapatite of enamel/dentin, without the involvement of microbial organisms. The resultant erosion lesion/defect may be manifested clinically by various topographic features that include tooth surface loss, chalky white or tan enamel, and brown caries-like dentin lesions. These features are subject to the

culprit acid, presence or absence of sugar in situ, and the biomechanical factors in the oral cavity.

Conclusion

Arising from the clinical examples given in this report and previously described in the literature, there are 3 types of lesions that constitute the contemporary profile of dental erosion. Some contemporary erosion lesions (Type I) display features that resemble those of the conventional TSL, which are similar to the clinical features of mechanical tooth wear. Others (Type II) are characterized by discolored enamel (either opaque white or tan, with or without brown spots) that resembles early carious lesions and/or dental fluorosis. The remaining set of lesions (Type III) display DBDD that is remarkably similar to that of dental caries, severe dental fluorosis, and/or tetracycline staining. The genesis of these lesions and their respective clinical features differ. In order to achieve an accurate differential diagnosis, one should investigate the systemic, dental, and dietary health histories, as well as assess caries risk for possible causative etiologies and other factors that may contribute to the discoloration of the dental hard tissues. The clinical characteristics of each lesion, such as location, proportions, distribution; radiographic images; and associated periodontal health; and a review of the patient's oral hygiene regimen must be taken into consideration as important tools to solve the diagnostic puzzle.

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