Dental Erosion: Understanding This Pervasive Condition

JÚNIO S. ALMEIDA E SILVA, DDS, MSC*†, **LUIZ NARCISO BARATIERI,** DDS, MSC, PhD‡, **EDSON ARAUJO,** DDS, MSC, PhD§, **NICOLAS WIDMER,** DDS, PhD¶

ABSTRACT

Dental erosion is a contemporary disease, mostly because of the change of the eating patterns that currently exist in society. It is a "silent" and multifactorial disease, and is highly influenced by habits and lifestyles. The prevalence of dental erosion has considerably increased, with this condition currently standing as a great challenge for the clinician, regarding the diagnosis, identification of the etiological factors, prevention, and execution of an adequate treatment. This article presents a dental erosion review and a case report of a restorative treatment of dental erosion lesions using a combination of bonded ceramic overlays to reestablish vertical dimension and composite resin to restore the worn palatal and incisal surfaces of the anterior upper teeth. Adequate function and esthetics can be achieved with this approach.

CLINICAL SIGNIFICANCE

It is essential for the practitioner to establish a correct and early diagnosis of dental erosion, as this condition is of growing concern and is becoming more prevalent in current society.

(J Esthet Restor Dent ••:••–••, 2011)

INTRODUCTION

Tooth wear seems to be one of the most concerning problems of the current dental practice. The etiology of this wear comes from well-known mechanical processes, such as attrition, abrasion, and abfraction, and also from a chemical process, which is dental erosion.

Currently, dental erosion is a great challenge for the practitioner as far as diagnosing the condition, identifying the etiological factors, executing an adequate treatment, and instituting preventive measures.

Tooth wear, when noticed, is frequently diagnosed as abrasion and attrition. Usually, when lesions caused by erosive challenges are identified, the lesions are already in advanced stages. There are occasions when patients notice that their teeth are shortened or thinner before their dentists.

As a matter of fact, most practitioners do not seem to be sufficiently informed and are unable to fully establish a correct diagnosis regarding the etiology of tooth wear. Although there is usually not only one etiological factor contributing to the loss of hard dental tissue, it is important that dental practitioners be aware that

*PhD student, Operative Dentistry Division, Federal University of Santa Catarina, Florianópolis, Brazil
†Visiting Researcher, Department of Prosthetic Dentistry, Ludwig-Maximilians University, Munich, Germany
‡PhD Professor and Chair, Operative Dentistry Division, Federal University of Santa Catarina, Florianópolis, Brazil
§PhD Assistant Professor, Integrated Clinic, Operative Dentistry Division, Federal University of Santa, Catarina, Florianópolis, Brazil
¶DDS, PhD, Federal University of Bern, Switzerland

 DOI 10.1111/j.1708-8240.2011.00451.x

erosion lesions are sequela from intrinsic or extrinsic acid attacks and may possibly be identified years after the beginning of the erosion activity.

This article reviews the literature with regards to the main aspects of dental erosion while also presenting a case report of a restorative treatment of dental erosion lesions using a combination of bonded ceramic overlays to reestablish vertical dimension and composite resin to restore the worn palatal and incisal surfaces of the anterior upper teeth.

DENTAL EROSION

Dental erosion is a progressive loss of dental tissue caused by chemical processes without bacterial involvement.[1] It is a "silent," multifactorial disease, and is highly influenced by personal habits and lifestyles. Erosion can be attributed to numerous factors, including the ingestion of organic and inorganic acidic substances. Acids responsible for dental erosion come from intrinsic and extrinsic sources.[1–3]

Extrinsic erosion is caused by exogenous acids that may come from an individual's diet, work environment, swimming pool water, medicine, and from some drugs.[2] Intrinsic erosion is the result of endogenous acids that contact teeth during vomiting and regurgitation events, especially in patients who suffer from gastroesophageal reflux disease (GERD) and bulimia nervosa.[1,2]

Dental erosion occurs either by hydrogen ion attack or by the action of chelating anions.[4] The process of dental erosion is modulated by various determinants and modifying factors, which will be discussed further.

BIOLOGICAL CONSEQUENCES

The result of constant exposure to erosive agents promotes changes in the physical properties of dental structures. The result of episodic exposure to acidic challenges decreases enamel microhardness,[5] which upon exposure to mechanical forces, makes the dental surface more susceptible to disruption. When the erosive process occurs in dentin, the dentin-pulp complex responds to the attacks by producing reactionary dentin and occluding the dentinal tubules in order to compensate for the tissue loss. If the progression of dental erosion surpasses the dentin-pulp complex reparative capacity, there might be some complications, such as toothache, dental sensitivity, pulpal inflammation, pulp necrosis, and periapical lesions.[3]

CLINICAL CONSEQUENCES

The primary signs of dental erosion are: diminishing enamel luster; absence of macroscopic plaque; and dental surfaces that have become rounded and polished because of the loss of microanatomy. After the initial dental erosion, some features can be observed, such as smoothing out of developmental pits and grooves, dentin exposure, prominent restorations that are elevated above the surrounding tooth structure, and well-defined concavities of dentin on the occlusal and incisal surfaces, especially on the cusp tips of the posterior teeth.[6] In more advanced cases, extensive mineral loss can lead to tooth shortening, which can promote functional and esthetic problems.

Convex teeth areas, such as proximal ridges, gradually become flat and even concave. In severe cases, dental morphology can be totally lost (Figures 1 and 2). Dental erosion in patients who have exposed root dentin may

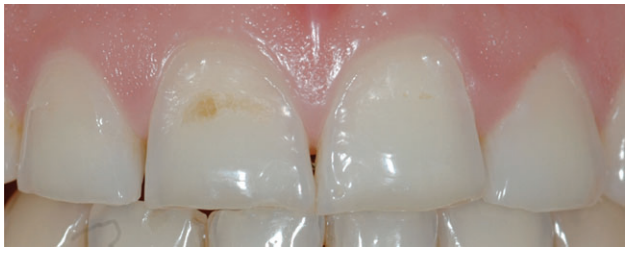

FIGURE 1. Dental erosion by abusive intake of citric fruit juices. Maxillary teeth display loss of microanatomy and polished and rounded surfaces. Observe the dentin exposure on the labial surface of the maxillary right incisor. The proximal ridges of the maxillary left and right incisors present a diminishing of the natural convexity.

DOI 10.1111/j.1708-8240.2011.00451.x

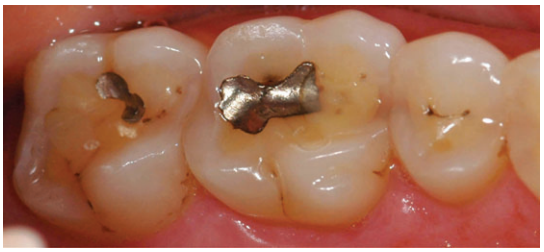

FIGURE 2. Dental erosion by bulimia nervosa. Observe the loss of occlusal enamel, dentin exposure and prominent amalgam restorations on the molars. Dentin concavities, which are present on the occlusal surfaces of the molars, are formed due to the difference of the acid dissolution progression between dentin and the surrounding enamel. The premolar occlusal surface displays a loss of microanatomy, diminishing enamel luster, and a smoothing out of the developmental pits and grooves.

develop more rapidly because of the lower dissolution resistance that dentin presents.[3–6] If there is no professional intervention and elimination of the etiological factors, the outcome of this disease may be the total loss of the teeth.

PREVALENCE AND INCIDENCE OF DENTAL EROSION

Both the prevalence and incidence of dental erosion have considerably increased, mainly among young adults and children.[1–10] Lifestyles have changed through the decades, with the consumption of acidic food and carbonated beverages increasing.[7] Currently, fast food consumption is a common factor in society. The popularity of this kind of food item, along with the appealing publicity that seduces children and teenagers, may bring some harmful consequences, such as dental erosion.

Soft drink consumption in the United States increased by 300% over the last 20 years, with serving sizes increasing from 185 g in the 1950s to 340 g in the 1960s and to 570 g in the late 1990s.[11] It seems unavoidable that the increase of soft drink intake may lead to pathological tooth wear. Around 1995, between 56 and 85% of school-aged children in the United States consumed at least one soft drink daily.[12]

Generally, specific epidemiological surveys of tooth wear are not routinely undertaken. There are, however, some isolated surveys that evaluate the prevalence of dental erosion in different populations with different ages.[9–13] It is difficult to establish comparisons between surveys because of the different samples and evaluation methods applied on each one. However, there is a consensus that tooth wear is a common condition and can be started as soon as the first tooth surface reaches the oral cavity and is exposed to erosive agents. Deciduous and permanent teeth are equally involved. Additionally, if occlusal stress is present, the progression of erosion increases considerably.[9] Erosive tooth wear can be found on every surface of all teeth.[9] Taking into consideration the social and cultural differences of each population, a common factor is evident: there is a greater prevalence of dental erosion among young adults and children, which is associated with the consumption of acidic food and beverages, psychosomatic eating disorders, and current lifestyles.

RISK GROUPS

Risk groups are comprised of those individuals that present one or more dental erosion etiological factor(s). In order to didactically organize them, they have been divided into extrinsic and intrinsic erosion groups. It is important though to emphasize that there are patients who present both types of dental erosion (extrinsic and intrinsic) simultaneously.

Extrinsic Erosion

This group is constituted by those whose teeth are frequently attacked by exogenous acids, such as soft drinks, fruit juices, pickles, fresh fruits, and yogurt; essentially, the majority of food that presents a pH lower than the critical value for enamel demineralization.[14] Also included in this group are individuals that: abusively use vinegar on salads or low pH mouth rinses[15]; are daily exposed to acidic working environment[16]; frequently ingest electrolytic replenishers[2]; and take illicit drugs, such as methamphetamine, cocaine, and ecstasy[1,17,18] (Figure 1).

 DOI 10.1111/j.1708-8240.2011.00451.x

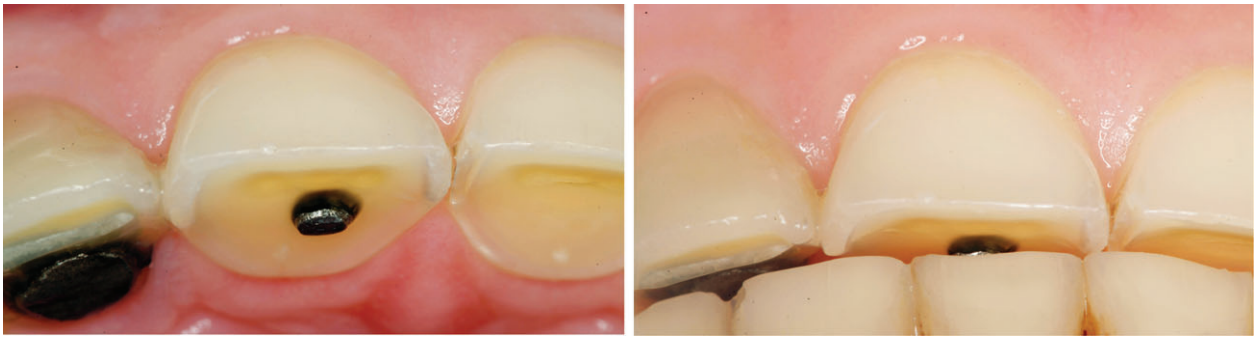

FIGURE 3. Dental erosion by gastroesophageal reflux disease. Observe the loss of palatal enamel of the maxillary teeth. Notice that, when occluding (right), the mandibular right incisor exclusively contacts the amalgam restoration of the maxillary right incisor.

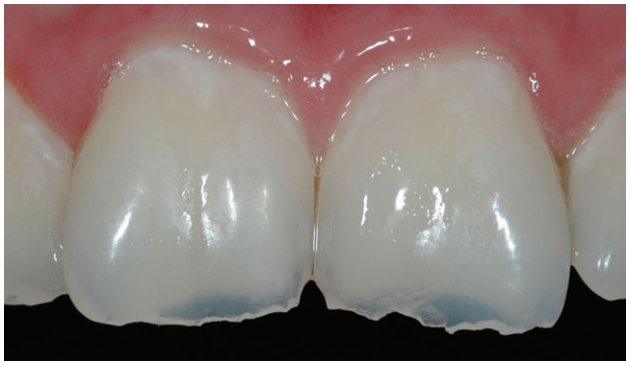

FIGURE 4. Young patient with gastroesophageal reflux disease. Notice that, due to the intrinsic source of acids, the palatal surfaces are worn more rapidly than the labial surfaces, thus leaving the incisal third with a sharpened blade appearance.

Intrinsic Erosion

This group is composed of patients whose teeth are attacked by endogenous acids during recurrent vomiting, regurgitation, or reflux. Gastroesophageal reflux, which is the major cause of intrinsic erosion, is a condition where acids from the stomach are regurgitated back to the oral cavity,[9] thus resulting in constant oral exposure to an acidic environment.[1] Psychosomatic eating disorders, such as bulimia nervosa and anorexia nervosa, are usually the main cause of vomiting and regurgitation. Also included in this group are alcoholics and patients that present gastrointestinal disorders, hiatal hernia, peptic, and duodenal ulcers.[2–6] (Figures 2–4).

PATHOPHYSIOLOGY OF DENTAL EROSION

The direct effect of the attack of hydrogen ions on the tooth surface is to combine with carbonate, phosphate, or both, and detach the mineral ions from the surface. Some acids, such as citric acid, have a more complex interaction. In water, these acids are dissociated into hydrogen ions and acidic anions. The hydrogen ions act exactly as aforementioned and directly attack the crystal surface whereas the acidic anions act by chelating calcium from the crystal surface. These acids have a double action and are very damaging to the tooth surface.[4]

How Enamel and Dentin React to Attacks by Erosive Agents

In order to contact enamel, a solution has to diffuse through the acquired pellicle, only then can it interact with enamel.[7] On the enamel surface, the hydrogen ions or chelating agents start to dissolve the enamel crystal. First, the prism sheath area and then the prism core are dissolved, leaving a honeycomb appearance. Then, fresh and unionized acids will diffuse into the interprismatic areas of enamel and further dissolve the mineral content underneath the surface. This further acid attack will lead to an outflow of ions and a rise in the pH in the surrounding environment.[3,4,7] When there is an elevation of the surrounding pH because of saturation of mineral ions, remineralization of the softened tissues that have not been directly etched away may occur, from the released ions and from other sources. If

DOI 10.1111/j.1708-8240.2011.00451.x

fluoride is present in the oral environment, fluorapatite can be formed, which is less soluble than the original enamel.[4]

The effects of acids on dentin have been largely investigated, as the length of time that teeth remain in the mouth is increasing, with dentinal coronal and root exposures that are gradually becoming common because of tooth wear and gingival recession, respectively.[19] The initial events in dentin demineralization are similar to those that occur in the enamel. However, because of the high organic content of dentin, diffusion of the demineralizing agents and mineral ions are partially stopped by the organic matrix, which acts as a barrier to acid diffusion and mineral release, thereby reducing the progression of the erosive process.[12–24] This difference in erosive processes between enamel and dentin does not mean that the erosive process in dentin is slower, on the contrary, the dentin substrate is more susceptible to acid dissolution because its hydroxyapatite crystals are smaller than that of enamel. Thus, there is a larger susceptible area for acid attack,[4] as found in dentinal concavities, or dental erosion clinical signs that are formed because of the different rates of acid dissolution between dentin and the surrounding enamel.

DETERMINANT FACTORS ON THE MODULATION OF DENTAL EROSION

The development of dental erosion is strongly influenced by determinant factors (biological, chemical, and behavioral) and by modifying factors (knowledge, systemic health, and socioeconomic status).

Biological Factors

Saliva

Saliva is the most important biological factor for the modulation of dental erosion. Saliva protects teeth by diluting and eliminating erosive agents from the oral environment and through its acid neutralization and buffering capacity. Saliva plays a role in reducing demineralization by forming acquired pellicle and aiding the adsorption of proteins and glycoproteins, while enhancing remineralization by providing calcium, phosphate, and fluoride to eroded enamel and dentin.[25] The relevance of saliva on the erosion process may be demonstrated by in vitro—no salivary protection and in situ—with salivary protection comparisons. In a study by West and colleagues[26] enamel erosion was drastically reduced, by the order of 10 times, in the in situ model.

The quantity and quality of saliva are equally important on the modulation of erosive agent attacks on the tooth surface. The bicarbonate level of saliva is directly related with salivary flow; therefore, saliva produced at low flow rates presents low pH and a reduced buffering capacity.[2] The impact of dental erosion in patients with salivary flow deficiencies clearly demonstrates the importance of saliva on the erosive process, which is demonstrated in studies that have linked low buffering capacity and salivary flow to dental erosion.[27–30]

Acquired Pellicle

Acquired pellicle is a protein-based layer that is rapidly formed on the tooth surface, shortly after its removal by tooth brushing, prophylaxis, or chemical dissolution.[19] This pellicle protects teeth because it acts as a barrier, or a semipermeable membrane, which prevents the direct contact of erosive agents on the tooth surface and serves as reservoir of remineralizing electrolytes,[31] which may influence the development of erosion.[19] With this in mind, it is important to instruct patients to not brush their teeth immediately before consuming acidic food or drink because it removes the acquired pellicle, thus leaving teeth less protected.

Tooth Structure

Susceptibility to dental erosion may vary according to each patient, as the developmental pattern of this disease may be influenced by the clinical history of each patient. This different developmental pattern is caused by different situations that can impair or modify the tooth, such as mechanical or chemical processes that include caries, erosion, abrasion, attrition, and abfraction.[19] With regards to tooth composition, exposure to saliva and fluorides have demonstrated effectiveness on the remineralization of eroded enamel. Therefore, a structural difference, such as the formation

 DOI 10.1111/j.1708-8240.2011.00451.x

of fluorapatite, may influence and modulate the development of dental erosion.[32,33]

There is some controversy regarding the susceptibility of deciduous teeth to dental erosion compared to the permanent dentition. Because of differences in mineralization, permanent teeth are less prone to acid dissolution than deciduous teeth.[19] However, it has also been reported that there are no differences in acid dissolution susceptibility between these two substrates when exposed to acidic beverages in an in vitro comparison.[34] Although there is a higher prevalence of dental erosion among children when compared with adults, this difference might be caused by a greater consumption of acidic food and beverages and to the overlap of the erosive challenge with abrasive procedures, as the primary enamel is supposed to be less resistant to abrasion.[19,34–36]

Soft Tissues

Soft tissues play a role in tooth wear. One of the most severe manifestations of dental erosion is found on palatal surfaces of the upper teeth due to mechanical abrasion from the tongue on the dental tissue just after the consumption of an erosive agent.[2]

BEHAVIORAL FACTORS

Although biological factors influence the response of each person to dental erosion, behavioral patterns influence the biological response to erosive challenges because they are capable of modifying the oral environment by turning it more or less susceptible to the development of dental erosion. Behavioral aspects influence the intensity, localization, and type of erosion lesions.

Unhealthy Lifestyles

Alcoholic individuals may be at risk of developing dental erosion due to the symptoms of alcoholism, such as recurrent vomiting and gastrointestinal reflux.[37]

The usage of drugs such as "ecstasy" (3,4-methylenedioxy-methamphetamine) is known for causing tooth wear. Ecstasy users report symptoms such as dry mouth, hyperthermia, clenching, and grinding during dances or "raves" even hours after the mental effects of the drugs, indicating that the physical wear may continue for some time after the drug consumption. The dry mouth and general hyperthermia are usually relieved by drinking soft drinks, many of which are acidic and thus, potentially erosive to the teeth. The influence of grinding or clenching from bruxist activity is heightened in a mouth with poor salivary lubrication.[17] As indicated previously, a low salivary flow theoretically means the presence of a low buffering capacity; thus, acid erosion is increased under this condition. This combination of habits and symptoms may manifest as greater levels of tooth wear.[38] The common reluctance of the patient to admit to the use of such drugs impairs the establishment of an accurate diagnosis and appropriate treatment plan.

Healthy Lifestyles

Although it seems to be contradictory that a healthy lifestyle may be linked to a disease, it is important to emphasize that people involved in sports and exercise may be at risk of developing dental erosion due to the consumption of sport drinks, replenishers, fruit juices, and other acidic beverages. The benefits of exercise are well proven; however, exercise increases the loss of body fluids and may lead to dehydration and a reduction of salivary flow. The need for fluid intake, to satisfy an energy requirement, during a time of decreased salivary flow can create a proper condition for the development of dental erosion. The consumption of fruits and juices is part of several diet plans, which also may put those on such plans at greater risk of developing dental erosion.[2]

Nutritional Habits

Nutrition plays a key hole in dental erosion. The ingestion of low pH products may cause demineralization of dental hard tissues. Dental erosion can occur due to the frequent intake of soft drinks, fruit juices, pickles, fresh fruits, yogurt, and other food items.

DOI 10.1111/j.1708-8240.2011.00451.x

Habits such as lemon sucking and soft drink swishing expose enamel and dentin to an acidic environment for a longer period of time, which may cause greater demineralization. The consumption of acidic foods and beverages among patients with dental erosion is higher than among regular patients.[39]

Phosphoric acid, usually found in soft drinks, is three times more erosive than organic acids. The incidence of dental erosion in a population grows with an increase in consumption of soft drinks. Citric, tartaric, maleic, and lactic acids are examples of organic acids. Citric and maleic acids are predominantly found in fruits and in their derivate products. Tartaric acid is present in grapes and wines. Dental erosion may occur when any of these beverages or foods is abused.[40,41]

Vegetarian diets, in which fruit consumption may be more than 66% of the overall food intake, may lead their followers to develop dental erosion.[40]

CHEMICAL FACTORS

The term "chemical factors" is used to describe parameters inherent to erosive beverages, food, and other products.[42] The chemical properties of the food play an important role in dental erosion. The erosive potential of foods and beverages does not depend exclusively on the pH value, but also upon their mineral content, buffering capacity, and calcium-chelation properties. The pH value and calcium and fluoride content of a drink or foodstuff determines the degree of mineral saturation with respect to the teeth, which is the driving force for erosion,[42] as supersaturated solutions are not capable of dissolving tooth structure. There is no clear-cut critical pH for erosion as there is for caries, for even at a low pH, it is possible that other factors are strong enough to prevent erosion.[42] Therefore, components such as calcium, phosphate, and fluoride may lessen the erosive capacity of some drinks and foods.[9] For instance, some orange juices are enriched with calcium in order to reduce their erosive potential.[43]

MODIFYING FACTORS OF DENTAL EROSION

Systemic Health

Some systemic diseases, such as Sjögren's syndrome; medicines, such as acetylsalicylic acid when taken daily; hydrochloric acid for treatment of stomach disorders, diuretics and antidepressive medicines and therapies that involve irradiation of salivary glands all adversely affect salivary production, thus interfering with the biological protection provided by saliva. Even without excessive exposure to erosive agents, hyposalivation may induce dental erosion, as low salivary flow leads to low buffering capacity; therefore, acid elimination in the oral cavity is decreased as well as the saliva remineralization capacity.[44]

Knowledge

An awareness of the risks and activity of dental erosion, as well as an understanding of the erosive potential of drinks and foodstuff, is an important aspect in changing the initiation and progression of this disease. Patients who show signs and symptoms of dental erosion are often not aware of, and might be confused about, the erosive characteristics of items of their diet[7] or about any modifying factors their dietary intake might have. A thorough knowledge of all the dental erosion features is also needed by the dentist in order to determine the patient's risk of dental erosion and to institute preventive and curative measures.

Socioeconomic Status

Socioeconomic status might be linked to many of the previously mentioned factors, such as systemic health and knowledge, and thus indirectly influence the development of dental erosion. They can determine the quality of the dental evaluation, which might lead to an incorrect diagnosis and treatment. Nevertheless, whether dental erosion is a disorder of affluence remains unclear. However, social deprivation might also be related to nonfluoridated districts. Children in nonfluoridated districts are 1.5 times more likely to

 DOI 10.1111/j.1708-8240.2011.00451.x

have smooth surface wear compared with children in fluoridated districts.[45] Although the literature concerning the influence of socioeconomic status on dental erosion is still conflicting, the trend is for more tooth wear among the children not exposed to fluoridation of their residential water supply, further supporting a protective role.[45]

INTERACTION OF DENTAL EROSION AND MODIFYING FACTORS

Many people, with different modifying factors, frequently ingest fruit juices, carbonated beverages, and acidic foods that have an erosive potential. Therefore, the question is: Why do many people maintain their teeth for a lifetime, considering that they are constantly exposed to erosive challenges every day? On the other hand, why do some people suffer rapid and irreversible destruction of their teeth by erosion?[2]

The development and intensity of dental erosion depends on the interplay of the previous factors.[7] Determinant factors play a direct role in the development of dental erosion, whereas modifying factors synergistically act on this disease. Over time, the biological, behavioral, and chemical factors, along with the modifier factors and their possible combinations, might start or prevent dental erosion.[7] Hydrogen ion concentration alone does not explain the erosive potential of food and drink. All factors must be taken into consideration. The interplay of these factors is crucial and helps explain why some individuals exhibit more erosion than others, while also addressing the reason why, even when exposed to the same acid challenge in their diets, some people present distinct patterns of this disease, since each patient may show different modifying factors that produce different developmental patterns of dental erosion.

CASE REPORT

The following case is a restorative treatment of intrinsic erosion lesions, involving ceramic restorations on the maxillary and mandibular posterior teeth and composite resin restorations on the anterior maxillary teeth.

The etiology of this specific dental erosion was gastroesophageal reflux, which was corrected using a multidisciplinary approach involving a gastroenterological evaluation and treatment. The patient's main complaint was wear of the posterior teeth. Although the posterior teeth displayed noticeable wear, a clinical exam showed wear on the palatal surfaces of the anterior maxillary teeth as well.

The treatment plan was divided into two steps: increase of vertical dimension of occlusion (VDO) with indirect ceramic restorations on the posterior segment and direct composite resin restorations on the anterior segment.

After plaster models of both jaws were taken and wax build-up, a new VDO was created by the technician. The new VDO was tested on the patient with provisional overlays placed on the maxillary and mandibular posterior teeth. Then, the posterior teeth were prepared and ceramic onlays and overlays were placed on the posterior maxillary and mandibular teeth, with the exception of the maxillary left and right second molars, which did not display wear. Once the new VDO was reestablished with the posterior ceramic restorations, space was made to restore the worn palatal and incisal surfaces of the anterior maxillary teeth with composite resin.

Ceramics IPS Empress e.max (Ivoclar Vivadent, Schaan, Liechtenstein) was the material of choice for posterior segment due to its high longevity, maintenance of esthetic features such as anatomical form, color, adhesive bonding to dental hard tissues,[46,47] and also because one of the patient's demands was a long-lasting, esthetic and not time-consuming rehabilitation. Since no severe wear was present on the labial surfaces of the maxillary anterior teeth only the worn palatal and incisal surfaces were restored with composite resin because it is a reversible non-invasive technique that allowed us to preserve the labial surfaces of the maxillary anterior teeth. It is an esthetic solution and not time-consuming compared with indirect restorations. Function and esthetics were achieved with this approach (Figures 5–17).

DOI 10.1111/j.1708-8240.2011.00451.x

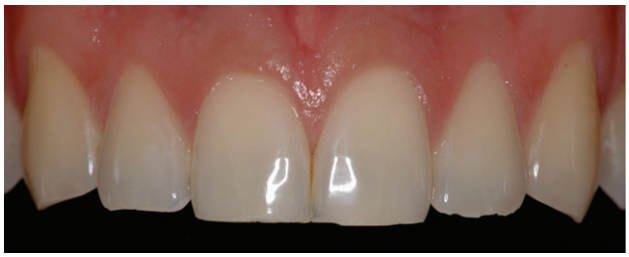

FIGURE 5. Observe the erosive tooth wear at the labial surface as well as at the incisal third of the maxillary incisors.

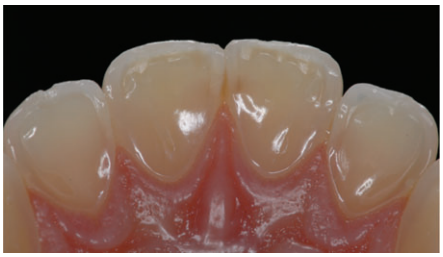

FIGURE 6. Observe the palatal erosive tooth wear.

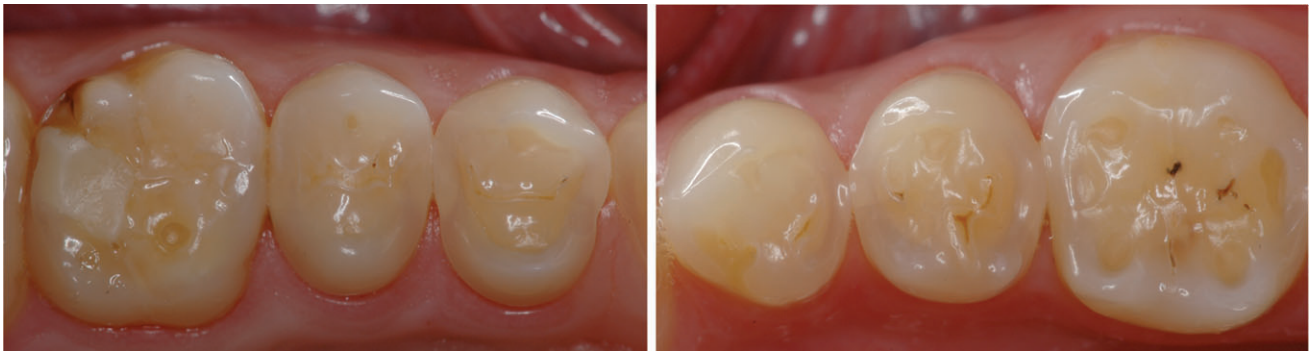

FIGURE 7. Right hemi-arches.

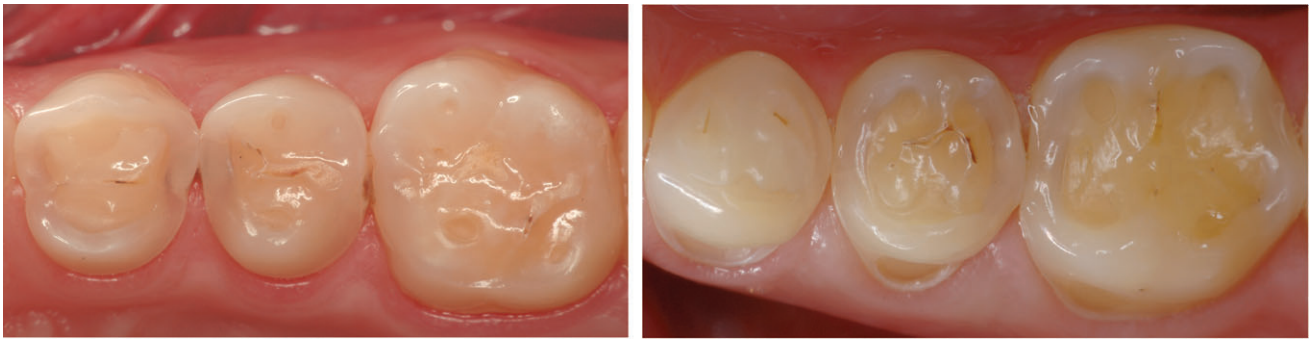

FIGURE 8. Left hemi-arches.

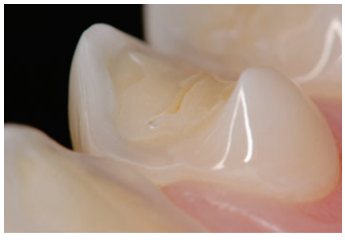

FIGURE 9. Observe the erosive tooth wear severity. Almost the entire occlusal enamel has been worn away.

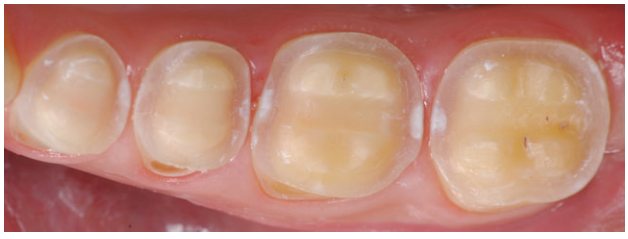

FIGURE 10. Occlusal view of the mandibular posterior teeth preparations.

 DOI 10.1111/j.1708-8240.2011.00451.x

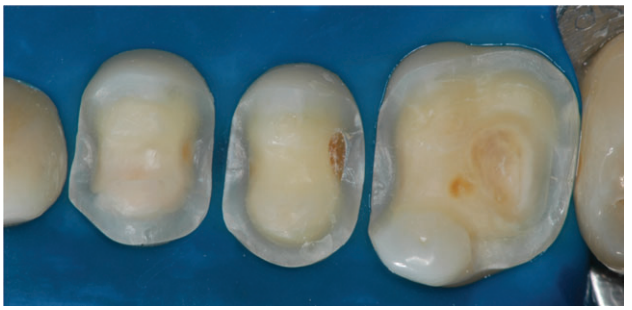

FIGURE 11. Occlusal view of the maxillary posterior teeth preparations before the luting procedures.

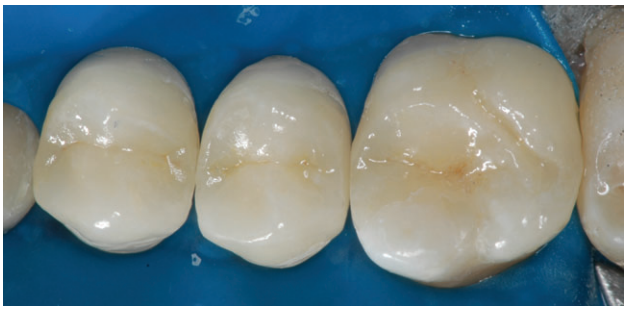

FIGURE 12. View after placement of the ceramic restorations on the maxillary posterior teeth.

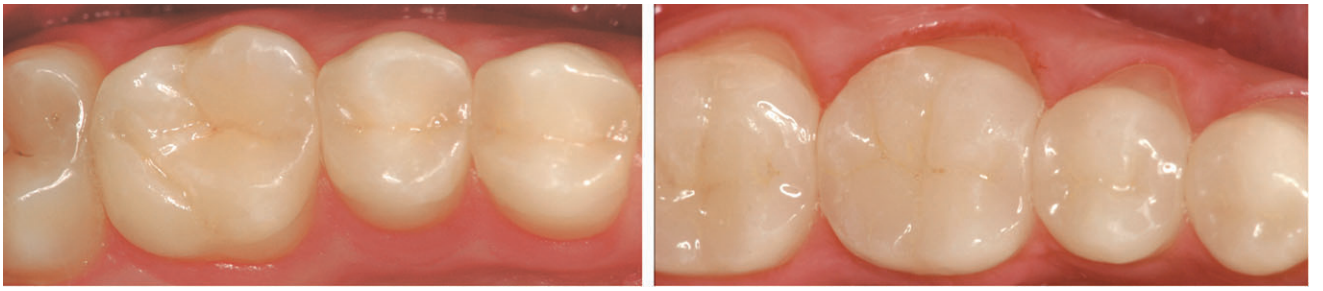

FIGURE 13. Right hemi-arches. Occlusal views after placement of the ceramic restorations.

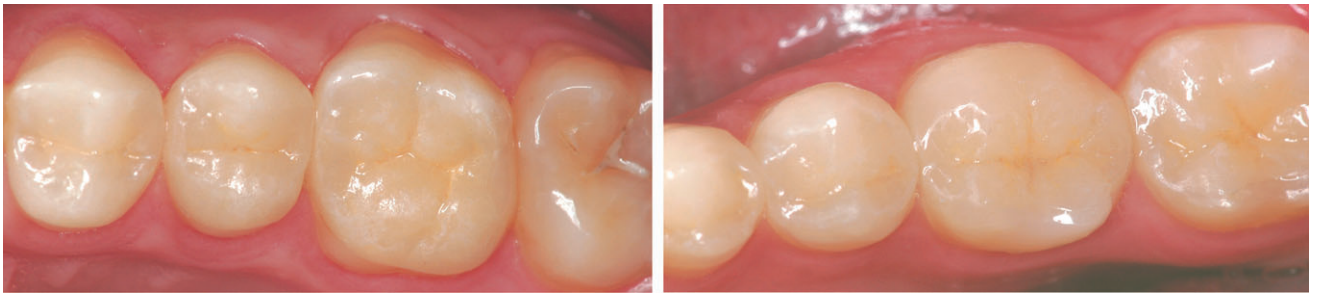

FIGURE 14. Left hemi-arches. Occlusal views after placement of the ceramic restorations.

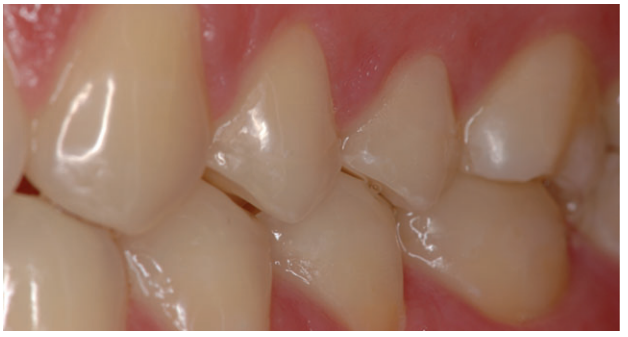

FIGURE 15. Lateral views after restorative treatment.

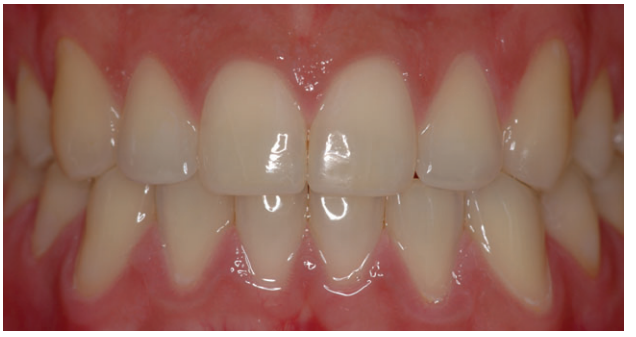

FIGURE 16. Frontal view after composite resin restorations, note the increase of vertical dimension.

DOI 10.1111/j.1708-8240.2011.00451.x

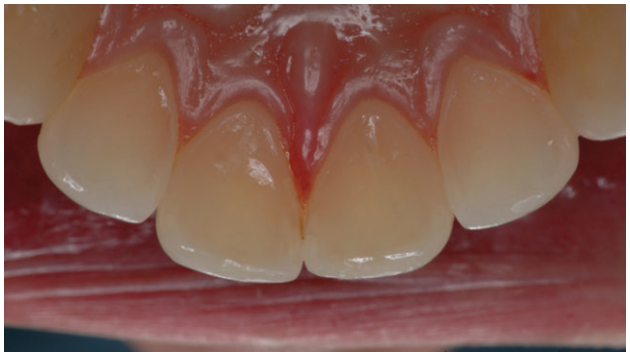

FIGURE 17. Composite resin restorations of the palatal surfaces of the maxillary anterior teeth.

CONCLUSION

Regardless of the etiology of dental erosion, it is rarely diagnosed at its early stages. Often, a multidisciplinary approach, potentially involving gastroenterological and psychological evaluations, is necessary. Awareness and understanding of this disease and the factors that modify it is crucial for establishing an early diagnosis and preventive and/or restorative treatment.

DISCLOSURE AND ACKNOWLEDGEMENTS

The authors thank Sylvio Monteiro Jr., DDS, MSc, and PhD, for his assistance. The authors do not have any financial interest in the companies whose materials are included in this article.

REFERENCES

1. Lussi A. Erosive tooth wear—a multifactorial condition of growing concern and increasing knowledge. In: Lussi A, editor. Dental erosion—from diagnosis to therapy. Bern: Karger; 2006, pp. 1–8.
2. Nunn JH, Gordon PH, Morris AJ, et al. Dental erosion—changing prevalence? A review of British national childrens' surveys. Int J Pae Diatr Dent 2003;13(2):98–105.
3. Jaeggi T, Lussi A. Prevalence, incidence and distribution of erosion. In: Lussi A, editor. Dental erosion—from diagnosis to therapy. Bern: Karger; 2006, pp. 44–65.
4. Shipley S, Taylor K, Mitchell W. Identifying causes of dental erosion. Gen Dent 2005;53(1):73–6.
5. Moss SJ. Dental erosion. Int Dent J 1998;48(6):529–39.
6. Ganss C. Definition of erosion and links to tooth wear. In: Lussi A, editor. Dental erosion—from diagnosis to therapy. Bern: Karger; 2006, pp. 9–16.
7. Featherstone JDB, Lussi A. Understanding the chemistry of dental erosion. In: Lussi A, editor. Dental erosion—from diagnosis to therapy. Bern: Karger; 2006, pp. 66–76.
8. Attin T, Koidl U, Buchalla W, et al. Correlation of microhardness and wear in differently eroded bovine dental enamel. Arch Oral Biol 1997;42(3):243–50.
9. Cardoso AC, Canabarro S, Myers SL. Dental erosion: diagnostic-based noninvasive treatment. Pract Periodontics Aesthet Dent 2000;12(2):223–8.
10. Luo Y, Zeng XJ, Du MQ, Bedi R. The prevalence of dental erosion in preschool children in China. J Dent 2005;33(2):115–21.
11. Calvadini C, Siega-Riz AM, Popkin BM. US adolescent food intake trends from 1965 to 1996. Arch Dis Child 2000;83(1):18–24.
12. Gleason P, Suitor C. Children's diet in the mid 1990s: dietary intake and its relationship with school meal participation. US Department of Agriculture, Food and Nutrition Service, Office of Analysis, Nutrition and Evaluation 2001.
13. Lussi A, Shaffner M, Hotz P, et al. Dental erosion in a population of Swiss adults. Community Dent Oral Epidemiol 1991;19(5):286–90.
14. Al-Malik MI, Holt RD, Bedi R. The relationship between erosion, caries, rampant caries and dietary habits in preschool children in Saudi Arabia. Int J Pediatr Dent 2001;11(6):430–9.
15. Pontefract H, Hughes J, Kemp K, et al. The erosive effects of some mouthrinses on enamel. A study in situ. J Clin Periodontol 2001;28(4):319–24.
16. Amin WM, Al-Omoush SA, Hattab FN. Oral health status of workers exposed to acid fumes in phosphate and battery industries in Jordan. Int Dent J 2001;51(3):169–74.
17. Richards JR, Brofeldt BT. Patterns of tooth wear associated with methamphetamine use. J Periodontol 2000;7(8):1371–4.
18. Milosevic A, Agrawal N, Redfearn P, et al. The occurrence of toothwear in users of ecstasy (3,4-methylenedioxymethamphetamine). Community Dent Oral Epidemiol 1999;27(4):283–7.
19. Hara AT, Lussi A, Zero DT. Biological factors. In: Lussi A, editor. Dental erosion—from diagnosis to therapy. Bern: Karger; 2006, pp. 88–99.
20. Vanuspong W, Eisenburger M, Addy M. Cervical tooth wear and sensitivity: erosion, softening and hardening of

 DOI 10.1111/j.1708-8240.2011.00451.x

dentine: effects of pH, time and ultrasonication. J Clin Periodontol 2002;29(4):351–7.
21. Hara AT, Ando M, Cury JA, et al. Influence of the organic matrix on root dentin erosion by citric acid. Caries Res 2005;39(2):134–8.
22. Meurman JH, Frank RM. Scanning electron microscopic study of the effect of salivary pellicle on enamel erosion. Caries Res 1991;25(1):1–6.
23. Featherstone JDB, Rodgers BE. Effects of acetic, lactic and other organic acids on the formation of artificial carious lesions. Caries Res 1981;15(3):377–85.
24. Lussi A, Hellwig E. Erosive potential of oral care products. Caries Res 2001;35(Suppl 1):52–6.
25. Mandel ID. The functions of saliva. J Dent Res 1987;66:623–7.
26. West NX, Maxwell A, Hughes JA, et al. A method to measure clinical erosion: the effect of orange juice consumption on erosion of enamel. J Dent 1998;26(4):329–35.
27. Jarvinen VK, Rytomaa II, Heinonen OP. Risk factors in dental erosion. J Dent Res 1991;70(6):942–7.
28. Meurman JH, Toskala J, Nuutinen P, et al. Oral and dental manifestations in gastroesophageal reflux disease. Oral Surg Oral Med Oral Pathol 1994;78(5):583–9.
29. Bartlett DW, Coward PY, Nikkah C, et al. The prevalence of tooth wear in a cluster sample of adolescent schoolchildren and its relationship with potential explanatory factors. Br Dent J 1998;184(3):125–9.
30. Rytomaa I, Jarvinen V, Kanerva R, et al. Bulimia and tooth erosion. Acta Odontol Scand 1998;56(1):36–40.
31. Hannig C, Hannig M, Attin T. Enzymes in the acquired enamel pellicle. Eur J Oral Sci 2005;113(1):2–13.
32. Feagin F, Koulourides T, Pigman W. The characterization of enamel surface demineralization, and associated hardness changes in human and bovine material. Arch Oral Biol 1969;14(12):1407–17.
33. Zero DT, Fu J, Scott-Anne K, et al. Evaluation of fluoride dentifrices using a short-term intraoral remineralization model [abstract 1363]. J Dent Res 1994;73:272.
34. Lussi A, Kohler N, Zero D, et al. A comparison of the erosive potential of different beverages in primary and permanent teeth using an in vitro model. Eur J Oral Sci 2000;108(2):110–4.
35. Jones SG, Nunn JH. The dental health of 3-year-old children in east Cumbria. Community Dent Health 1995;12(3):161–6.
36. Millward A, Shaw L, Smith AJ, et al. The distribution and severity of tooth wear and the relationship between erosion and dietary constituents in a group of children. Int J Paediatr Dent 1994;4(3):151–7.
37. Hede B. Determinants of oral health in a group of Danish alcoholics. Eur J Oral Sci 1996;104(4 Pt 1):403–8.
38. Zero DT, Lussi A. Behavioral factors. In: Lussi A, editor. Dental erosion—from diagnosis to therapy. Bern: Karger; 2006, pp. 100–5.
39. Gandara BK, Truelove EL. Diagnosis and management of dental erosion. J Contemp Dent Pract 1999;1(1):16–23.
40. Ganss C, Klimec J, Borkowski N. Characteristics of tooth wear in relation to different nutritional patterns including contemporary and medieval subjects. Eur J Oral Sci 2002;110(1):54–60.
41. West NX, Hughes JA, Addy M. Erosion of dentin and enamel in vitro by dietary acids: the effect of temperature, acid character, concentration and exposure time. J Oral Rehabil 2000;27(10):875–80.
42. Lussi A, Jaeggi T. Chemical factors. In: Lussi A, editor. Dental erosion—from diagnosis to therapy. Bern: Karger; 2006, pp. 77–87.
43. Grenby TH. Lessening dental erosive potential by product modification. Eur J Oral Sci 1996;104(2 Pt 2):221–8.
44. Yip KHK, Smales RJ, Kaidonis JÁ. The diagnosis and control of extrinsic acid erosion of tooth substance. Gen Dent 2003;51(4):350–3.
45. Bardsley PF, Taylor S, Milosevic A. Epidemiological studies of tooth wear and dental erosion in 14-year-old children in North West England. Part 1: the relationship with water fluoridation and social deprivation. Br Dent J 2004;197(7):413–6.
46. Conrad HJ, Seong WJ, Pesun IJ. Current ceramic materials and systems with clinical recommendations: a systematic review. J Prosthet Dent 2007;98(5):389–404.
47. Della Bona A, Kelly JR. The clinical success of all-ceramic restorations. J Am Dent Assoc 2008;139(Suppl):8S–13S.

Reprint requests: Júnio S. Almeida e Silva, DDS, MSC, Goethestrasse 70 Apt. 314, LMU Dental School, 80336 Munich, Germany; Tel.: 0049 89 51609439/15259723581; email: juniosantos1710@yahoo.com.br or Junio.Silva@med.uni-muenchen.de

This article is accompanied by commentary, Dental Erosion: Understanding This Pervasive Condition, Terry Donovan, DDS
DOI 10.1111/j.1708-8240.2011.00452.x

 DOI 10.1111/j.1708-8240.2011.00451.x