



Dental erosion in children: A literature review

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Abstract

Epidemiological studies have shown that the prevalence of dental erosion in children varies widely between 2 and 57%. Changes seen in dental erosion range from removal of surface characteristics to extensive loss of tooth tissue with pulp exposure and abscess formation. Symptoms of dental erosion range from sensitivity to severe pain associated with pulp exposure. The etiology of dental erosion is dependent on the presence of extrinsic or intrinsic acid in the oral environment. Extrinsic sources of acids in children include frequent consumption of acidic foods and drinks, and acidic medications. Regurgitation of gastric contents into the mouth, as occurs in gastroesophageal reflux, is the most common source of intrinsic acid in children. A multitude of factors may modify the erosion process, such as saliva, oral hygiene practices, and presence or absence of fluoride. When dental erosion is diagnosed, it is important to investigate and identify the acid source, and to determine if the process is ongoing. The aim of treatment is to eliminate the cause of acid exposure, and to minimize the effects of acid exposure where it is not possible to remove the acid source. Restoration of the dentition involves stainless steel crowns to restore lost vertical dimension, and composite resin for esthetics. (Pediatr Dent 23:37-43, 2001)

Erosion is a chemical dissolution of the dental hard tissues by intrinsic or extrinsic acids.¹ In recent years, dental erosion is increasingly recognized as an important cause of tooth structure loss, not only in adults, but also in children and adolescents.²⁻¹² Dental erosion may cause tooth sensitivity and altered occlusion, and in severe cases, may result in pulp exposure and abscesses. However, the pathogenetic mechanisms, diagnostic criteria, and preventive strategies of the condition are still not well established. The aims of this paper are to review the prevalence, clinical manifestations, and etiology

of dental erosion in children, and to provide guidelines on the preventive and restorative options for this condition.

Prevalence of erosion

Wide-ranging prevalences have been reported in both primary and permanent dentitions (Table 1). The reasons for the wide range of prevalence may be related to the relatively small number of subjects in the majority of studies and the use of different criteria for diagnosis.³ However, when erosion into dentin is considered, studies show prevalences of around 30% in primary molars of 5-year-olds, and 2% in incisal surfaces of permanent incisors in 14-year-olds. For example, in the primary dentition, the UK Child Dental Health Survey showed that the prevalence of erosion on palatal surfaces of the primary teeth was 8% in 2-year-olds and 52% in 5-year-olds, with the proportion of children exhibiting erosion extending into dentin being 24% in 5-year-olds.⁴ A study by Millward et al.⁷ in children 4-16 years of age, found dentin exposure in 30% of primary molars.

In the permanent dentition the prevalence of erosion on palatal surfaces was 8% in 7-year-olds and rose to 31% in 14-year-old children.⁴ Children exhibiting erosion extending into dentin was 2% in 15-year-olds.⁴ In other studies, Bartlett et al.⁵ found enamel erosion in 57% of 11 to 14-year-olds, and dentin erosion in 2%. Milosevic et al.⁶ in a random sample of 14-year-old children found 30% had exposed dentin incisally, and 8% had exposed dentin on occlusal and/or lingual surfaces. In the primary dentition, it is thought that the reduced thickness of enamel and greater acid solubility contributes to the higher susceptibility to erosion.^{3,12}

Children with cerebral palsy are thought to have an increased prevalence of tooth wear, which has been attributed to

Table 1. Prevalence of Dental Erosion in Children

Authors/ year	Number of subjects	Mean Age of subjects (yrs)	Diagnostic criteria	Prevalence of erosion
Millward et al. 1994 ⁷	101	10	Dentin exposure	30% of primary molars
Milosevic et al. 1993 ⁶	1035	14	Dentin exposure	30% incisal 8% occlusal or lingual
Bartlett et al. 1998 ⁵	210	12	Dentin exposure	2% on incisal and palatal of permanent incisors
O'Brien 1994 ⁴	2000	5 - 15	Dentin erosion	24% primary teeth 2% permanent teeth

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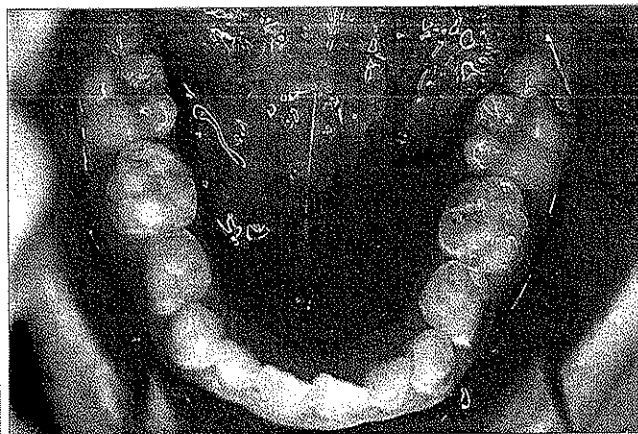


Fig 1A, 1B. Maxillary and mandibular teeth of boy aged 8 years who had a history of gastroesophageal reflux due to incompetent lower esophageal sphincter. Erosion grade 3 as described by Aine et al.¹¹ is clearly seen on the primary teeth. Note also the more severe erosion on the palatal/lingual surfaces as distinct from the erosion seen on labial surfaces in Figure 2, where erosion was attributed to consumption of acidic drinks.

both oral parafunctional activity, and softening of the enamel from gastroesophageal reflux.⁸

Clinical manifestations of erosions

Appearance and distribution of erosion lesions

It is now thought that both lingual and buccal surfaces may be affected in erosion lesions resulting from both intrinsic and extrinsic sources of acid.¹³ Anatomical factors related to movements of the tongue, lips, and cheek may affect the distribution of the erosion lesions.¹³ In addition, salivary factors such as pellicle formation appear to affect development of erosion.¹⁴

Physiologic tooth wear is normally contributed by a combination of abrasion, attrition, and erosion. However in any one individual, each may be seen in differing proportions, complicating diagnosis. Furthermore, enamel softened by erosion is likely to be more susceptible to abrasion and attrition.

Erosion usually manifests as concave loss of tooth surface. In contrast, tooth attrition or physiological wearing away of dental hard tissue as a result of tooth-to-tooth contact, causes incisal or occlusal surface loss of tooth substance, resulting in the formation of facets.^{9,10} In addition, erosion lesions may be distinguished from attrition where defects on opposing teeth cannot be brought into occlusal contact, demonstrating the characteristic "cupping" of erosion.¹⁰

Erosion may also be distinguished from abrasion, which is the pathologic wearing away of tooth substance by an abnormal mechanical process independent of chewing.⁹ Abrasion is more likely to affect buccal or cervical surfaces of teeth, and is often caused by toothbrushing.¹⁵ In contrast, abfraction describes a wedge-shaped defect with a sharp outline at the cemento-enamel junction (CEJ) and is thought to be due to occlusal forces causing microfractures of enamel and dentin at the CEJ.¹⁶ The role of abfraction in tooth wear and erosion is still unclear.⁹

Initially, erosion may be manifested by a slight loss of surface luster only detectable when the enamel is cleaned and dry.¹⁰ Sensitivity or fracturing of thinned incisal edges may be the first signs of erosion because of its insidious nature.¹⁰ The erosion progresses until the more yellow underlying dentin becomes visible through the thinned overlying enamel.¹⁰ These lesions have a dished out, hard, smooth appearance.

Several different classifications have been used in the literature to describe dental erosion. The number of different indices proposed to date suggests that an index fulfilling all relevant criteria has not been found.³ In addition, most have been designed to measure tooth surface loss in adults, and are not always suitable for children.

The Smith and Knight Tooth Wear Index¹⁷ is often used in adults, but it scores all types of wear: attrition, abrasion, and erosion. Each tooth is scored by examining four surfaces: cervical, remainder of buccal or labial surface, lingual or palatal surface, and the occlusal or incisal surface, and scoring the severity of tooth loss on each surface from grades 0 (no erosion) to grade 4 (severe erosion with pulp exposure). A modified, more detailed version of this index is used by O'Sullivan et al.¹⁸ in which erosion in children with gastroesophageal reflux was measured. In this index, site, severity, and area affected are scored for each tooth, using severity scores code 0 (no erosion) to code 5 (severe erosion with pulp exposure). This index is useful in that site, severity, and surface area of erosion is scored on each tooth. On the other hand, the amount of detail is lengthy to record in this index.

A simpler and more practical classification of dental erosion in children with gastroesophageal disease has been proposed by Aine et al.¹¹ Each tooth is scored, ranging from grade 0 where there is no erosion, to grade 3 where there is exposure of dentin at the bottom of holes in the occlusal surface. This classification is specific for children with gastroesophageal reflux and is suitable for scoring primary, mixed, and permanent dentitions. It was found that most children with pathologic gastroesophageal reflux (GER) exhibited dental erosions of the same type but with varying severity. The disadvantage of this index is that it does not specify site of erosion on each tooth or the extent of surface area involved. None of the indices give any indication of the restorative needs of teeth affected, which may be important for clinicians treating these patients.

Severe dental erosion lesions in children may be seen in Figures 1, 2, and 3. These are the result of GER, and excessive frequent consumption of acidic drinks.

Dental complications of erosion

Dental erosion may cause a number of clinical problems including esthetics. Severe erosion usually results in enamel

fracture, which progresses to shortening of the teeth and loss of occlusal vertical dimension.^{10, 12}

Dentin sensitivity and difficulty in eating are common problems of dental erosion, particularly if erosion is rapid and progressive. Rapid loss of tooth structure from dental erosion in children with immature teeth and large pulps are likely to lead to pulpal inflammation and exposures.^{10, 12}

Etiology of dental erosion

The underlying etiology of dental erosion is a source of acid, which may be intrinsic or extrinsic, acting on a susceptible tooth. In addition, there are many modifying factors affecting the host which significantly affect tooth susceptibility to dental erosion. Parafunctional habits may also contribute to tooth wear in teeth that have been softened by acid demineralization.¹⁹

Extrinsic sources of acids

Extrinsic causes of dental erosion may arise from several sources, including those which may be occupational, in medications, or through lifestyle practices. (Table 2) Although not affecting children, occupations involving exposure to acids in the workplace may contribute to dental erosion. Workers in factories in which there are acidic fumes or aerosols involving sulfuric acid, such as in "battery factories, and hydrochloric acid, such as in galvanizing factories, have been shown to have a higher prevalence of erosion.¹⁵ Several other occupations have been implicated in increased tooth surface loss, including professional wine tasters, printers, and workers in munitions factories. Improper monitoring of pH in gas chlorinated swimming pools has been reported to be the cause of dental erosion in competitive swimmers.¹⁵

Dehydration following sporting activities may contribute to erosion when reduced salivary flow causes decreased buffering. This may be exacerbated by consumption of acidic drinks such as sports drinks, fruit, and soft drinks.¹⁵

Chewable Vitamin C preparations may cause erosion when consumed frequently and left in direct contact with the teeth.¹⁵ Aspirin, when chewed daily over extended periods, has been reported to cause erosion in children.¹⁵ Erosion has been associated with the consumption of citrus fruits, low pH carbonated drinks, cider vinegar, and sports drinks.²⁰

Consumption of alcohol, for example wine and when acidic carbonated drinks are used as mixers, may also contribute to erosion.¹⁵

Titrateable acidity of foods and drinks

The pH of the oral cavity affects the solubility of dental tissues. The solubility increases by a factor of seven to eight with each decrease of pH by 1 unit when oral pH decreases from normal (pH 6.5) to acidic.²¹ As the critical pH at which enamel dissolution occurs is 5.5, acidic products with a pH below 4 will result in erosion.²²

In relation to enamel dissolution, the actual H⁺ concentration of acidic dietary substances available to interact with the tooth surface (or titrateable acidity) is more important than actual pH.¹⁵

Modifying effects of other constituents of food and beverages such as calcium, phosphate and fluoride concentration may also be exerted. Factors including the acid type and physical and chemical properties may affect the salivary clearance rate of acids from the mouth.¹⁵

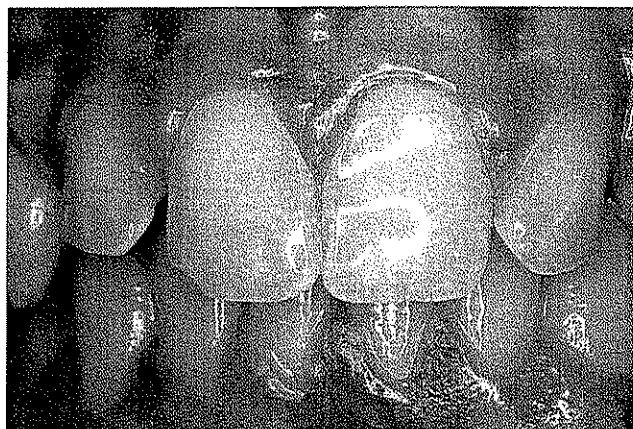


Fig 2. Anterior teeth of a 12 year-old boy who had frequent consumption of acidic drinks. Note the etched appearance of the left central incisor (grade 1 erosion) and the thinning of the incisal edge of the left central incisor (grade 2 erosion).

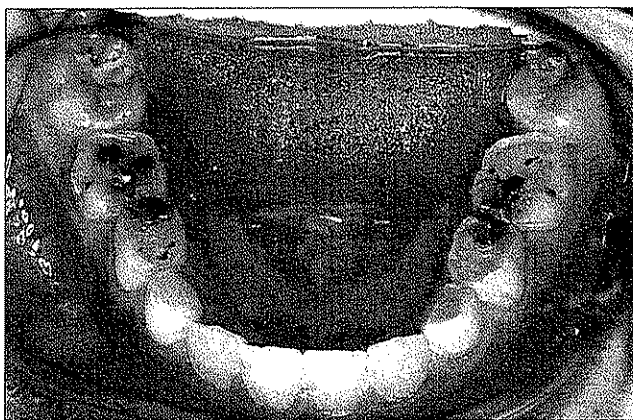


Fig 3. Mandibular teeth of a 9-year-old boy who had frequent consumption of acidic drinks. Note the appearance of the amalgam restorations sitting above the remaining tooth structure, also characteristic of grade 3 erosion as in the classification by Aine et al 1993.¹¹

Erosive capacities of different test substances are significantly associated with their titrateable acidity, pH, phosphate content, and fluoride content.²² Furthermore, a high pattern of consumption of acidic beverages such as cola drinks may present a higher risk of causing erosion than based solely on their chemical properties.¹⁵

Types of acidic foods and drinks associated with erosion

Consumption of acidic foods and beverages has been shown to contribute to dental erosion. In a clinical trial which investigated the effects of acidic beverages on human teeth in a group of dental students, the effect of daily ingestion of different amounts of acidic beverages on macroscopic and microscopic changes in the labial surface of maxillary anterior teeth was examined.²³ The students were divided into groups, drinking either orange juice, grapefruit juice or carbonated cola, and subdivided into groups of five who drank either 6, 12, 18, or 24 ounces of the juice or carbonated beverage per day. A group of 10 students served as controls and refrained from ingesting all forms of citrus fruits and carbonated beverages. The study reported that the first appearance of any microscopic alteration of the enamel surface occurred between the fourth and sixth weeks, and that all experimental groups were found to have some alteration of surface enamel. Orange juice was found to

Table 2. Extrinsic Acid Sources Which Have Been Implicated in Erosion

Workplace/ Occupational	Medication	Diet
Acidic environments such as in fertilizer, battery, munitions, printing, or galvanizing plants	Acidic medications, e.g. chewable vitamin C tablets, aspirin	Frequent consumption of acidic foods, e.g., citrus fruits, pineapple, salad dressing containing vinegar
Swimming in improperly chlorinated pools		Frequent consumption of acidic drinks, e.g., cola, sports drinks, fruit juice
Professional wine tasters		Sucking on citrus fruits
Sporting activities causing dehydration followed by acidic sports drinks		Alcohol, e.g., wine, spirits mixed with acidic soft drinks

cause less erosion than grapefruit juice or carbonated cola beverage. However, even within the high consumption groups, some students did not experience any detectable erosion, suggesting that there may be biological modifying factors present.²³

Sports drinks have also been evaluated in other studies. Citric acid is frequently included in sports drinks for its refreshing taste, but has been found to be highly erosive. The demineralizing effect of citric acid is exceptionally great because its chelating effect on enamel calcium continues even after the pH rises.²⁰

Frequency of consumption

Jarvinen et al²⁰ found a strong association of dental erosion in patients who consumed citrus fruits more than twice a day, soft drinks daily, and apple vinegar or sports drinks once a week or more.

Other studies have also shown an increase in the mean frequency of consumption of fruit drinks, carbonated beverages and fruit juices were each associated with an increase in the severity of erosion. Of note was the finding that bedtime consumption of fruit juices was strongly associated with the most severe cases of erosion,⁷ suggesting that the erosive potential of fruit juices was probably the highest when salivary flow is the lowest.

Intrinsic sources of acids

The propulsion of gastric contents into the mouth, such as in gastroesophageal reflux,²⁴ is the most common source of intrinsic acids in the mouth. Since the acidity of the stomach may be below pH 1, dental erosion has been observed in disorders associated with chronic vomiting, persistent regurgitation or gastroesophageal reflux, or with protracted rumination. Conditions in which propulsion of gastric contents occur include disorders of the upper gastrointestinal tract, specific metabolic and endocrine disorders, medication side effects, and drug abuse, as well as psychosomatic disorders such as stress induced psychogenic vomiting, anorexia, bulimia nervosa, and rumination.²⁵ In addition, psychological stress may produce changes in esophageal contractions and lowering of lower esophageal sphincter pressure.²⁶

Gastric dysfunction is one of the principal risk factors associated with dental erosion. Patients reporting symptoms such as vomiting once or more per week, experiencing acid tastes, belching, heartburn, stomach-ache, or pain on awakening, have 31 times higher incidence of dental erosion when compared to controls.²⁰ However, although the acidity of the gastric con-

tents is high, it has been reported that erosion is not seen clinically until gastric acid has acted on the teeth regularly several times a week for a period of at least 1-2 years.²⁵

The prevalence of dental erosion in patients with reflux has been reported by several authors. Meurman et al²⁷ examined 117 patients with reflux disease and reported that 28 patients (24%) had dental erosion. In these studies it was also found that the number of patients with low salivary buffering capacity was higher

among those with erosion than those without. Bartlett et al.²⁸ examined 36 patients who were investigated because of their palatal dental erosion and found that 23 (64%) had gastroesophageal reflux. They concluded that patients presenting with palatal dental erosion should be investigated for gastroesophageal reflux, even in the absence of clinical symptoms of reflux.

In children, the literature is limited on the role of gastroesophageal reflux in erosion. Taylor et al² reported an 8-year-old female with extensive loss of enamel on all surfaces of her remaining primary teeth, who on investigation, was found to have asymptomatic gastroesophageal reflux.

Aine et al.¹¹ found erosive lesions in 15 of 17 children aged 22 months to 16 years with pathological GER. Seven children out of 15 had dentin exposure. It was concluded that loss of dental hard tissue is an important sign of pathological GER and that dentists become capable in screening and identifying clinically important silent GER.¹¹

O'Sullivan et al¹⁸ examined children attending hospital clinics with symptoms of GER whose reflux index was 10% (e.g., moderate to severe) or more. Evidence of erosion was seen in 17% of children with only one child having erosion that involved dentin. In all children with erosion, only the primary dentition was affected: generally the palatal surfaces of the maxillary primary incisors.

Factors modifying the erosion process

Individual responses may also influence the extent and distribution of erosion lesions. These include the manner in which the erosive fluid is taken into the mouth, the tooth surfaces that come into contact with the fluid, and the duration of contact with the teeth. This, in turn, is influenced by swallowing habits, motions of the lips and cheeks, and access to saliva. Other host factors are also considered to modify the erosion process, such as the buffering capacity of the saliva, the chemical and physical properties of enamel, and the shape and contour of the teeth.²³

Saliva

Of the factors modifying the erosion process, saliva is probably the most important as it is known to have protective properties against dental erosion,¹⁵ but the nature of this role is not fully established.¹⁴

A number of previous studies have examined various salivary parameters and their relationship to dental erosion. Saliva forms the pellicle that protects enamel from acid demineralization.¹⁴ The thickness of pellicle varies within different areas

of the mouth and this may influence the sites and severity of erosion.²⁹ Unstimulated salivary flow rate has been directly associated with dental erosion.^{20,24,30} A direct relationship has been demonstrated between reduced salivary flow rates and oral clearance of dietary acids and buffering capacity.³⁰ The bicarbonate level of saliva is related to the flow rate; hence lower buffering capacity is seen in saliva produced at a low flow rate.³⁰ Salivary buffer capacity has also been found to be significantly lower in patients with erosion than controls.^{31,32}

Decreased salivary flow due to dehydration may also contribute to reduced protection by saliva from intrinsic and extrinsic acids.¹⁹

Oral hygiene practices

Dental erosion is frequently seen in individuals with a high level of oral hygiene.³ The reason for this may be that removal of pellicle by abrasive containing toothpastes removes the protection of tooth structure provided by pellicle to acids.²⁹ Initially, the demineralization occurring after acid consumption is reversible, and may be remineralized by salivary minerals.³³ However, enamel and dentin initially demineralized by acid may be easily removed by toothbrushing in a process of abrasion, which accelerates the erosion. Hence, the practice of toothbrushing immediately after consuming acidic beverages may increase tooth loss.¹⁵ The alternative of brushing before meals is also likely to have similar effects in that the removal of salivary pellicle will render the enamel surface more susceptible to acid attack during the meal.³³

Fluoride

It has been shown that the addition of fluoride to acid solutions decreases the amount of erosion in animals.³⁴ Furthermore, softening of enamel by cola beverage in vitro may be inhibited by high concentration fluoride varnish,³⁵ and less tooth wear occurs when a fluoride dentrifice is used than when a non-fluoride dentrifice is used in vitro.³⁶ Fluoride exposure from water fluoridation and in supplement form in the first 12 years of life appears to confer some resistance to excessive tooth wear from acid erosion in adulthood, as it does for resistance to demineralization by dental caries.³⁷ Application of 2000 ppm sodium fluoride solutions immediately before toothbrushing significantly reduces abrasion of eroded dentin in vitro.³⁸

Guidelines for the management of erosion

Diagnosis of erosion and risk factors

A thorough medical history is important to identify any causative medical conditions.³⁹ This should be followed up by the pediatric dentist with a diet history of several days to elicit possible dietary causative factors.³⁹ Patients who have erosion of the palatal surfaces of maxillary teeth should be investigated for a history of reflux symptoms. If periodic symptoms of heartburn, epigastric pain, or regurgitation are experienced, with or without recurrent hoarseness and laryngitis, early referral to a gastroenterologist is desirable.²⁸

While there is little in the literature about dental health of children with gastroesophageal reflux, it is reasonable to suppose that the acidic environment may predispose these patients to greater susceptibility to dental caries as well as erosion. All patients with gastroesophageal reflux should be referred for

dental assessment and preventive advice to minimize caries and erosion and for treatment of existing lesions. In addition, tooth erosion may be the first clinical sign of gastroesophageal reflux in asymptomatic cases, and investigation resulting from the observation of erosion may lead to medical diagnosis of gastroesophageal reflux in previously undetected cases. Hence the pediatric dentist may play an important role in the overall management of these patients.

Monitoring

Indications: Monitoring is important when erosion has been diagnosed to determine whether the process is ongoing or has ceased as a result of medical treatment or management of dietary acid intake.

Objectives: To determine whether medical intervention or dietary change is effective in removing the acid source.

Long term monitoring of the rate of erosion to determine if the process is ongoing is important. Study models and photographs may be taken for this purpose.³⁹ Comparison with clinical photographs and study models is helpful in determining erosion progression. In addition, a silicone rubber index fabricated over the study models in the areas demonstrating erosion can be used to ascertain whether the erosion has progressed. The index is cut with a sharp scalpel through the area of interest and placed in the patient's mouth over the erosive lesion to determine whether there is a gap indicating severe ongoing erosion. Small increments in erosion will probably not be visualized with this technique, however the more accurate method of examination of replica models using scanning electron microscopy is impractical in practice.³

Often physicians look to dentists for evidence that medical therapy for gastroesophageal reflux, such as H₂ antagonist medication, is efficacious so that they can avoid repeating invasive 24 hour pH probes and endoscopies. However, because erosion occurs slowly over a long period of time, it would be impractical to rely on measurement of ongoing erosion as a clinical indicator of successful medical therapy. In this regard in the short term, a simple clinical guide for evidence that erosion, and hence gastroesophageal reflux, has ceased may be the absence of tooth sensitivity.⁴⁰

In the long term, erosion should be followed up at each six monthly review and more often if symptoms do not subside.

Dietary modification

Indications: Dietary advice to decrease the intake of extrinsic acids is necessary when acidic intake is found to be excessive and is a likely etiology in the erosion exhibited by the patient.

Objectives: To decrease total acidic intake or at least limit these to meal times to allow remineralization to occur, resulting in the reduction or elimination of the erosion process.

If the acid found to be causing the erosion is dietary in origin, advice must be centered on decreasing the consumption of acidic foods and drinks, and confining the intake of acidic foods and drinks to meal times, and refraining from drinking or eating juice or fruit before bedtime.³⁹ In addition, acidic drinks should be swallowed immediately and not "swished" around the mouth.

Finishing a meal with something neutral or alkaline may be beneficial. Eating foods with a high content of calcium, phosphate such as milk, cheese, lipids, or buffering substances may also reduce erosive potential of acids.³⁹ In addition, patients

should be instructed to avoid brushing immediately after consuming acidic food or drink, as this is likely to accelerate abrasion.¹⁵

Dietary monitoring for acid intake should be performed by the dentist, who can correlate the dietary findings with the dental results. Dietary monitoring should be done monthly, commencing at the beginning of treatment, but the intervals may be increased when patient compliance to an altered diet is obtained.

Fluoride

Indications: Where there is established erosion, fluoride may be used to minimize hard tissue loss and control sensitivity.

Objectives: to reduce acid solubility of enamel and dentin and favor remineralization of acid softened tooth structure. Use of fluoride will also aid in reduction of sensitivity.

A daily neutral sodium fluoride mouthrinse or gel to combat enamel softening by acids and control pulpal sensitivity may be prescribed.³⁶ Sugar free chewing gum to stimulate saliva flow may also be advised.³⁹ A twice daily 0.05 % neutral fluoride mouthrinse or twice weekly, a concentrated (1.23%) neutral fluoride gel may be prescribed.³⁹

Restorative treatment

Indications: Restorative treatment is necessary to restore lost tooth structure subsequent to loss by erosion for both function and esthetics.

Objectives: Primary teeth should be restored to maintain them until exfoliation. The restoration should provide pulpal protection to maintain vitality and minimize sensitivity. In permanent teeth, function and esthetics should be restored and vitality maintained.

Although it has been suggested that restorative treatment is unwise while erosion is ongoing, this approach is probably unrealistic¹² because many patients, particularly children and adolescents, do not comply with dietary advice. Restorative treatment is usually necessary for several reasons. First, tooth coverage is necessary to alleviate pain in exposed hypersensitive dentin. Second, restorations may be necessary to improve esthetics, and restore occlusal vertical dimension in heavily eroded dentitions. Third, prevention of further loss of tooth structure may be achieved through protection provided by restorations.

For posterior primary molars, placement of extra-coronal stainless steel crowns is frequently the only way of providing relief of symptoms and protection from further wear and to maintain the tooth until it is due to exfoliate.¹²

Enamel loss on anterior teeth may best be restored with composite resin. The material offers excellent aesthetics, and there have been no reports suggesting that bonding to eroded enamel is affected. On the palatal aspect, however, resins are difficult to apply and easily fractured. It may be necessary to restore the incisal aspect with composite resin and place a more durable restoration on the palatal aspect. Materials suitable for this purpose are yellow gold and nickel-chromium alloys, fabricated as veneers and cemented to the palatal surfaces of worn incisors.¹² Using conservative adhesive techniques, surface active composite luting agents are used in conjunction with sand-blasted nickel-chromium alloy. A similar technique may be used for posterior teeth where nickel-chrome onlays are cemented to restore the occlusal surfaces of eroded molar teeth.

In both anterior and posterior teeth, no tooth preparation is required, conserving the maximum amount of tooth structure.¹²

Future studies

More research should be initiated to identify children at risk to erosion so that effective preventive strategies can be instituted. Research into the relative erosive potential of common drinks and foods would enlighten the public as to what foods may damage the teeth. In addition, future research into the roles of saliva and medical conditions in the pathogenesis of the erosion lesions may help further the understanding of this complex condition.

Conclusions

1. Dental erosion may be caused by exposure of the teeth to frequent consumption of acidic drinks or foods, environmental exposure to acids, or by reflux of gastric acid into the mouth.
2. Clinical manifestations of dental erosion are loss of enamel and dentin, with consequent dental sensitivity, loss of occlusal vertical dimension, and poor esthetics.
3. Children presenting with dental erosion should undergo a thorough evaluation to identify the source of the acids causing the erosion. In the case of gastroesophageal reflux, medications may be necessary. In addition, the erosive potential of acid may be decreased by dietary alterations, fluoride supplementation, and restorative care.

References

1. Pindborg J, Pathology of dental hard tissues. 1970: Copenhagen: Munksgaard, 312.
2. Taylor G, Taylor S, Abrahams R, Mueller W: Dental erosion associated with asymptomatic gastroesophageal reflux. *J Dent Child* 3:182-85, 1992.
3. Shaw L, Smith A: Dental erosion—the problem and some practical solutions. *Brit Dent J* 186:115-18, 1998.
4. O'Brien M: Children's dental health in the UK 1993. 1994: Office of population censuses and surveys. London: HMSO.
5. Bartlett D, Coward P, Nikkah C, Wilson R: The prevalence of tooth wear in a cluster sample of adolescent schoolchildren and its relationship with potential explanatory factors. *Brit Dent J* 184:125-29, 1998.
6. Milosevic A, Young P, Lennon M: The prevalence of tooth wear in 14-year-old school children in Liverpool. *Comm Dent Health* 11:83-86, 1993.
7. Millward A, Shaw L, Smith A, Rippin J, *et al.*: The distribution and severity of tooth wear and the relationship between erosion and dietary constituents in a group of children. *Int J Paediatric Dent* 4:152-57, 1994.
8. Shaw L, Weatherill S, Smith A: Tooth wear in children: An investigation of etiological factors in children with cerebral palsy and gastroesophageal reflux. *J Dent Child* 65:484-86, 1998.
9. Imfeld T: Dental erosion. Definition, classification and links. *Eur J Oral Sci* 104: 151-55, 1996.
10. Lazarchik D, Filler S: Effects of Gastroesophageal reflux on the oral cavity. *Am J Med* 103:107S-13S., 1997.
11. Aine L, Baer M, Maki M: Dental erosions caused by gastroesophageal reflux disease in children. *J Dent Child* 60:210-14, 1993.
12. Harley K: Tooth wear in the child and the youth. *Br Dent J* 186:492-96, 1999.

13. Jarvinen V, Rytomaa I, Meurman J: Location of dental erosion in a referred population. *Caries Res* 26:391-96, 1992.
14. Hannig M, Balz M: Influence of in vivo formed salivary pellicle on enamel erosion. *Caries Res* 33:372-79, 1999.
15. Zero D: Etiology of dental erosion- extrinsic factors. *Eur J Oral Sci* 104:162-77, 1996.
16. Grippo J, Simring M: Dental "erosion" revisited. *JADA* 126:619-28, 1995.
17. Smith B, Knight J: An index for measuring the wear of teeth. *Brit Dent J* 156:435-38, 1984.
18. O'Sullivan E, Curzon M, Roberts G, Milla P, *et al.*: Gastroesophageal reflux in children and its relationship to erosion of primary and permanent teeth. *Eur J Oral Sci* 106:765-69, 1998.
19. Khan F, Young W, Daley T: Dental erosion and bruxism. A tooth wear analysis from South East Queensland. *Aust Dent J* 43:117-27, 1998.
20. Jarvinen V, Rytomaa I, Heinonen O: Risk factors in dental erosion. *J Dent Res* 70:942-47, 1991.
21. Gregory-Head B, Curtis D: Erosion caused by gastroesophageal reflux: diagnostic considerations. *J Prosthodont* 6:278-85, 1997.
22. Rytomaa I, Meurman J, Koskinen J: In vitro erosion of bovine enamel caused by acidic drinks and other foodstuffs. *Scand-J-Dent-Res*. 96:324-33, 1988.
23. Thomas A: Further observations on the influence of citrus fruit juices on human teeth. *NYS Dent J* 23:424-30, 1957.
24. Jarvinen V, Meurmann J, Hyvarinen H, Rytomaa I: Dental erosion and upper intestinal disorders. *Oral Surg Oral Med Oral Pathol* 65:298-303, 1988.
25. Scheutzel P: Etiology of dental erosion- intrinsic factors. *Eur J Oral Sci* 104:178-90, 1996.
26. Mittal R, Stewart W, Ramahi M, *et al.*: The effects of psychological stress on the esophagogastric junction pressure and swallow-induced relaxation. *Gastroenterology* 106:1477-84, 1994.
27. Meurman J, Toskala J, Nuutinen M, Klemetti E: Oral and dental manifestations in gastroesophageal reflux disease. *Oral Surg, Oral Med, Oral Pathol* 78:583-89, 1994.
28. Bartlett D, Evans D, Smith B: The relationship between gastroesophageal reflux disease and dental erosion. *J Oral Rehabil* 23:289-97, 1996.
29. Amaechi B, Higham S, Edgar W, Milosevic A: Thickness of acquired salivary pellicle as a determinant of the sites of dental erosion. *J Dent Res* 78:1821-28, 1999.
30. Woltgens J, Vingerling P, De Blick-Hogervors J, Bervoets D: Enamel erosion and saliva. *Clin Prev Dent* 7:8-10, 1985.
31. Gudmundsson K, Kristleifsson G, Theodors A, Holbrook W: Tooth erosion, gastroesophageal reflux and salivary buffer capacity. *Oral Surg Oral Med Oral Pathol Oral Radiol Endodont* 79:185-89, 1995.
32. O'Sullivan E, Curzon M: Salivary factors affecting dental erosion in children. *Caries Res* 34:82-87, 2000.
33. Kuroiwa M, Kodaka T, Kuroiwa M, Abe M: Brushing induced effects with and without a non-fluoride abrasive dentifrice on remineralization of enamel surfaces etched with phosphoric acid. *Caries Res* 28:309-14, 1994.
34. Sovari R: Effects of various sport drink modifications on dental caries and erosion in rats with controlled eating and drinking pattern. *Proc Finn Dent Soc* 85:13-20, 1989.
35. Sovari R, Meurman J, Alakuijala P, Frank R: Effect of fluoride varnish and solution on enamel erosion in vitro. *Caries Res* 28:227-32, 1994.
36. Bartlett D, Smith B, Wilson R: Comparison of the effect of fluoride and non-fluoride toothpaste on tooth wear in vitro and the influence of enamel fluoride concentration and hardness of enamel. *Br Dent J* 176:346-48, 1994.
37. Teo C, Young W, Daley T, Sauer H: Prior fluoridation in childhood affects dental caries and tooth wear in a south east Queensland population. *Aust Dent J* 42:92-102, 1997.
38. Artin T, Zirkel C, Hellwig E: Brushing abrasion of eroded dentin after application of sodium fluoride solution. *Caries Res* 32:344-50, 1997.
39. Imfeld T: Prevention of progression of dental erosion by professional and prophylactic measures. *Eur J Oral Sci* 104:215-20, 1996.
40. Wickens J: Prevention and maintenance. *Brit Dent J* 186:371-76, 1999.

ABSTRACT OF THE SCIENTIFIC LITERATURE



FLUOROSIS, FLUORIDATION, SOCIAL DEPRIVATION, TOOTHPASTE USE

The aims of this study were first to compare the prevalence and severity of fluorosis in young children residing in a fluoridated versus a non-fluoridated community and second, to examine the relationship between the occurrence of this fluorosis and the use of fluoridated toothpaste in childhood. Approximately an equal number of children (409 and 403 respectively) from a fluoridated and a fluoride deficient community were examined by a single examiner for the presence of fluorosis of the permanent incisor teeth. In addition, clinical photographs were taken for later examination in order to establish examiner reliability. Parents were also asked to complete a questionnaire on the child's early history of toothbrushing and the use of fluoridated toothpaste. Results showed the prevalence of fluorosis was 54% in the fluoridated community and 23% in the fluoride deficient area. Statistical analysis indicated that the area of residence and type of toothpaste used (adult versus children's) were significantly related to the presence of fluorosis. The authors concluded that fluorosis was more prevalent in fluoridated communities and that the use of a child's toothpaste (containing less ppm of fluoride) may help to decrease the prevalence of fluorosis.

Comments: This article reminds the practitioner that patients are exposed to fluoride from many different sources and we must consider all of them in order to decrease the chance of fluorosis. MM

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25 references