Dental Erosion in Gastroesophageal Reflux Disease

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Abstract

Dentists are often the first health care professionals to diagnose dental erosion in patients with gastroesophageal reflux disease (GERD). Gastroesophageal reflux (GER) is the passage of gastric contents into the esophagus, and GERD is defined as symptoms or complications of GER. Twenty-four-hour monitoring of esophageal pH is helpful in diagnosing GERD. Treatment of dental erosion resulting from GERD involves a multidisciplinary approach among family physician, dentist, prosthodontist, orthodontist and gastroenterologist. When possible, dental erosion should be treated with minimal intervention, and such treatment should include control of microflora, remineralization, adhesive restorations and use of biomimetic materials.

MeSH Key Words: *dental enamel/pathology; gastroesophageal reflux/complications; tooth erosion/etiology*

Dentists are often the first health care professionals to diagnose a systemic disease through observation of its oral manifestations. One such condition is gastroesophageal reflux disease (GERD), which may be evidenced by dental erosion. Dental erosion is defined as the progressive loss of hard dental tissues caused by a chemical process not involving bacterial action.[1] It has been associated with ingestion of acidic foods,[2] bulimia,[3] rumination and GERD.[4] In addition to causing dental erosion, undiagnosed and untreated GERD may also result in esophagitis, Barrett's epithelium, esophageal adenocarcinoma and aspiration pneumonitis of various degrees. It is therefore important that dentists recognize GERD so that timely preventive and treatment measures can be instituted. This paper discusses the relationship between dental erosion and GERD, the prevalence and causes of these conditions, diagnostic approaches and treatments.

Dental Erosion

Prevalence

In a 5-year longitudinal study,[5] 71% of children had erosive lesions of at least grade 1 affecting their primary dentition, and 26% had grade 2 erosions (**Table 1**). By 16 years of age, 12% had at least one permanent tooth with grade 1 erosion, and up to 0.2% of patients had at least one permanent tooth with grade 2 erosion.[5] Other studies have reported a similar prevalence of erosion in adults (between 5% and 16%).[6,7] It has been our observation, working in the dental department of a tertiary care facility with a catchment area of 10 million people, that many causes of dental erosion go unnoticed or undiagnosed in adolescence, and the problems are not identified until early adulthood, when the damage is much more severe and much more difficult to treat.

Causes

Erosion begins as superficial demineralization of the enamel, which can cause dissolution of the subsurface layers and eventual loss of tooth structure. Any acid with a pH below the critical pH of dental enamel (5.5) can dissolve the hydroxyapatite crystals in enamel. Gastric refluxate has a pH of less than 2.0 and thus has the potential to cause dental erosion.[8] In vitro experimental erosion has been shown to occur at an oral pH of less than 3.7.

Causes of dental erosion are classified as extrinsic or intrinsic. Extrinsic causes include carbonated or acidic

Figure 1: *Dental erosion, facial view of lips and teeth.*

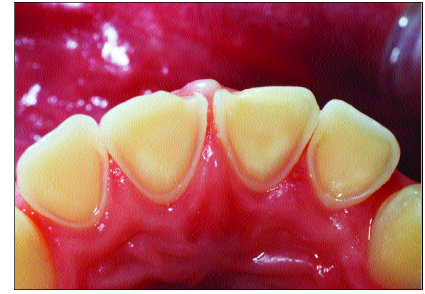**Figure 2:** *Dental erosion, facial view.*

Figure 3: *Dental erosion, palatal view.*

beverages, acidic foods,[2] citric lozenges, various medications, oral hygiene swab sticks, saliva substitutes,[10] recreational exposure to water in gas-chlorinated swimming pools[10] and occupational exposure to corrosive agents such as battery acid fumes and industry aerosols.[8,10]

Intrinsic causes of dental erosion include bulimia, rumination or voluntary reflux phenomenon, subclinical regurgitation due to chronic gastritis associated with alcoholism, xerostomia, malabsorption syndrome, chronic vomiting during pregnancy and GERD.[10–12] Meurman and others[13] examined 117 patients with GERD, of whom 28 (24%) also had dental erosion. Schroeder and others[4] identified dental erosion in 11 (55%) of 20 patients with GERD.

Clinical Presentation

The mandibular molars in both the primary and permanent dentitions are the teeth most commonly subject to erosion.[5] Patients exposed to extrinsic acids suffer more damage to the labial or occlusal surfaces of the upper anterior teeth,[8] with severity decreasing posteriorly, whereas intrinsic acid causes more damage to the lingual surfaces of the teeth. The pattern of erosion caused by intrinsic acid may be modulated by the protective influence of the tongue, which forces regurgitated acid over the tongue, along the palate and into the buccal vestibule.[8] Thinning of the enamel imparts an unesthetic yellowish hue to the teeth (**Figs. 1** and **2**). Eroded teeth have the appearance of having been lightly prepared for full-coverage restorations with a chamfer margin (**Fig. 3**) and are more prone to wear. Once dentin is exposed, the loss of dentin progresses faster than the loss of enamel, such that "cupping" of lesions on the occlusal surfaces occurs.[8] Amalgam restorations in eroded teeth appear highly polished and seem to "stand above" the tooth surface (**Table 2**).

Exposure of the dentinal tubules results in hypersensitivity to hot, cold, sweet and tactile stimuli. The pulp may eventually be exposed, with the attendant need for endodontic therapy.[8–10] Additional sequelae of dental erosion include compensatory eruption of eroded teeth, tipping and drifting of teeth, formation of diastemae, loss of vertical dimension, overclosure and bite collapse, all of which result in autorotation of the mandible and reduction of overjet toward or beyond an edge-to-edge incisal relationship. These sequelae can be exacerbated if attrition from bruxism is superimposed upon erosion, or if either the acidic oral environment or pre-existing or continuing erosion increases susceptibility to caries.

Treatment

The immediate goal in the treatment of dental erosion resulting from GERD is formulation of the correct differential diagnosis and prompt referral to a gastroenterologist. It is not unusual, particularly in medically underserviced regions of the country, to encounter waits of up to 6 months or more to see a medical specialist. In the meantime, it is important to provide symptomatic relief and to discourage further progression of the erosion.

It has been known for many years that demineralized lesions can be remineralized and repaired.[14] It is likely that the same factors controlling remineralization of carious lesions also control remineralization of areas of erosion.

Table 1 Erosion grading scale of Ganss and others[5]

Grade	Description
0	No visible erosion
1	Small pits and slightly rounded cusps, flattened fissures, moderate cupping, preservation of occlusal surface morphology
2	Depression of cusps with severe cupping and grooving, restoration margins raised above level of surrounding tooth, flattening of occlusal surface morphology

Table 2 Erosion grading scale of Eccles and Jenkins[9]

Grade	Description
0	No erosion
1	Loss of surface detail; change confined to enamel
2	Exposure of dentin affecting less than one-third of crown
3	Exposure of dentin affecting one-third or more of crown

Saliva is already supersaturated with calcium and phosphate ions, so if the ambient pH rises above pH 5.5, erosive lesions will begin to remineralize. Successful remineralization requires control of cariogenic microflora through diligent home care and reduction of intake of refined carbohydrates. Daily rinsing with 0.12% chlorhexidine[15] is helpful in reducing accumulation of bacterial plaque, especially during the early phase of patient education and motivation.

To prevent the acidity on the surface of the teeth from falling below pH 5.5, at which point demineralization occurs, the consumption of carbonated and acidic beverages must be curtailed. Saliva can reduce the potential for demineralization, and therefore any deficiency in flow rate or buffering capacity of the saliva should be noted.[16] If necessary, saliva substitutes can be recommended. Chewing antacid tablets or rinsing with a solution of sodium bicarbonate can neutralize the demineralizing effect of acid on the dentition.[8]

Fluoride facilitates remineralization and, because the critical pH of fluoroapatite is 4.5, confers greater resistance to demineralization.[17] Furthermore, fluoride is bacteriostatic[18] and buffers the pH on the surface of the tooth.[19] To maximize the potential for remineralization and minimize the potential for demineralization, daily use of both a neutral 0.05% fluoride mouth rinse and 1.1% fluoride toothpaste is recommended. In addition to the value of mouthguards as a physical barrier to protect the teeth from exposure to acid during periods of reflux, they are also useful carriers for fluoride gel.

Once the diagnosis of GERD has been established and the condition brought under control, some orthodontic treatment is usually necessary, unless wholesale crowning of one or both arches with an attendant increase in vertical dimension of occlusion is indicated. The dentition may have to be realigned to compensate for overeruptions, drifting and loss of arch length.

In view of the potential application of biomimetic materials and techniques[20] in the restoration of eroded teeth, and in keeping with a modern, minimally invasive approach to dentistry,[21] the natural tooth structure should be preserved whenever possible. Cupped lesions on cuspal tips and minor contour defects can be restored with adhesive resins.[22] Bonded porcelain restorations can be used to restore extensive loss of tooth structure in the anterior teeth.[23,24] There is some early evidence that the biomimetic principles used to restore anterior teeth can also be applied to the restoration of posterior teeth.[25–27]

Many posterior teeth can be treated ultraconservatively with directly applied composite resins, especially if the marginal ridges remain intact. When full coverage of eroded vital posterior teeth is indicated, indirect ceramic overlays may be considered[28] both to preserve natural tooth structure and to avoid traditional prosthodontic approaches

to inadequate clinical crown length, namely elective endodontic treatment, post and core fabrication or surgical crown lengthening.[20] In cases of advanced breakdown, these traditional approaches will in fact be necessary, and cemented ceramo-metal or ceramic crowns may be the treatment of choice.

Because many patients treated for dental erosion caused by GERD are young or middle-aged adults, most dental restorations will require replacement over the patient's lifetime. The speed of deterioration of restorations is determined by many factors, not least the presence of residual reflux, which may contribute to demineralization of the hard dental tissues, particularly in the area of the restoration margins. In the absence of adequate control of GERD, the restoration margins are at risk for development of caries. The most serious consequence is that the restoration will be so deeply undermined that it cannot be replaced (which necessitates removal of the tooth), or it cannot be replaced without further adjunctive periodontal or endodontic procedures. Rigorous medical follow-up for recurrent GERD is imperative to avoid this scenario.

GERD

Definition

GERD is an important cause of dental erosion. Gastroesophageal reflux (GER) is defined as the passage of gastric contents into the esophagus, whereas GERD is defined as symptoms or complications of GER.[29] The most widely accepted criterion for diagnosis of GERD is the occurrence of heartburn 2 or more times per week. However, although heartburn is specific for GERD, it is not very sensitive in this diagnosis. Thus, given the limitations of currently available diagnostic tests, the epidemiology of GERD has been difficult to ascertain.[30]

Prevalence

Estimates of the prevalence of GERD range from 6% to 10%,[31,32] although up to 59% of the population reports heartburn monthly,[32,33] up to 20% report weekly symptoms,[33] and 18% use prescription drugs to manage their symptoms.

Table 3 **Nonmedicinal treatment alternatives (lifestyle measures) for gastroesophageal reflux disease (Andreoli and others[41])**

Elevate head of bed
Avoid food and liquids 2–3 hours before bedtime
Avoid fatty and spicy foods
Abstain from smoking cigarettes and drinking alcohol
Reduce weight
Prophylactic use of liquid antacid (aluminum hydroxide-magnesium hydroxide), 30 mL 30 minutes after meals and at bedtime

Table 4 **Medical therapy for gastroesophageal reflux disease (Andreoli and others,[41] Rubin,[42])**

Therapy	Agent	Dosage
Acid-neutralizing agents	Sodium bicarbonate ($NaHCO_3$) (baking soda)	1 suppository PR 325–650 mg PO
	Magnesium hydroxide (milk of magnesia)	1.8–14.4 g qd
	Aluminates (Maalox, Pepto-Bismol)	15–45 mL q3–6h
Histamine-2 blockers	Cimetidine (Tagamet)	800 mg PO hs 400 mg bid
	Ranitidine (Zantac)	300 mg PO hs 150 mg bid
	Famotidine (Pepcid)	20 mg PO bid
	Nizatidine (Axid)	150 mg PO bid
Gastric emptying	Metoclopramide (Reglan)	10–20 mg PO, IM or IV (IV given over 1–2 min)
Prokinetic agents	Cisapride (Propulsid)	10–20 mg PO qid
Proton pump inhibitors	Omeprazole (Prilosec)	20–40 mg qd every morning
	Lansoprazole (Prevacid)	15–30 mg qd every morning
Surgery	Nissen fundoplication	

PR = per rectum, PO = per os, qd = every day, q3–6h = every 3–6 hours, hs = at bedtime, bid = 2 times daily, IM = intramuscular, IV = intravenous, qid = 4 times daily

Causes and Pathophysiology

In healthy individuals, most gastric refluxate is returned to the stomach by peristalsis stimulated by swallowing. The remaining refluxate is cleared by secondary peristalsis stimulated by direct contact of the juice with the esophageal mucosa.[34] In contrast, patients with GERD have delayed acid clearance. Bartlett and others[35] found that patients with dental erosion were less able to clear refluxate from the esophagus, and this problem appeared to be correlated with poor esophageal motility. Gastroparesis, increased abdominal distension and myopathy affecting gastrointestinal motility are all etiologic agents in GERD.

GERD has been classified into 2 types: physiologic and pathologic. The physiologic form occurs postprandially and is associated with eructation or belching. It is usually temporary and does not require medication. Physiologic GERD is common in infants, in whom it usually resolves spontaneously by 1 year. In some adults, however, pain and other symptoms may accompany belching. If clearance mechanisms cannot return the refluxate back to the stomach and the condition becomes chronic, it is known as pathologic GERD.[11,35] The demarcation between physiologic and pathologic GERD remains ill-defined because of a lack of consensus.[30]

Hiatus hernia can cause both physiologic and pathologic GERD. It may be associated with an incompetent reflux barrier but is not a prerequisite for GERD.[11] Some drugs (specifically nitrates and calcium-channel blockers) and cigarette smoking have also been implicated in GERD.

Clinical Presentation

Extra-esophageal manifestations of GERD are common and involve both soft and hard tissues.[36] Heartburn, noncardiac chest pain, chronic cough, chronic hoarseness, asthma[37] and idiopathic pulmonary fibrosis have all been associated with GERD.[38] Additional signs of GERD include gastric juice in the mouth, chronic laryngitis, laryngeal granuloma and ulcers, laryngeal carcinoma, chronic sore throat, subglottic stenosis, vocal cord polyps, nighttime cough and globus pharyngeus.

Reflux affects individuals differently at different times of the day. Some patients report continuous reflux throughout the day, whereas others experience it primarily nocturnally or intermittently during the daytime.[39]

Diagnostic Tests

There is as yet no single test that can consistently detect GERD,[30] although, depending on the clinical situation, reflux can be demonstrated with several diagnostic tests, such as barium esophagography, endoscopic examination, esophageal acid perfusion, measurement of lower esophageal sphincter pressure, mucosal biopsy, standard acid reflux test and radionuclide scintography.[40]

The most useful diagnostic tool currently available to diagnose GERD is 24-hour monitoring of esophageal pH[40] by means of a catheter passed through the nares to a point 5 cm above the lower esophageal sphincter. If the pH in the distal esophagus remains below 4.0 for more than 4% of the time, the condition is considered pathologic.[11,29]

Treatment

The goals of treatment for patients with GERD are multifocal. From the medical perspective, accurate diagnosis is imperative. Treatment may combine nonmedicinal therapy such as elevating the head of the bed and avoiding fatty and spicy foods (**Table 3**), as well as drug therapy (**Table 4**).

Medicinally, histamine-2 (H_2) blockers and drugs that enhance gastric motility have been the mainstay of treatment. Proton pump inhibitors are efficacious in controlling GERD refractory to therapy with H_2 blockers (**Table 4**).[42] Successful control of GERD by medicinal therapy is

confirmed through repeat monitoring of esophageal pH. When medicinal therapy is ineffective, surgical intervention (Nissen fundoplication) has been useful.[38]

The complications of untreated GERD include esophageal stricture, esophageal ulcer, Barrett's esophagus, increased risk of transformation to esophageal adenocarcinoma, pulmonary aspiration and upper gastrointestinal hemorrhage.[43]

Discussion

Treatment of dental erosion resulting from GERD involves a multidisciplinary approach among family physician, dentist, prosthodontist, orthodontist and gastroenterologist. Most patients with dental erosion who undergo treatment have been referred, not by physicians, but rather by the family dentist. This pattern reflects our belief that dentists are usually the first health care providers to recognize GERD because of its oral manifestations.

For many patients with dental erosion, there is sufficient evidence of pathological reflux, both clinically and on monitoring of esophageal pH, to warrant medical intervention. When medical treatment is indicated, a careful assessment of the risk-benefit ratio is required, because the consequences of long-term medication are unknown. Generally, medical treatment leads to amelioration of GERD and paves the way for dental treatment. Medical follow-up is necessary to monitor for recurrence of GERD, which would not only put healthy, unrestored tooth surfaces at risk of erosion, but would also risk undermining the usually extensive oral reconstruction.

Paradoxically, in some patients with dental erosion, in whom neither an extrinsic cause nor any intrinsic cause other than GERD can be identified, there is insufficient evidence of pathological reflux to justify medical treatment. This situation creates a dilemma. Withholding restorative treatment because a cause has not been identified could lead to further deterioration of the dentition, and the patient and his or her family may feel that the dentist lacks diagnostic acumen or has denied the patient necessary treatment. However, the provision of definitive restorative procedures in the continuing presence of an oral acid challenge will lead to premature failure of treatment and will likely leave the patient in worse condition than before treatment. Nevertheless, even when the cause of dental erosion cannot be ascertained, some form of reconstruction may be necessary. Therefore, when initiating restorative treatment in the absence of a definitive diagnosis of GERD, it behooves dental professionals to also prescribe strategies such as plaque control and reduction of intake of refined carbohydrates and carbonated beverages to maximize the potential for remineralization and optimize the pH-buffering capacity of the oral environment. Similarly, patient behaviour that might reduce putative GERD should be encouraged.

It is our hope that future collaboration between the disciplines of dentistry and gastroenterology will further elucidate the causal relationship between GERD and dental erosion. Furthermore, in restoring dental erosion, a minimally invasive approach that takes advantage of all the modern advances in fluoride use, adhesive dentistry and biomimetic materials should be employed whenever possible. ❧

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