Erosion Caused By Gastroesophageal Reflux: Diagnostic Considerations

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Dental erosion occurs with a high prevalence in the general population, but its impact on prosthodontic care is often unrecognized. The etiology of dental erosion is difficult to establish because it may result from a variety of causes and may have different presentations. Our purpose was to review the literature relevant to dental erosion and to report a patient presentation in which an unusual pattern of severe erosion was the dental manifestation of asymptomatic gastroesophageal reflux disease.

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LOSS OF TOOTH STRUCTURE over time may be considered to be normal or pathological, depending on the etiology and rate of loss. Normal loss of tooth structure is usually caused by functional microwear or attrition and proceeds at physiological levels throughout life. It is normally related to dietary and oral hygiene habits.[1] Normal vertical loss of dental hard tissues has been estimated to be about 65 μm/yr.[2] This may be three to four times higher in patients with bruxing habits.[3] Pathological loss of tooth structure occurs at an accelerated rate and may be caused by one or more processes: 1) abnormal attrition: caused by clenching or bruxing of one tooth surface against another; 2) abrasion: caused by physical wear by extraneous objects such as toothbrushes; 3) erosion: erosion may be defined as chemical damage, mostly demineralization by acids other than those produced by bacteria. Some chelation of the organic phase of the matrix may also occur[4]; and 4) developmental disturbances: these may be the result of amelogenesis imperfecta, dentinogenesis imperfecta, or environmental enamel hypoplasia. Enamel hypoplasia may be caused by nutritional deficiencies, hypocalcemia, exanthematous fevers, local infection or trauma, excess fluoride intake, or birth injuries. These disturbances can result in early and accelerated loss of tooth structure caused by deficient quantity or quality of enamel.

This article reviews the possible causes and diagnostic considerations for dental erosion, and presents a patient report describing the severe dental manifestations that can result.

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Etiology of Dental Erosion

The etiology of dental erosion is associated with one or a combination of several factors, making diagnosis difficult. Dietary acids, environmental exposure, and gastric dysfunction have all been shown to be potential etiologic factors for dental erosion.

Dietary Acids

Dietary acids in the form of carbonated drinks or citrus fruit usually affect the cervical third of the labial surfaces of the maxillary anterior teeth.[5] It is thought that the citrate present in these products binds the calcium in the dentin and enamel to form a soluble calcium citrate complex. Idiopathic erosion may occur by the same mechanism if a patient has a higher-than-normal salivary citric acid content.[6] Alcoholics have been documented as having increased tooth wear, although the mechanism is obscure. Ethanol-induced vomiting presents a feasible etiology,[7] as does subclinical regurgitation caused by

chronic gastritis[8] and erosion caused by the accompanying citrus juice mixers often used with the alcohol.

Environmental Exposure

Environmental exposure to acidic fumes or proteolytic enzymes in the workplace has been reported as an unusual etiologic factor in dental erosion.[9] Frequent swimming in chlorinated pools has also been documented[10] as causing loss of tooth structure, the anterior tooth surfaces being characteristically affected.[11]

Gastric Dysfunction

Gastroesophageal reflux disease or chronic vomiting associated with bulimia nervosa can result in an erosive loss of the lingual surfaces of the teeth.[12] This may be classified as a special type of erosion termed "perymylolysis" and is thought to be caused by a low pH along the lateral borders of the tongue combined with muscle hyperactivity of the tongue. This produces a combined erosive and mechanical action primarily along the palatal surfaces of the maxillary teeth.[13]

Erosion caused by gastroesophageal reflux affects the maxillary anteriors initially, but, as the erosive process continues, the lingual cusps of the maxillary posteriors and eventually the mandibular teeth may also be affected. Involvement of the mandibular teeth is infrequent[14] and indicates a longstanding problem. Symptoms such as hypersensitivity, tooth fracture, and pulpal pain may be associated with this progression.

The solubility of dental tissues is affected by the pH of the oral cavity. As a rough estimate, as the oral pH decreases from normal (pH 6.5) to acidic, the solubility can be calculated to increase by a factor of seven to eight times each time the pH decreases by 1 unit.[15] Gastric contents may have acidity below pH 1, and, therefore, regurgitation can have a severe demineralizing effect on tooth structure.

The presence of gastric dysfunction has been reported as 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 with controls.[16] Because it has been demonstrated that many patients with gastric reflux disease are asymptomatic, one may surmise that a larger-than-reported proportion of the population is at risk for dental erosion.

The following patient report illustrates the severe damage to the dentition that may result from undiagnosed gastroesophageal reflux disease, and outlines diagnostic measures used to identify reflux as an etiologic factor.

Clinical Report

A 32-year-old woman was referred to the Graduate Prosthodontics Department at The School of Dentistry, UC San Francisco. The patient complained that her teeth were showing early signs of wear and her appearance was compromised.

Intraoral examination revealed extensive loss of tooth structure and an unusual distribution of erosive-type lesions of the dentin and enamel. (Figs 1 and 2). This patient presented with almost complete loss of enamel, having secondary dentin or pulp exposure on most occlusal surfaces. Both the maxillary and mandibular teeth were equally affected.

The clinical findings were consistent with extreme wear or erosion. The dental lesions were assigned scores according to Smith and Knight's Tooth Wear Index,[17] which uses a numerical scale to measure various clinical presentations of loss of tooth structure. Nearly all teeth scored an index of 4 (the highest score).

Assessment of the patient's vertical dimension of occlusion was evaluated using speech, swallowing, and lateral cephalometric radiography. It was determined that the patient had lost vertical dimension of occlusion, and, therefore, that the loss of tooth structure had been relatively rapid, because a slow progression is thought to be compensated for by continual eruption of the dentition.[18] The patient reported that she had only noticed the dental changes in the past 5 years and was able to produce photographs to support her view that her facial height had rapidly changed (Figs 3 and 4).

An initial examination revealed that the patient's oral hygiene was good. All soft tissues and related structures were within normal limits. Pocket depths of 3 to 4 mm were noted. Pulp chambers of several maxillary and mandibular incisors were exposed. Salivary flow and consistency appeared normal. No caries were evident, and the patient had only one

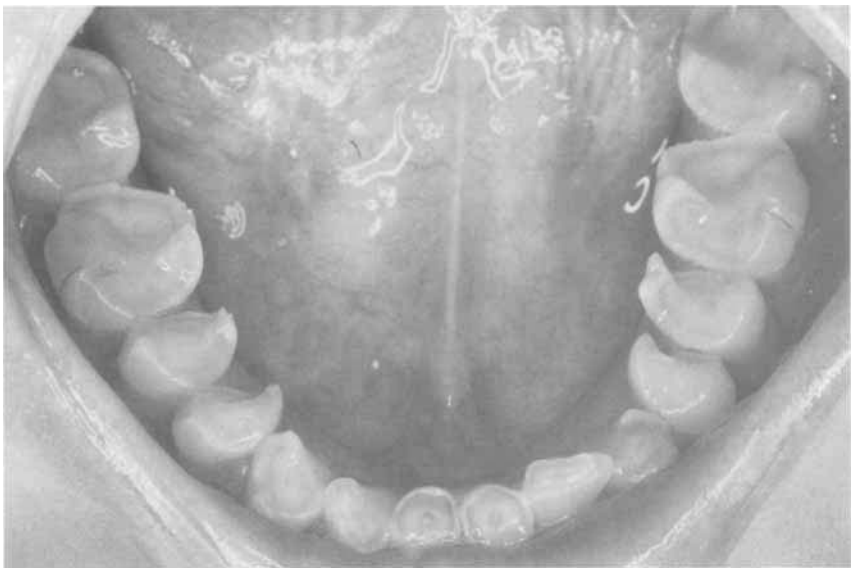

Figure 1. Occlusal view, maxillary arch.

occlusal amalgam restoration on the right maxillary second molar. The exposed dentin surfaces were hard and shiny and had a subjectively "normal" feel when examined with an explorer. The striking feature of the patient's presentation was that both buccal and lingual cusps were equally affected and that there were still sharp cusp remnants present on all teeth.

In maximum intercuspal occlusion, the patient closed to almost complete contact of all occlusal surfaces (Fig 5). The mandibular incisors closed into the extremely eroded maxillary cingulum areas and onto the areas of pulpal exposure that were present. In a closed position, there was no freedom for lateral movements (Figs 6 and 7). The patient reported no difficulty in eating her normal diet. The patient's mother was available for examination and did not show any similar features.

Medical History

The patient had a complicated medical history including juvenile onset diabetes mellitus and end-stage

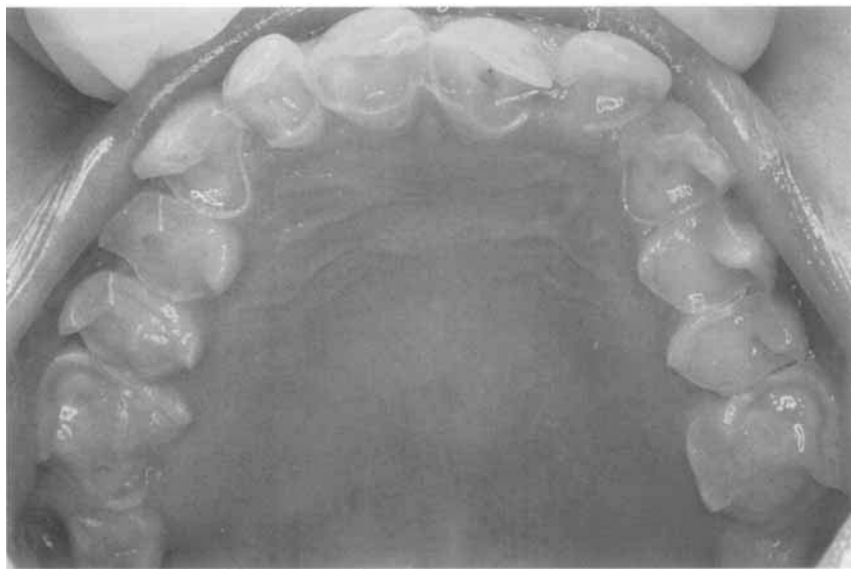

Figure 2. Occlusal view, mandibular arch.

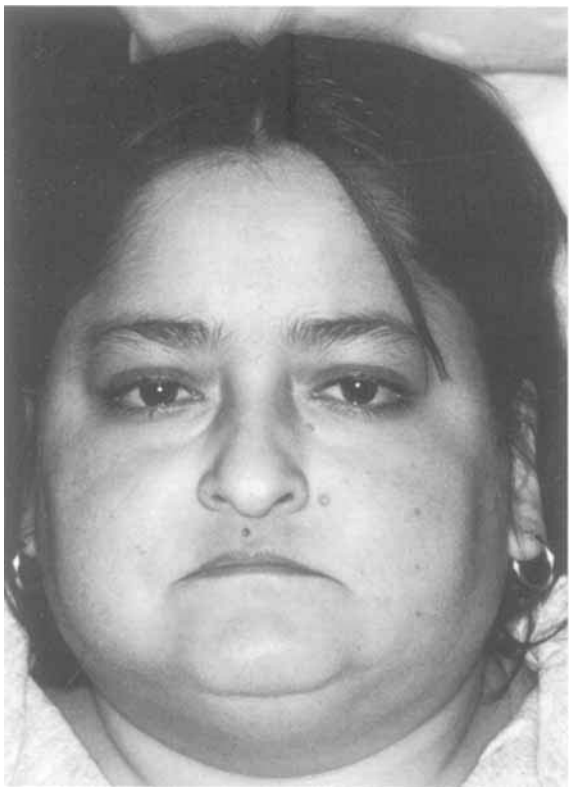

Figure 3. Patient at age 32.

renal disease with less than 5% kidney function. The patient had been on hemodialysis three times weekly for 5 years while waiting for a second transplantation opportunity. The first renal transplantation was performed in 1990. The following medications were being taken: ferrous fumarate, metolazone, furosemide, Premarin, Provera, Synthroid, prednisone, metoprolol, mevacor, and insulin. The medications were reviewed by the pharmacy service at UC San Francisco to determine if loss of tooth structure was a known possible side-effect of their use. None was noted.

Figure 4. Patient at age 22.

Diagnostic Tests

Further investigation was required to determine which of the many complicating medical and dental factors could be contributing to the extreme loss of tooth structure that the patient was experiencing. Radiographic examination revealed cup-shaped lesions on all the occlusal surfaces, roots, and pulp chambers, with normal morphology and some exposed pulp horns occlusally. There was evidence of widened periodontal ligaments associated with the mandibular first and second molars.

Salivary function was tested and proved to be within normal limits, although the buffering capacity was at the higher end of the normal range. This may be due to the effect of the antacids the patient was taking (Tums) to regulate phosphorus and potassium. Jarvinen reported no statistically significant difference between stimulated and unstimulated salivary flow rates and salivary pH between controls and erosion cases[19]; this was corroborated by Meurman et al.[20]

A temporomandibular disorders workup to assess bruxing and clenching proved negative. An extensive review of the patient's dietary habits and history of fluoride use also proved to be within the normal range. Analysis of the tooth structure was performed. A fractured maxillary molar cusp was harvested and provided a sample of both enamel and dentin. This was processed and examined with a scanning electron microscope. Dentin tubules appeared to be of normal size, shape, and orientation. A polished sample was subjected to Knoop microhardness testing and compared with normal controls. The Student's *t* test was applied to the results, and there was no statistically significant difference between the two. It was therefore determined that the structure of the teeth was normal and that the lesions were caused by chemical erosion, abrasion, or a combination of the two.

The dietary investigations and other special tests suggested no apparent external source for erosive

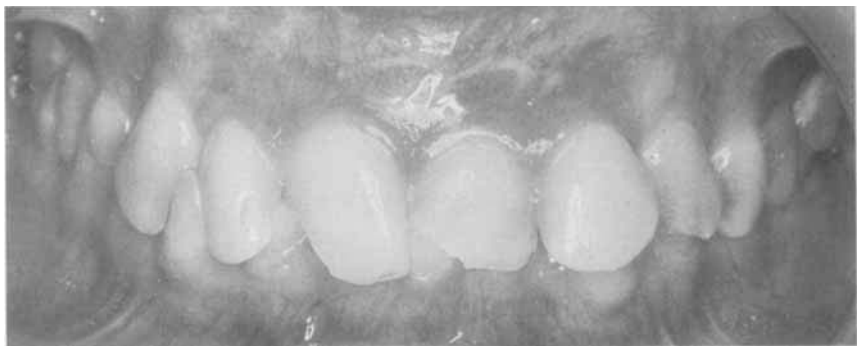

Figure 5. Anterior view of maximum intercuspal position.

chemicals, and it was therefore deemed appropriate to perform a 24-hour ambulatory gastroesophageal pH probe, even though the patient did not report any experience of reflux. The patient was referred to the Division of Gastroenterology at UC San Francisco, where a two-channel pH probe was placed transnasally to a position 5 cm above the lower esophageal sphincter (LOS). The probe consisted of two pH-sensitive electrodes, one at the tip and one 15 cm proximal to it. Information regarding the pH at the two electrode sites in the esophagus (5 cm and 20 cm above the LOS) was gathered every 4 seconds and stored on a portable data recorder that the patient kept with her. Data were recorded over a 24-hour period, during which the patient was asked to keep a log of meal times and her bodily position, either supine or upright. At the end of the observation period, the information was downloaded to a personal computer and graphs were generated (Figs 8 and 9). The readings were compared with established normals and given a score according to a protocol developed by Johnson and DeMeester[21] (Table 1). The Johnson and DeMeester scoring system is the usual method for reporting the various parameters of gastroesophageal reflux. Reflux was defined as occurring when the pH was less than 4. Six components were measured and obtained from the data recorded: % time pH was less than 4 for 24 hours, % time for supine and upright positions, number of single episodes, number of episodes greater than 5 minutes, and the time of the longest episode. The 24-hour score was determined by calculating the number of standard deviation equivalents in each measured value of the six components starting at a fixed reference point placed 2 standard deviations below the respective measured mean value in the group of control patients (asymptomatic). This provided a numerical score for comparing the 24-hour results of the control versus the symptomatic patients.

The results of pH manometry for this patient showed reflux at the distal sensor for 89% of the 24-hour period compared with a norm of less than

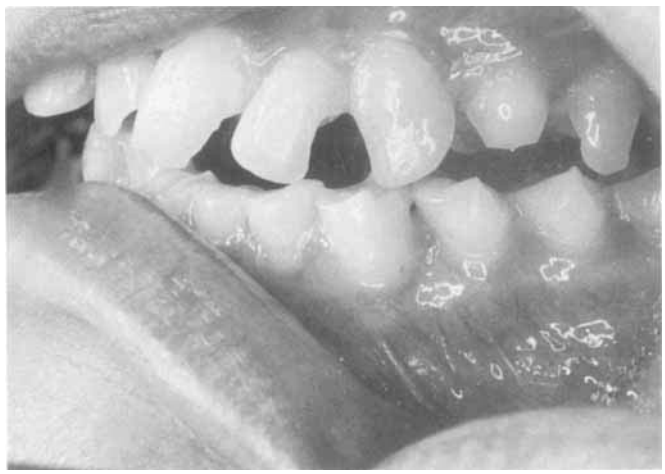

Figure 6. Lateral view on closing.

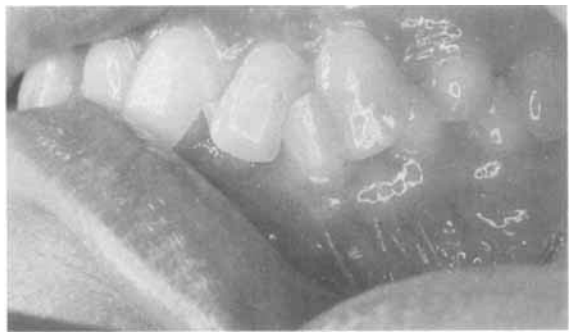

Figure 7. Lateral view, maximum intercuspal position.

5%. One episode lasted 434 minutes and occurred while the patient was in the upright position. The second and more proximal probe also showed evidence of reflux, approximately 5% of the time. The pH probe does not record reflux in the hypopharynx, but one could postulate that the patient was refluxing far more proximally into the pharynx and mouth. These results are abnormal and demonstrate that the esophagus was almost continuously bathed in an extremely acidic solution; therefore, it may be inferred that the oral cavity was also under the influence of the acidic esophageal environment.

Discussion

Dental erosion often has a multifactorial etiology and is sometimes the presenting sign of an underlying condition such as gastroesophageal reflux disease (GERD). This case report represents an extreme example of erosion involving mainly the occlusal surfaces, the lingual surfaces being relatively unaffected. A 24-hour ambulatory gastroesophageal reflux study has revealed a pattern of almost continual asymptomatic reflux, which has been associated with dental erosion.[22-24]

Direct oral pH measurements were not completed in this study. However, in a study of 36 patients, Bartlett et al showed a significant ($p < .002$) correlation between the pH recorded in the distal esophagus and the oral cavity.[25] Although Barlett was able to successfully measure oral pH with a telemetry capsule, the device is still experimental. The standard 24-hour esophageal pH probe is readily available, is usually covered by medical insurance, and provides valuable diagnostic information. Gastroesophageal reflux can be treated using 20 mg omeprazole twice per day or 400 mg cimetadine twice per day. This regime can produce rapid and almost-complete resolution of the condition with minimal

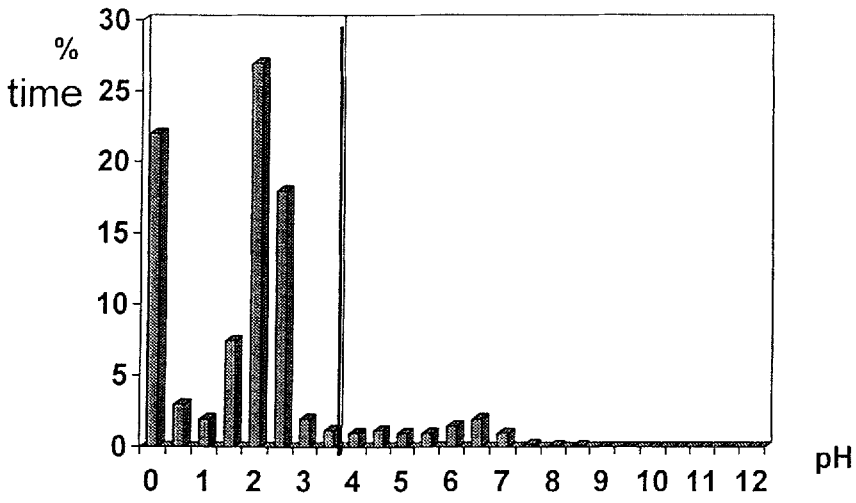

Figure 8. Frequency histogram, channel 1. Percentage of total observation time at pH values. (Data recorded every 4 seconds over a 24-hour period, 5 cm proximal to gastric sphincter.) For 18,222 samples (*bar width* = 0.5 pH).

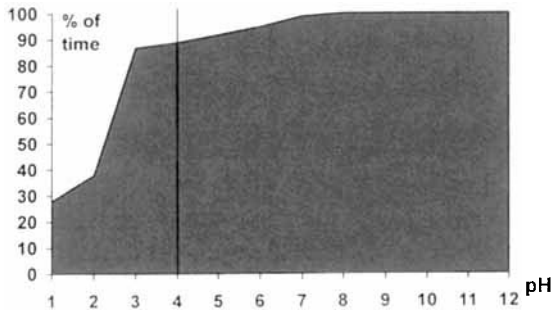

Figure 9. Cumulative histogram, channel 1. Percentage of total observation time Below pH values. (Data recorded every 4 seconds over 24-hour period, 5 cm proximal to gastric sphincter.) For 18,222 samples.

Table 1. Patient's Recorded Values Compared With Normals

Parameter	*Value*	*Normal*	*Score*
% time reflux upright	88.0	<6.3	44.4
% time reflux supine	90.0	<1.2	193.1
% time reflux total	89.0	<4.2	64.4
Episodes > 5 min/24 h	7.0	<3	5.9
Longest episode (min)	434.0	<9.2	161.0
Total episodes	77	<50	4.8
Composite score		<22	473.5

NOTE. From Johnson and DeMeester.[21]

side-effects. Therapy is usually long-term. After further tests and endoscopy, the patient may opt for surgery to permanently resolve the problem.

Summary

The prosthodontist is frequently called upon to restore badly broken-down dentitions involving severe dental erosion. It is very important to determine the etiology of the lesions before restoration. It is therefore important to consider and test for gastroesophageal reflux in cases in which dental erosion has occurred and the etiology cannot be established. Twenty-four-hour pH monitoring allows the clinician to reconcile clinical findings with pH data and is a useful addition to the available armamentarium for diagnosis. In the clinical report described, pH monitoring provided data that enable the diagnosis of the previously unrecognized condition of GERD. Appropriate treatment can result in the establishment of a stable environment for dental rehabilitation. It is only after establishing such a stable environment that the rehabilitation can be completed with a positive prognosis.

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