

CLINICAL REPORT

Gastroesophageal Reflux Disease, Tooth Erosion, and Prosthodontic Rehabilitation: A Clinical Report

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Gastroesophageal reflux disease (GERD) is a relatively common gastrointestinal disorder in the United States. The reflux of acid adversely affects the mucosal lining of the esophagus and is responsible for dental erosion. This article briefly reviews the etiology, risk factors, and medical management of GERD. The patient presentation describes the rehabilitation of a young adult with GERD who needed multidisciplinary care.

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GASTROESOPHAGEAL REFLUX disease (GERD) is a relatively common gastrointestinal disorder in Western society. Heartburn is reported at least once a week by 15% of Americans and daily by 7%.¹ This symptom is caused by a backflow of gastric acid and other gastric contents into the esophagus. Normally, the lower esophageal sphincter (LES), the anatomic location of the gastroesophageal junction, and the crural diaphragm prevent the movement of fluid or solid matter from the stomach into the esophagus. Reflux occurs when the LES relaxes, causing loss of the pressure gradient between it and the stomach. The refluxed material may reach the cervical esophagus, pharynx, and oral cavity. The relationship between GERD and dental erosion has been well documented in the literature.⁷⁻¹⁴

The medical concern is that acid reflux in the esophagus may damage the mucosal lining. Reflux esophagitis can be mild, involving only microscopic changes in the cells of the mucosa, or erosive, causing bleeding and superficial linear ulcers. From a dental standpoint, acid reflux in the oral cavity causes the loss of coronal tooth structure by chemical erosion. It has been reported that patients

experiencing vomiting 1 or more times a week, heartburn, belching, pain on awakening, acid taste, or stomach pain have dental erosion 31 times more frequently than controls.²

Risk factors for GERD include obesity, hiatal hernia, and pregnancy.³ Approximately 50% of persons over age 50 have hiatal hernias; however, as many as 84% of patients with erosive esophagitis have hiatal hernias.³ Gastroesophageal reflux and heartburn are reported by 45%–85% of women during pregnancy.⁴ Substernal burning after eating, the most common symptom, is worsened by fatty or spicy foods, large meals, alcohol, or caffeine. Recumbency, heavy lifting, or bending following a meal may cause food or liquid to rise into the throat. Treatment of mild cases of GERD may involve life-style changes including modified diet, decreases in the volume of food or liquid, sleeping with the head of the bed raised 4 to 6 inches, weight reduction, and use of over-the-counter antisecretory agents. In more severe GERD, histamine-2 (H²) receptor blocking agents (nizatidine, 150 mg twice a day; ranitidine, 150 mg twice a day; cimetidine, 300 mg/day) are prescribed for 6 to 12 weeks to provide symptomatic relief. For patients resistant to H² receptor blockers or patients with severe GERD, proton pump inhibitors (PPIs) to provide strong acid suppression is the treatment of choice. Rabeprazole (20 mg/day), omeprazole (40 mg/day), and lansoprazole (30 mg/day) are commonly prescribed PPIs.

Several testing procedures may be used to confirm the diagnosis of GERD. Endoscopy of the esophagus with biopsy is the standard procedure for

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documenting the type and extent of tissue damage. Barium esophagography is used to identify any stricture in patients with severe dysphagia. The 24-hour ambulatory esophageal pH manometry test is the best study for determining the severity of the gastric reflux into the esophagus.⁵ This test is performed by passing a pH probe sensitive catheter through the nose into the esophagus. The pH probe is positioned above the LES. Connected to the other end of the catheter is a small computer that is worn around the waist. The pH probe is left in place for 24 hours. During this time the patient may consume a normal diet but is instructed to not swim, bathe, or shower.

This report presents the clinical manifestations, diagnosis, and medical and dental management of a patient with GERD with severe tooth erosion.

Clinical Report

A 14-year-old Caucasian male first presented to the author for a dental evaluation in 1998. The patient was referred by his pediatrician, who had noted the patient's significant loss of coronal tooth structure and made a diagnosis of bruxism. Clinically, the appearance of the teeth was inconsistent with that usually seen in people with bruxism. Besides the occlusal/incisal surfaces, loss of tooth structure also occurred on the buccal and lingual surfaces. This indicates that erosion, rather than attrition, was responsible for the destruction.

The patient's medical history revealed that he experienced periodic regurgitation of stomach acid and food from 1993 to 1998. He also felt some retrosternal burning several times a month. The patient's previous dentist fabricated a bite guard appliance to control tooth wear that the patient had used from 1995 to 1998. It was clear that the patient needed further medical evaluation to adequately address his dental needs.

The patient was referred to a gastroenterologist for an upper gastrointestinal (GI) radiograph to evaluate possible gastric outlet obstruction and a 24-hour ambulatory esophageal pH manometry test. The results of the upper GI radiograph were normal; the results of the 24-hour pH reflux monitoring test indicated episodes of reflux. The patient was placed on omeprazole, 20 mg/day, and instructed to elevate the head of the bed 6 inches. The patient's symptoms then resolved.

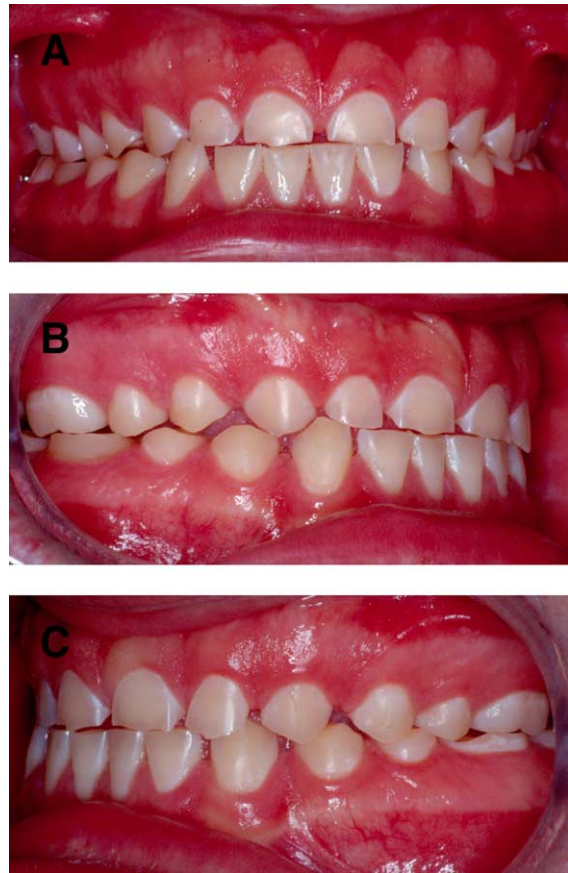


Figure 1. Pretreatment. (A) Front view. (B) Right lateral view. (C) left lateral view.

This patient needed a multidisciplinary approach to dental rehabilitation. Because of the large volume of tooth structure that had been lost, it was necessary to restore the patient's occlusal vertical dimension to permit the fabrication of cast restorations for the anterior and posterior teeth (Fig 1 A-C). The diagnostic waxing procedure on casts mounted in centric relation on a semiadjustable arcon articulator indicated that orthodontic treatment was needed so that a mutually protected occlusion could be developed prosthetically. A semiadjustable articulator was selected because the occlusal scheme provided anterior disocclusion. The patient's 4 impacted third molars were removed before orthodontic treatment. Crown-lengthening procedures were performed surgically on the mandibular left second premolar, first molar, and second molar to expose sufficient tooth structure to permit the placement of orthodontic bands. Composite resin was bonded to the occlusal surfaces of the remaining posterior teeth to provide sufficient

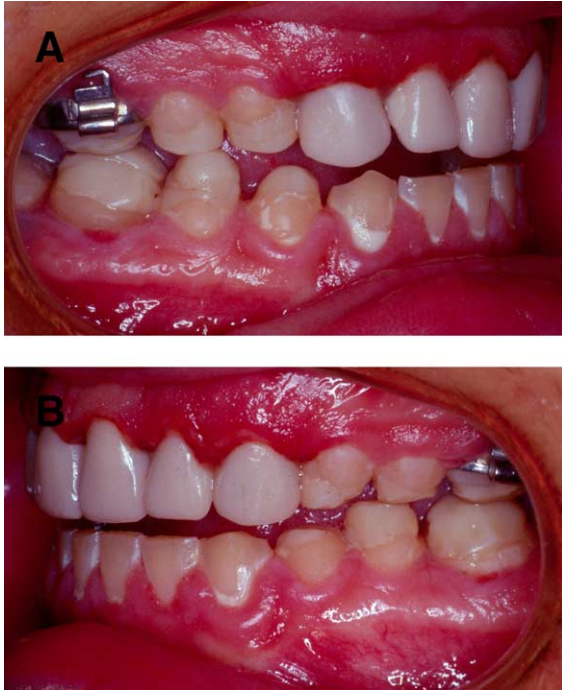


Figure 2. Composite resin bonded to posterior occlusal surfaces to increase the vertical dimension of occlusion. (A) Right lateral view. (B) Left lateral view.

attachment area for the orthodontic appliances. The initial increase in occlusal vertical dimension occurred at this time. All teeth received orthodontic bands and/or brackets.

As the orthodontic treatment was nearing completion, another diagnostic waxing procedure was performed to assess the interarch relationship at the desired occlusal vertical dimension. A full contoured wax-up on the mounted casts made it possible to evaluate tooth dimensions to achieve optimal esthetics.

Additional composite resin was bonded to the occlusal surfaces of the posterior teeth to achieve the same increase in the occlusal vertical dimension that had been developed on the mounted casts (Fig 2A, B). I prefer to use a processed acrylic bite guard to assess changes in the occlusal vertical dimension, but this approach would have interfered with this patient's ongoing orthodontic treatment. The patient had no difficulty adapting to the new occlusal vertical dimension; he experienced no muscle pain or discomfort and no difficulty in function. The orthodontic treatment was completed in December 2001.

Root canal therapy was performed on both maxillary canines and the maxillary left lateral incisor to permit fabrication of cast dowel and cores to develop adequate retention and resistance form in the tooth preparations. All of the teeth in both the maxillary and mandibular arches were prepared, and provisional restorations were fabricated at the new occlusal vertical dimension.

The patient experienced significant gingival hyperplasia during orthodontic treatment (Fig 3). This necessitated performing a gingivoplasty using an electrosurgical unit to facilitate tooth preparation and impression-making procedures. Noncompliance with daily oral hygiene procedures made it necessary to reduce the hyperplastic tissue both at the time of tooth preparation and again when impressions were made.

The definitive restorations were fabricated first for the maxillary and mandibular anterior teeth, then for the opposing posterior teeth on the left side, and finally for those on the right side. Metal ceramic restorations were placed on all teeth except the second molars. The second molars received full-veneer gold crowns due to restricted interocclusal space and clinical crown length. Prosthetic treatment was completed in August 2002 (Fig 4 A-C).

Discussion

Dental erosion is defined as the loss of tooth structure due to a chemical process that does not involve bacterial action and may be multifactorial in origin. Erosion is not an uncommon finding during oral examination; a prevalence as high as 42% has been reported.⁶ Erosion may be due to extrinsic sources of acid, such as acidic foods, drinks, and acidic medications; however, the most common source of intrinsic acid in children is regurgitation of gastric contents into the oral cavity, as occurs in GERD.¹⁵



Figure 3. Front view illustrating postorthodontic gingival hyperplasia.

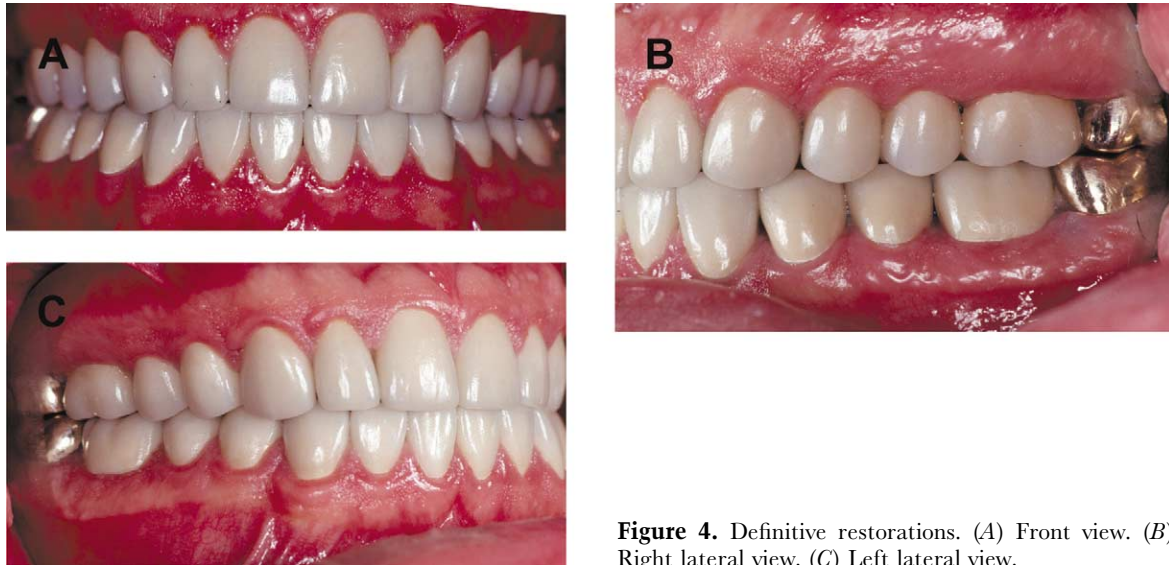


Figure 4. Definitive restorations. (A) Front view. (B) Right lateral view. (C) Left lateral view.

The pattern of loss of tooth structure is similar to that seen in bulimia nervosa. The palatal surfaces of the maxillary teeth are affected first. Erosion of the occlusal surfaces of the posterior teeth in both arches and the labial or buccal surfaces results from an extended period of acid reflux. The lower anterior teeth are the last to be affected.¹⁶

Many patients with GERD do not experience heartburn, belching, unexplained sour taste, or regurgitation. This condition has been termed “silent GERD.”¹⁷ Enamel erosion of the posterior teeth may be the first symptom of GERD. Thorough history taking and oral examination are essential to eliminate bulimia nervosa, attrition, and abrasion as possible causes for the lost tooth structure. Referral to a physician or gastroenterologist for appropriate testing is necessary to confirm a diagnosis of GERD. Dental rehabilitation should not be initiated until medical treatment has eliminated the acid reflux.

Summary

Gastroesophageal reflux disease affects all age groups. The prosthodontist must take this into consideration when faced with the task of restoring teeth with significant loss of coronal tooth structure. GERD by itself or in combination with attrition or abrasion may be responsible for the loss. A thorough diagnostic evaluation is necessary to ascertain the possible medical and/or dental sources for the problem. In the case presented here, the

patient’s previous dentist was treating him for a dental condition (bruxism) when in fact a medical condition (GERD) was responsible for the lost tooth structure. The patient was subsequently referred for a medical evaluation. The medical diagnosis was made, the appropriate pharmacologic agent was prescribed, and the GERD was controlled. After medical control of the GERD was established, the patient’s dentition was restored to correct form, function, and esthetics with an expectation of a favorable long-term prognosis.

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