



Gastroesophageal reflux and dental erosion: case report

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Gastroesophageal reflux (GER) is defined as the involuntary passage of gastric contents into the esophagus. It may be primary, due to anatomical or physiological abnormalities or secondary, due to conditions such as anxiety, intolerance to certain foods, metabolic disorders, and reactions to certain drugs. Other medical problems such as infection, intestinal obstruction, intestinal atresia, or pyloric stenosis and intracranial pathologies such as hydrocephalus, neoplasia, or a subdural hematoma have also been implicated in GER.¹

Important features in the pathophysiology of GER include increased abdominal pressure, transient lower esophageal sphincter relaxations (TLESR), and decreased low esophageal sphincter tone.² Other related factors include increased gastric volume, xerostomia, increased acid or pepsin production, or a defect in the mucins forming the protective lining of the stomach.³

The occurrence of GER is difficult to characterize as most individuals experience occasional episodes of reflux, and the point at which normal physiology develops into pathosis is difficult to describe. Approximately 7% of the general population experiences symptomatic GER on a daily basis, and 36% suffer at least one monthly episode.⁴ The prevalence of GER in the pediatric population is not known. Diagnosis is usually confirmed by the detection of pH values < 4 for at least 6 hr during a 24-hr period using an indwelling esophageal pH probe. pH monitoring has been proposed to be the gold standard for detection of pathogenic GER.^{2,5,6} Prolonged (ambulatory) pH monitoring is the most sensitive technique, but its drawback is that it may not be able to distinguish benign from pathologic GER. The pH of the esophagus normally fluctuates between 4 and 7. The reflux index is the percentage of time when the lower esophageal pH is < 4. In general, children with a reflux index of 5–10% (mild) or 10–20% (moderate) will often be controlled by conservative or pharmacologic treatment modalities, but those with an index of 30% or greater (severe) are more likely to require surgical management.¹

Endoscopy is particularly useful in the pediatric population. This technique can be used to examine the

esophageal mucosa for evidence of inflammation such as granularity, bleeding, ulceration, sloughing, exudate, and stricture. A positive biopsy may be expected to yield evidence of histological changes such as elongation of papillae to two-thirds of the mucosal thickness, basal hyperplasia, ulceration, inflammation, fibrosis, and the appearance of columnar epithelium.⁷ Radiographic visualization techniques such as an upper gastrointestinal (UGI) series with barium feeding or scintigraphy with a Tc-99m-labeled feeding are more commonly used when a structural abnormality is suspected as the underlying cause,⁸ and as such are less useful in children.

Dental implications

Dental erosion is defined as the loss of dental hard tissue by a chemical process which does not involve bacteria.⁹ It is thought that this requires a pH of < 4 for at least 5% of each 24-hr period. Because the pH of the gastric contents is consistently less than 1, reflux into the oral cavity overwhelms local buffering, resulting in surface enamel dissolution. Severity of symptoms ranges from opacities or white spots to a flattening of the cusps and eventual dentin exposure.^{10,11} If this is a slowly progressing condition, narrowing of the pulp chambers may occur due to the deposition of secondary occluding dentin. If progression is more rapid, the pulps may be clearly visible through the occlusal surface due to the loss of hard tissue, and eventually exposed, resulting in pulpal infection and abscess formation.¹¹ Both primary and permanent teeth may be affected. Several systems used to classify the severity of tooth wear^{12,13} may not be appropriate to describe erosion in its early stages as they focus on advanced hard tissue loss as a result of a combination of erosion, abrasion, and attrition. The typical distribution of dental erosion lesions due to acid reflux involve the lingual surfaces of the maxillary teeth and facial surfaces of the maxillary incisors and canines,^{14,15} while erosion lesions due to the ingestion of acidic foodstuffs are usually confined to the facial surfaces of the anterior teeth. However, it has been reported by Järvinen that the cause of dental erosion cannot reliably be identified by the location of the lesions, and that any tooth can be

involved depending on the movements of the tongue, cheek, and lips.¹⁶

Loss of tooth structure to acid erosion can have varying consequences such as sensitivity, flattening of the molar cusps with a possible loss of vertical dimension, cavitation, and pulp exposure. Due to the nature and distribution of erosion lesions, they offer little mechanical retention and present problems regarding functional and esthetic restoration.¹⁷

Therapy

Conservative management of GER includes the avoidance of carbonated beverages, spicy foods, tea, coffee, and decreasing dietary fat. Sleeping in the prone position is to be avoided, as is eating within 3 hr of retiring.

Pharmacological agents

Antacids to increase pH and deactivate pepsin are first-line pharmacological agents in the management of GER. Prokinetics such as cisapride (Propulsid) are of use to increase gastrointestinal contractile amplitude and improve antero-duodenal coordination. Metoclopramide increases lower esophageal sphincter tone and stimulates UGI tract motility. Histamine₂-receptor antagonists (H₂RAs) act on gastric parietal cells to inhibit basal and nocturnal acid secretion and include cimetidine, famotidine, nizatidine, and ranitidine. Omeprazole is for use only when H₂RA therapy has failed.⁶ It acts by inhibiting activity of the H⁺/K⁺ pump in gastric parietal cells, but has many side effects and is used with extreme caution.

Surgical Intervention

Surgical intervention is used infrequently in children and only when medical management has failed, when an esophageal stricture is present, in Barrett's esophagus, and when GER is secondary to a brain injury. The most commonly used procedure, the Nissen fundoplication, has two benefits, 1) passively increasing the lower esophageal sphincter (LES) pressure and 2) dynamic augmentation of the pressure when the stomach contents also fill the part of the fundus wrapped around the LES.²

This purpose of this paper is to report a case of GER, a preschool child presenting with severe enamel erosion in the absence of systemic symptoms.

Case Report

History and chief complaint

A 3.25-year-old Caucasian female with the chief complaint of "a loss of enamel and discolored teeth" reported an unremarkable medical history and no unusual dietary or other habits. She appeared to be in good general health and was bright and cooperative. Her mother stated that she had been given a fluoride supplement as a baby, but that prescriptions had not

been refilled beyond 1 year of age because of concerns of enamel fluorosis. Her 6-year-old brother was healthy and caries free. No other family members were affected. Within the previous 6 months, she had seen a general dentist who had commented on the condition but had not recommended a specific follow-up.

Clinical examination

A clinical examination revealed widespread enamel demineralization on the apical 50% of the labial surfaces of both the maxillary and mandibular primary teeth, and cavitation with exposed dentin on the labial surface of both of the maxillary primary lateral incisors, with the left being more severely affected. There were cervical lesions on labial surfaces of the maxillary primary central incisors (Fig 1) where

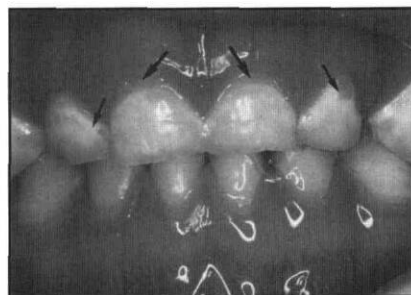


Fig 1. Anterior view showing demineralized enamel and "dished out" lesions of cervical area of maxillary central incisors.

the enamel was very thin. An amalgam restoration had recently been placed by her general dentist on the buccal surface of the primary lower left first molar. There was no evidence of caries at this time and the soft tissues were healthy. The treatment rendered included the restoration of the maxillary left primary lateral incisor with light-cured glass ionomer cement (good retentive properties and fluoride releasing agent), a prophylaxis to remove plaque and allow visualization of all surfaces for a complete diagnosis, and a topical application of 1.23% acidulated phosphate fluoride gel in an attempt to arrest the demineralization and induce remineralization.

The parents were instructed in oral hygiene procedures using a soft toothbrush and fluoride dentifrice and advised to apply a fluoride rinse daily. When questioned, the parents did not report the excessive consumption of acidic foods or drinks such as fruit drinks or sodas. A differential diagnosis was developed which included dental erosion due to unreported acidic food or drink (especially fruit juice or carbonated beverages) and GER. Due to the absence of any other symptoms, the most likely cause was thought to be diet-related which was not being reported by the parents, although they were extensively questioned in this area. Nursing caries and eating dis-

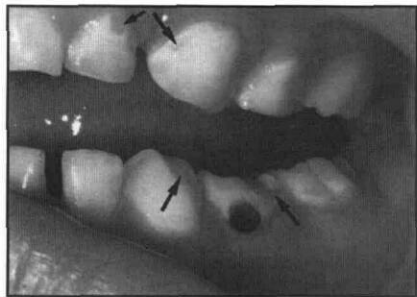


Fig 2. Demineralization lesions of left mandibular and maxillary teeth. Notice the extensive tissue loss from the first mandibular molar and adjacent canine tooth.

orders were not included in the differential diagnosis due to the atypical distribution of the lesions and the age of the patient. The recall interval was set for 3 months to determine if the condition was actively progressing or a symptom of a past pathology.

At the recall appointment cavitation in the apparent absence of caries was noted adjacent to the restoration placed 3 months previously (Fig 1). The child's parents reported compliance with the daily fluoride application as prescribed. The lesion was restored and the patient discharged for a further 3 months. Again, no systemic symptoms were reported.

When the patient was seen again 3 months later, the mandibular left primary canine and primary first molar demonstrated increased erosion (Fig 2). The parents now revealed a history of the patient failing to gain weight, difficulty in sleeping at night, and abdominal discomfort which they had dismissed, thinking it was a way of postponing bedtime. At this visit a tentative diagnosis of GER was made, and the patient was referred to a pediatric gastroenterologist for examination and treatment. A diagnostic endoscopy was performed revealing evidence of gastritis, inflammation of the small intestine, and esophageal bleeding with probable evidence of gastric reflux. The therapy prescribed was milk of magnesia as required, pepcid (Famotidine)—0.25 tsp every morning and Propulsid—3.5 mL, 20 min before meals and before retiring.

Six months since the start of systemic therapy there were still intermittent complaints of esophageal pain, but all medication except milk of magnesia were discontinued. She had demonstrated a weight gain of 4 pounds in the preceding 2 months which contrasted the single pound gained in the entire previous year. Her appetite had increased and the dental condition appeared stable. She was then placed on routine 6-month recall visits.

Discussion

Dental erosion from dietary sources (e.g. fruit juice, carbonated beverages, citrus fruit, especially sucking

lemons and sal y limon) or produced endogenously from chronic vomiting or regurgitation¹⁸ is an irreversible condition which is often a symptom of systemic illness.^{9,10,18-21} Anecdotal evidence suggests that erosion is increasing in the pediatric population. This is supported by a limited study of 14-year-olds in Liverpool, England.²² Cases of enamel erosion associated with GER have been reported in children attending a pediatric outpatient clinic for GER disease.¹⁰ In 1992, Taylor¹¹ described a similar case of widespread erosion in a child of 8, however, enamel erosion has never been reported before in a 3-year-old child as the major presenting symptom of GER.

Parents will often fail to mention symptoms such as sleep problems or failure to gain weight, which could suggest a diagnosis of GER, thinking they are irrelevant to a dental problem. However, even in the absence of systemic symptoms, GER should be considered in the differential diagnosis of the preschool child with erosion lesions which cannot be ascribed to dietary sources. In the older child, a differential diagnosis for enamel erosion should include eating disorders such as anorexia nervosa, bulimia nervosa, and rumination.²³ A confirmatory diagnosis of GER is only possible, however, following a thorough diagnostic evaluation by a gastroenterologist. Dental erosion appears to be a more common finding in patients with GER than previously thought, and it has been suggested that dental erosion be considered an atypical manifestation of GER disease.⁹ Dental management should include dietary counselling, daily topical fluoride application, sodium bicarbonate rinses, fissure sealant placement, and restoration of severely affected teeth with adhesive restorations or crowns.

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