Dental caries and achalasia: A case report and review of the literature

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Citation

Abstract
Various pathologies caused in the oral cavity by achalasia and other diseases accompanied by achalasia have been reported in the literature. This case report is to present severe dental caries in a patient with idiopathic achalasia. A 16-year-old male patient with achalasia admitted with chief complaint about dental caries. Deep dentinal caries are noticed anterior and posterior regions in the vestibular surfaces of cervical regions in mandibula and maxilla. No erosion was observed. Deep dentinal cervical caries were restored with composite restorations and the patient followed up.

INTRODUCTION
Dental erosion is the loss of compact tissue taking place in the teeth as a result of the acidic effects of various diseases, including anorexia nervosa, bulimia nervosa, chronic alcoholism, gastro-esophageal reflux disease, and esophageal achalasia. Esophageal achalasia is a primary esophageal motility disorder developing secondary to autoimmune destruction of the myenteric plexus, resulting in failure of relaxation of the lower esophageal sphincter muscle in the cardia and aperistalsis in the smooth muscles of esophagus. It has an incidence varied from 0.03 to 1/100 000 people per year, and both sexes are equally affected. It has been also called as cardiac achalasia, cardiospasm or esophageal aperistalsis. It may develop primarily or secondarily. The primary type is idiopathic, whereas the secondary type, also known as pseudoachalasia, may develop in older ages secondary to neoplasias infiltrating the sphincter such as gastric, esophageal, pulmonary, pancreatic, hepatic, colonic and prostatic carcinomas, lymphomas, adenocarcinomas of the cardia, and mesotheliomas. Secondary achalasia has been reported to constitute 3% of all cases of achalasia. As a result of failure of relaxation in the muscle of cardia and esophageal aperistalsis, the nutrients fail to reach the stomach and cause dilatation of the esophageal walls in the upper part of the muscle, followed by digestive disorders.

The esophagus includes afferent fibers extending along the vagus and splanchnic nerves. Swallowing of the secreted saliva rinses the oral, pharyngeal and esophageal regions. Increased salivary secretion is a reflex developed towards protection of these regions. Likewise, esophageal problems including cancers, foreign bodies, and obstructive disorders such as achalasia, gastro-esophageal reflux disease, and inflammatory diseases such as varicella zoster and herpex infections also cause siaolorhea. Achalasia is one of the causes of obstructive dysphagia. When the esophagus is obstructed, it mechanically causes a retrograde flow or regurgitation of the saliva as a result of the esophago-salivary reflex. Prolonged obstructions result in widening of the esophageal walls and increased hypersalivation reflex. Dental erosions may develop as fermented nutrients return to the oral cavity. Other than this, achalasia lies among the risk factors of esophageal cancers.

The main clinical symptoms of achalasia are dysphagia, regurgitation, weight loss, retrosternal burning sensation, position-related nocturnal cough, obstruction of the respiratory tract, stridor, and chest pain radiating towards the back, chin, neck and arms. It may coexist with various syndromes such as Smith-Lemly-Opitz, Allgrove or 4A syndrome(alacrima, achalasia, autonomic disturbance, and ACTH insensitivity), triple-A (alacrima-achalasia-adrenal insufficiency), and Sjögren’s syndromes, and ectodermal dysplasia. Differential diagnosis of achalasia includes gastro-esophageal reflux disease, diffuse esophageal spasm, pancreatic cysts, and anorexia nervosa. Achalasia lies among the obstructive causes of esophageal disorders.
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Diagnosis and evaluation of severity of achalasia requires barium X-ray imaging, endoscopy, esophageal pH monitoring, manometry and computed tomography. Esophageal manometry is used for definitive diagnosis. 

The treatments of esophageal achalasia include medical treatment, endoscopic balloon dilatation, botulinum toxin injection, and Heller myotomy. Follow-up of the patients is particularly important regarding the risk of carcinomas. Currently, the most commonly preferred criterion of treatment in gastroenterology clinics endoscopic balloon dilatation.

CASE REPORT

A 16-year-old male patient was admitted to our clinic with the complaints of short-lasting pain in all of his teeth triggered by tooth brushing and cold stimuli. In his medical history, it has been learned that he had been diagnosed with Hodgkin’s lymphoma 10 years prior to his admission, received chemotherapy and radiation therapy, and the disease was in the state of remission. It has been also learned that the patient was diagnosed with achalasia 1 year prior to admission. A short time following the diagnosis of achalasia, the patient was referred to our clinic due to the observation of discoloration and cavitations in teeth bilaterally in both jaws. Extraorally, bilateral submandibular lymphadenopathies were observed. Intraoral examination revealed deep caries cavitations in the vestibular surfaces in the cervical regions of the teeth numbered 11, 12, 13, 14, 15, 16, 17, 21, 22, 23, 24, 25, 27, 33, 41, 42, 43, and 44, and plaques and calculus in the surfaces of all teeth (Figure 1). No erosions were found on the other surfaces of the teeth. Anamnesis of the patient revealed that he was fed with liquid nutrients with high carbohydrate content for months following the diagnosis of achalasia, and lacked the habit of plaque control. It has also been learned that he received botulinum toxin injections and underwent endoscopic balloon dilatation for the treatment of achalasia, and his disease remitted with treatment.

The caries lesions were restored following scaling and root planning in our clinic (Figure 2). The patient was educated for oral health and followed up.

DISCUSSION

In achalasia, pathologies such as aspiration pneumonia, bronchial asthma, acute respiratory tract obstruction, cricopharyngeal dysfunction, sialorrhea, and dental erosion as well as esophageal symptoms may be encountered. Halitosis is a frequently encountered symptom in achalasia and gastro-esophageal reflux disease. Besides, the tooth surface is exposed to acid attacks during regurgitation or only fermented foods, which is among the important symptoms of such diseases. Observation of erosion in teeth as a result of gastric acid rising up to the oral cavity aids the clinicians in diagnosis of latent reflux.

Crookes et al. has reported that the type of acid in achalasia is lactic acid. In case such diseases are not treated, it should be regarded that acidic attack shall persist.

Also, various pathologies caused in the oral cavity by other diseases accompanied by achalasia have been reported in the
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literature. Allgrove (4-A) syndrome is characterized by shrinkage and spasticity of the tongue, disruption of the gag reflex, paresthesias of the palatal region, and oral candidiasis. In triple A (achalasia-alacrima-adrenal insufficiency) syndrome, formation of dental caries can be accompanied by dental dysplasia. Also, dysplastic enamels, anterior cross-bite and superior micrognathia have been reported in a case of ectodermal dysplasia presenting with achalasia. Blam et al. have evaluated the diagnosis and severity of achalasia with radiographic findings, compared these findings with the symptoms of patients, and expressed that besides typical symptoms such as dysphagia, regurgitation, and chest pain, increased dental biofilm accumulation, nocturnal oral mucus discharge, and halitosis may also be atypically seen.

In the literature, few reports regarding the effects of achalasia on dental tissues have been published. It has been reported that dental erosions caused by fermented foods (not regurgitated gastric juice) appear in presence of achalasia, and these erosions have been reported to be on the palatal surfaces of anterior teeth. In this case, xerostomia might have been developed following radiotherapy that has been performed during the treatment of primary disease, and might have formed the basis for caries. However, the formation of dental caries was observed after achalasia, which had developed later, was diagnosed. Deep dentinal caries are noticed most commonly in the maxillary anterior and posterior regions and less commonly in the vestibular surfaces of cervical regions in the anterior mandibular teeth. No erosion compatible with the index described by Smith and Knight in which the teeth erosions were classified was observed. Rather than the formation of erosions, the formations and localizations of caries suggest that they arose from malnutrition and disorders of oral hygiene. In anorexia nervosa, dental erosions as well as nutrition with foods with high carbohydrate contents, ingestion of dextrose tablets or vitamin C with sucrose contents have been shown to be significant factors in development of the formation of caries. Despite the scarcity of studies regarding the association of achalasia with dental caries, the risks of dental erosions and caries have been reported to be high in gastro-esophageal reflux disease, which, like achalasia, is one of the most important causes of regurgitation. In other study pH monitoring were performed in achalasia and reflux disease in esophagus and acidic pH in achalasia was approximately pH: 4 and in reflux disease approximately pH: 3.

In another study in which bulimia nervosa has been investigated, bulimia-associated erosions have been reported to be on the palatal surfaces of all teeth in the upper jaw, while the erosions associated with chronic regurgitation were formed on the palatal surfaces of the molar teeth in the maxillae.

In our study, no erosion was formed on the palatal surfaces of teeth. Frequent recommendation of liquid diets with high carbohydrate contents in treatment of achalasia and lack of oral hygiene care habits might be regarded as significant causes of the formation of dental caries.

In conclusion, we suggest that direct effects of the acidic contents on the oral cavity in achalasia as well as malnutrition and failure of oral hygiene care and plaque control in these patients might be the significant factors for caries formation in these patients.

References

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