Review Article

Oral cavity and eating disorders: An insight to holistic health

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Abstract

Oral health is vital to the general well being and is a time-tested indicator of the systemic health of an individual. Oral cavity may be the primary site affected in endocrine disorders, renal disorders, gastrointestinal, cardiovascular, hematological, autoimmune cutaneous disorders, and psychosomatic disorders. Eating disorders (primarily Anorexia nervosa and bulimia) are psychosomatic disorders having multifaceted etiology, and characterized by abnormal eating patterns. In many cases, the oral cavity may be the only site of the manifestations of eating disorders. An oral physician may often unveil the mystery of this underlying systemic pathology by a vigilant and meticulous examination of the oral cavity. This not only helps in nabbing the disease in its early course but also prevents the patients from the appalling consequences due to the disease. This article aims to highlight the etiopathogenesis and various oral features in eating disorders. The oral physician should be familiar with the bizarre oral features of eating disorders and should work in close connection with other healthcare physicians to prevent the psychosomatic and systemic consequences.

Keywords: Anorexia nervosa, bulimia, eating disorders, oral manifestations

Introduction

Published literature has established an association between mental and general physical health, although the connection between oral and mental well being remains an unexplored subject.[1] Eating disorders (EDs) are psychosomatic ailments, associated with abnormal eating patterns, disrupted physical and psychosocial abilities, indigent life quality, suicidal tendencies, and potentially fatal systemic complications causing higher death rates.[2-4] EDs exhibits certain manifestations in the oral cavity, either directly or through nutritional deficiency. Tooth erosion; dental caries, qualitative and quantitative salivary changes (xerostomia, altered buffering capacity, and salivary pH), gingival and periodontal diseases, and oral mucosal lesions (palatal erythema and ulcers) constitutes the primary oral features of EDs.[5] Previously conducted studies have suggested that features of EDs are noticeable primarily within the oral cavity during the first 6 months of abnormal behavior.[6] Hence, early and accurate diagnosis of the condition is imperative, considering the associated psycho-somatic and oral health consequences.[7] EDs are now regarded as a social epidemic; AN and BN are

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considered as the commonest disorder among adolescents in western nations.[8]

Classification

Based on the diagnostic and statistical manual of mental disorders (DSM-5), EDs can be categorized into Anorexia Nervosa (AN), Bulimia Nervosa (BN), Binge Eating Disorder (BED), and Other Specified Feeding or Eating Disorders (OSFED).[5]

AN is identified primarily by weight loss and food limitation.[7] AN is an intricate disorder and leads to diminished self-esteem, and susceptibility to habit-forming behaviors (narcotics, alcohol, tobacco, sugar, sodas, endurance sports, etc.).[9] According to DSM-5, there are three diagnostic criteria for AN.[10] [Box 1] AN is further sub-classified into restrictive type; where abstinence/reduced feeding, and strenuous workouts result in weight loss or purgative type; where the weight loss is accomplished by induced vomiting, and the excessive intake of diuretics, laxatives, and/or appetizer's suppressors.[2]

BN is identified by first overeating followed by adopting incongruous compensative practices, viz. self-inflicting vomit, excessive intake of laxatives, and strenuous workouts.[7] Gag reflex is usually induced by fingers or sometimes using pencils/combs, leading to the development of callus on the dorsal aspects of the fingers (Russell's sign).[10] Box 2 defines the three diagnostic criteria for BN [Box 2].[11]

Box 1: Diagnostic criteria for anorexia nervosa

1. Restriction of energy intake relative to requirements leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. There is a refusal to maintain body weight ≥85% of that expected for the patient's age and height
2. An intense fear of gaining weight or becoming fat, despite underweight
3. A distorted view of one's body weight, size or shape exists. Also, there is undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.

Box 2: Diagnostic criteria for bulimia nervosa

1. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
   A) Eating in discrete time (for example, within 2 hours) an amount of food that is definitely larger than what most individuals would eat in a similar period under similar circumstances
   B) A sense of lack of control over eating during the episode (for example, a feeling that one cannot stop eating or control what or how much one is eating)
2. Recurrent inappropriate compensatory behaviours in order to prevent weight gain, such as self-induced vomiting; misuse of laxative; diuretics, or other medications; fasting; or excessive exercise
3. The binge eating and inappropriate compensatory behaviours both occur, on average, at least once a week for 3 months
4. Self-evaluation is unduly influenced by body shape and weight
5. The disturbance does not occur exclusively during episodes of anorexia nervosa

EDs have a gender predilection, as it is seen primarily in adolescent females, with a mean age of occurrence being 16 and 25 years for AN and BN, respectively. AN primarily affects the upper- and middle-class individuals, in contrast to BN, which affects individuals of all social-economic strata.[12] However, BED has a higher prevalence among elderly males.[5]

EDs are frequently accompanied by a wide array of comorbid conditions like cardiovascular diseases, endocrine system dysfunction, gastrointestinal, pulmonary, and musculoskeletal ailments. The impact of comorbid conditions is influenced by the patient's age, a period of irrational feeding, speed, and severity of weight loss.[13]

Table 1 depicts the various comorbidities associated with EDs.

Etio-Pathogenesis

The precise etiopathogenesis of EDs is obscure, although a wide array of factors (biological and socio-psychological factors) has an essential role play. However, the primary factor in the genesis and development of EDs remains indeterminate self-esteem and displeasure with bodily looks.[14] Some people may also have a genetic predisposition. To summarize, EDs are mostly the result of a snugly woven network of psychological factors, personality traits, and environmental factors, like a peer and parental pressure, child maltreatment, social isolation, and cultural differences.[15]

Table 2 summarizes the various etiological factors associated with EDs.

Eating Disorders and Oro-Dental Features

The earliest features of EDs are noticeable primarily in or around the oral cavity; hence, the oral health physicians may be the first to come across undiagnosed patients with EDs.[13]

The oral signs and symptoms of EDs are usually seen either due to nutrition deficits or due to the long-standing history of self-induced vomiting. However, improper personal hygiene, incongruous eating patterns, and particular medications may worsen the condition.[16]

The effects of EDs on oral health were reported by Hellstrom and Hurst *et al*. Three main types of oral pathologies are seen: (a) Dental erosion or pathological tooth loss. (b) Dental caries caused due to microbial action. Dental plaque microbes produce organic acids; thus ensuing the demineralization of enamel and dentin. (c) Chronic self-induced vomiting or protracted fasting may result in decreased salivary production. Certain antidepressant medications may further exaggerate these effects.[1]

Figure 1 depicts the oral features in EDs.

Dental erosion

Published literature has established dental erosion as the most salient and commonest oral feature of EDs.[17] Dental erosion

Table 1: Medical alterations in eating disorders

PARAMETRES	ANOREXIA NERVOSA (AN)	BULLIMIA NERVOSA (BN)
Physical examination findings	Dry skin, lanugo hair formation (only with severe weight loss), acrocyanosis, alopecia, low body temperature, dehydration, retardation of growth and pubertal development	Erosion of dental enamel, parotid/salivary gland enlargement (Sialadenosis), Callus/scars on the skin of the back of the hand resulting from inducing the gag reflex (Russel's sign), dehydration
Cardiovascular system	Bradycardia, hypotension ECG abnormalities (mostly prolonged QT-interval), pericardial effusion, edema (before or during refeeding)	ECG-abnormalities (cardiac arrhythmia, prolonged QT-interval)
Endocrine	Ovarian changes Ammenorhea Reduced estrogen and progesterone Increased cortisol and growth hormone	Decreased Estradiol and Leptin levels
CNS	Cerebral changes Epileptic fits	Cognitive damage Tetany
Musculo-skeletal	Osteoporosis Pathological fractures	Myopathy
Gastrointestinal system	Impaired gastric emptying, pancreatitis, constipation Leukocytopenia, thrombocytopenia,	Esophagitis, pancreatitis, delayed gastric emptying
Blood	Thrombocytopenia, anemia, Hypokalemia, hyponatremia, hypomagnesiemia, hypocalcemia, hypophosphatemia (during refeeding), low glucose levels, AST↑, ALT↑ (with severe fasting or beginning of refeeding), cholesterol ↑	Hypokalemia, hyponatremia, hypomagnesiemia (caused by diarrhea), hypocalcemia, metabolic alkalosis (in case of severe purging), metabolic acidosis (in case of severe laxative abuse)

Table 2: Etiological factors associated with eating disorders

Biological	Genetic predisposition, epigenetic mechanism, impaired fat metabolism, irregularities of hypothalamus-pituitary-adrenal axis, elevated levels of auto antibodies, brain calcifications, lesions of the temporal or frontal lobe of brain.
Psychological	Psychoneurosis, morbid fear of obesity, depression, schizophrenia, poor self-esteem, anxiety, loneliness, obsessive-compulsive disorders, borderline personality disorders, attention deficit hyperactivity disorders, narcissistic, histrionic or avoidant personalities, perfectionist personality body dysmorphic disorders
Socio-cultural factors	Physical, sexual, verbal abuse, childhood negligence bulling and social isolation, peer pressure, mass media influence
Demographic	Females, higher or middle economic class
Personal habits	Drug abuse, alcoholism, consumption of non-nutrition items
Family	High familial expectations/dysfunctions, family history of psychoneurosis, positive family history
Occupation	Models, actors, dancers, athletes, celebrities

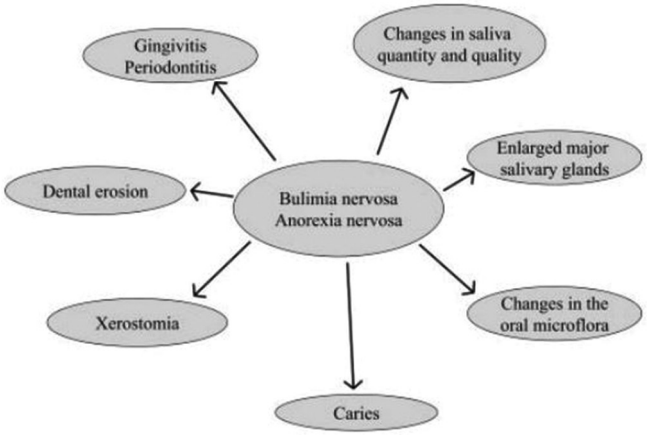

Figure 1: Possible oral features in eating disorders

is described as gradual, irreversible destruction of the calcified tooth framework, caused by non-bacterial chemical action. Initially, it appears as soft smooth-shining glossy tooth surfaces; gradually progressing to flat or superficial, flat indentations, coronal to the cementoenamel junction. Advanced lesions appear as cup-shaped defects on the occlusal/incisal aspects of the tooth, with markedly altered tooth morphology.[2] Holst and Lange (1939) coined the term 'perimylolysis' and refers to palatal erosions on the maxillary teeth.[12]The mandibular lingual tooth surfaces are usually spared as they are shielded by the tongue and saliva from sublingual and submandibular glands[18]

Figure 2 Perimylolysis presenting as palatal erosion on the maxillary teeth.

The origin of acid produced dictates the site predilection, with intrinsic (gastric) and extrinsic (dietary) acid causing palatal and labial erosion, respectively. It is also essential to delineate the erosion pattern due to BN and gastroesophageal reflux, with the former affecting the mandibular anterior lingual tooth surfaces and the latter affecting the occlusal and lingual aspects of the posterior teeth.[16]

Patients with tooth erosion are more prone to dentinal sensitivity. The interplay between the intensity of the tooth erosion and

sensitivity has further reinforced the vomit-associated reflux effect as the primary cause.[2] Nevertheless, eds cannot be considered as the sole etiology for hypersensitivity and may occur secondary to palatal/cervical erosion, or dental caries as periomylolysis.[15] Dentin hypersensitivity is described as a sharp, fleeting, and localized dentinal pain due to a thermal/chemical trigger. Hypersensitivity usually occurs at the cervical region owing to the thinnest enamel layer; thus unveiling the dentin and dentinal tubules.[18]

Dental caries

Dental caries is one of the important features in EDs. Dental caries usually has a multifactorial etiology; hence, its occurrence cannot be exclusively attributed to EDs.[15]

Personal oral hygiene, genetic tendency, malnutrition, dietary cariogenicity, fluoride exposure during odontogenesis, and specific drug intake usually accounts for the differences in the caries prevalence in EDs. The carious lesions seen in patients with EDs exhibit the following characteristics: (a) preponderance to cervical caries and (b) leathery dentinal lesion with large areas of undermined enamel.[19]

The chronic self-inflicted vomits, excessive intake of the laxatives, diuretics, and/or appetite suppressors, and strenuous workouts usually provoke unremitting dehydration. This, in turn, imparts a negative influence on the volume of salivary production and secretion. The intake of antidepressants as a therapeutic regimen for eating disorders may further induce a xerostomic effect, thus, worsening the scenario. The interrelation between the increased salivary viscosity and a decreased buffering capacity may culminate in an acidic salivary pH, thus, serving as a contributing factor for demineralization and decay of the tooth structure.[2]

Salivary glands

Sialadenosis is regarded as a chronic presentation in patients with EDs, more commonly seen in patients with Bulimia.[16]

Sialadenosis is frequently described as a relapsing, bilateral, asymptomatic, non-inflammatory, non- neoplastic salivary gland enlargement, and does not affect the gland functioning.[20]

Lavender (1969) first suggested the association between chronic vomiting and bilateral parotid enlargement and demonstrated that bilateral parotid enlacement may sometimes the first noticeable feature of bulimia.[21] Five studies have demonstrated the enlargement of parotid glands among the bulimic subjects.[7,15,22-24]

Peripheral autonomic neuropathy appears to be the main reason for sialadenosis; thus resulting an increased acinar protein production and/or an interrupted granular release. Zymogen granules accumulation in the acinar cells causes parotid hypertrophy and impaired salivary secretion.[25]A study by Donath *et al.* demonstrated that the salivary synthesis and secretion is controlled by myoepithelial cells and postganglionic sympathetic neurons. The degenerative alteration in these regulatory cells is believed to be the cause of sialadenosis.[26,27]

The disorder is mostly seen in patients with BN who expel by vomiting.[28] Generally, the salivary swellings are soft and asymptomatic,[29,30] although rarely painful swellings are also seen.[29] Painful swellings are associated with different etiology.[31]

Figure 3 Sialadenosis in bulimia nervosa.

Necrotizing sialometaplasia is regarded as an acute presentation of EDs, primarily in bulimia.[32] The first documentation of necrotizing sialometaplasia (NS) was made by Abraham *et al.* NS is defined as an inflammatory, self-limiting, necrotizing pathology and primarily affects the minor salivary glands of the hard palate.[33]

NS is regarded to arise due to ischemic necrosis of the salivary gland lobules, although, the exact etiopathogenesis is not fully elucidated. Habits (smoking and alcohol), trauma (ill-fitting denture, recent surgery, and tooth extractions), pulmonary and systemic ailments, bulimia, and anorexia may serve as potential

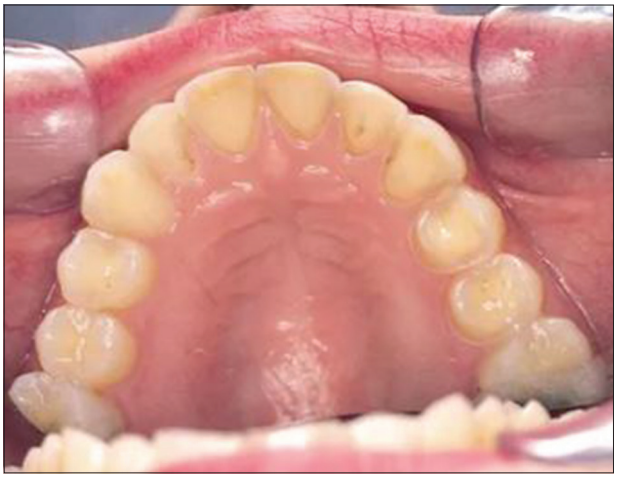

Figure 2: Erosion on the palatal aspect of maxillary teeth

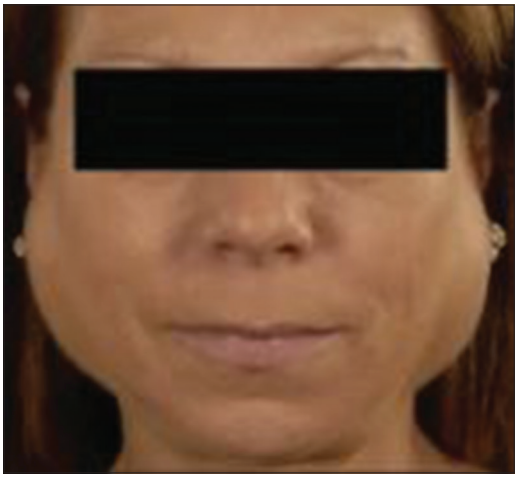

Figure 3: Bilateral Parotid swelling in bulimia

predisposing factors.[34-36] Schoning *et al.* reported two cases of NS in BN subjects.[37]

Figure 4 Necrotizing sialometaplasia in bulimia nervosa.

The patients with ED may exhibit quantitative/qualitative alterations in saliva, that is, altered salivary flow rates or altered salivary composition or both.[23,28,30,38-43] Metabolic acidosis/alkalosis seen in the patients with ED could be recognized as an indirect effect of the altered biochemical salivary composition.[8]

Xerostomia refers to the subjective perception of dryness of mouth and is defined as a deficiency in the amount of salivary production and the buffering capacity. Hyposalivation is usually seen in anorexic patients during protracted starvation compared to the binge eating patterns in bulimia.[18]

Published literature has demonstrated the effect of EDs on the salivary flow rates.[23,39,41,44] Predisposition to increased caries activity, opportunistic mucosal lesions (candidal infections), and taste alterations have been established with decreased salivary flow rates.

The patients with BN have been comprehensively evaluated for the qualitative salivary content .[41-44] According to a study by Riad *et al.*,[41] significantly greater levels of amylase were reported in both unstimulated and stimulated salivary samples of the patients with BN. However, a study by Tylenda *et al.*[42] reported an insignificant difference in the salivary composition of bulimic and control patients. The study also suggested that the oral changes in EDs result primarily due to cariogenic dietary patterns and binging/purging habits and not due to physiologic salivary alterations.

It is essential to maintain the critical salivary pH as acidic pH causes enamel demineralization and promotes the survival of acidogenic and cariogenic microbes (e.g., S. mutans, and Candida).[17]

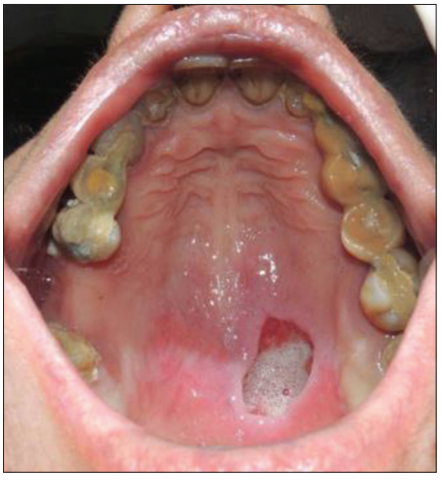

Figure 4: Necrotizing sialometaplasia presenting as ulcer on the posterolateral aspect of palate

Research has reported elevated amylase levels in 25%-60% of bulimic patients.[29,45] Serum amylase levels has attracted significant attention due to their diagnostic and regulatory role.[29,30,40,44-47]

Eating disorders and oral function

Owing to prolonged gastric purging, bulimic patients undergo the desensitization process and become habituated to suppress the most strenuous self-inflicted pharyngeal stimulus.[44]

Gustometric studies have revealed taste alterations and hypogeusia in AN and BN subjects,[48] thus promoting self-starvation in such patients. There exists a wide array of factors affecting the taste perception in the patients with ED. For example, diminished fungi form tongue papillae in anorexia nervosa and palatal abnormalities due to chronic self-induced vomiting in bulimic patients.[49] However, these findings were refuted by other studies.[50,51] Patients with ED may also present with dysphagia (diffculty in swallowing). Johansson *et al.* reported the occurrence of a throat lump (Globus sensation) in these individuals.[7]

Obsessive-compulsive disorders in the form of compelling repetitive tooth brushing, a focus on hygiene, chewing gum intake, and parafunctional habit of nail-biting (onychophagy) causing tooth attrition are also associated with EDs.[9]

Gingivitis and periodontitis

The patients with ED lack meticulous oral hygiene; hence, they are more vulnerable to gingivitis and periodontitis. Vitamin C deficiency may result in marginal gingivitis.[10] Gingival recession occurs mainly in adult patients, either due to traumatic brushing or by constant acid attacks.[9] However, cases where gingivitis and periodontitis occur in young adolescents, a differential diagnosis of eating disorders should always be given consideration.[16] Periodontal health may further be affected by micronutrient deficit (e.g., zinc, iron, selenium, calcium, copper, and magnesium).[2]

Oral mucosal lesions

Numerous oral mucosal entities arise due to dietary imbalances (vitamin and hematinic); thus hindering the revival and regeneration of the oral mucosa.[17] Erythematous palate and trauma-induced ulcerations of the soft palate and pharynx are usually seen in bulimics due to chronic acidic contact and the repeated digital trauma.

Angular cheilitis/Cheilosis is primarily a feature of BN, where the angles of the mouth typically appear pale and macerated.[16] Angular cheilitis is primarily due to chronic candidal infection, although, may also occur as a result of a coexisting staphylococcus infection.[52]

Burning sensation or stomatodynia is also seen in EDs. The patients report with the perception of burning/stinging of the tongue or oral cavity, despite the non significant

clinico-pathological findings. Burning sensations may occur secondary to the underlying psychological (anxiety, depression, and stress) and neurological disorders. Oral mucosa atrophy, owing to nutritional deficiencies and chronic vomiting may also serve as a contributory factor to the burning sensations in such patients.[2]

Oral microflora

Owing to chronic self-induced vomiting resulting in acid regurgitation, anorexic and bulimic patient's saliva have an increased concentration of acidogenic and cariogenic microbes.[53] Furthermore, a viscous salivary flow rate with its low pH may facilitate the growth of aciduric oral microbes.[54] Studies have demonstrated that increased salivary loads of S. sobrinus are congruous in bulimics; thus, suggesting the significance of S. sobrinus salivary culture as a diagnostic aid in patients with bulimia.[53]

Table 3 summarizes the common oral features of EDs.

The primary healthcare professional plays a role in the delineation of in the identification of the varied patterns of disordered eating. The primary physician must be familiar with the various risk factors and oral presentation of EDs and should be able to provide initial intervention in such patients. The primary physician should be efficient in making decisions and be able to differentiate the patients warranting urgent hospitalization; thus, directly influencing the patient care and outcomes. Also, the primary healthcare professional is often a member of a multidisciplinary team effort including a physician, nutritionist, dentist and psychiatrist.[55]

Numerous individual remedial measures (e.g., attitude modification, psychotherapy, pharmacological therapy, and family therapy) are proposed for EDs. Hence, a multidisciplinary therapeutic protocol beneficial to the varied morbid states may be implemented. However, the precise therapeutic regimen still needs to be accomplished.[56]

Conclusion

EDs are typified by abnormal eating patterns and altered body weight and usually linked with numerous psycho-somatic sequelae, leading to impecunious quality of life and higher death rates.[57] These subjects have an increased propensity to develop poor oral health. A snugly woven network of deleterious cariogenic diet, self-inflicted vomit, impaired altered salivary composition and poor oral hygiene usually predispose for some oral disorders such as tooth erosion, dental caries and dry mouth.[58] The primary healthcare physician cannot only serve as a portal to specialized medical care but also has a pivotal function in motivating both the individual and the peer group for a speedy recovery.[59] The specific course of therapy for EDs still needs to be accomplished, owing to the plethora of etiologic factors.[60] However, the associated complications like electrolyte imbalance with dehydration, hypotension, bradycardia, arrhythmias, and suicide tendencies may warrant urgent hospitalization.[61]

Table 3: Summary of oral features in eating disorders

Oral Tissue	Manifestation	Causes
Dentition	Enamel erosion, perimolysis (dental erosion on the palatal surfaces of teeth), tooth sensitivity	Vomiting, salivary gland manifestations of ED affecting salivary flow rate, buffering capacity and pH of saliva resulting in erosion.
	Caries	Poor oral hygiene, excessive consumption of carbonated drinks, sweets, caffeinated drinks or sports drinks for stamina.
Oral mucosa	Mucosal atrophy, glossitis, oral ulcerations, erythematous lesions of the soft palate	Nutritional deficiency including iron and vitamin deficiency
	Erythematous lesions of the soft palate and pharynx	Trauma caused by inserting foreign objects into the oral cavity to induce vomiting
	Candidiasis	Opportunistic infection by Candida albicans due to nutritional deficiencies, salivary dysfunction, secondary infection of mucosal lesions induced by trauma
	Angular cheilitis	Nutritional deficiency, candidal infection or concomitant candidal and staphylococcal flora
Periodontal and gingival tissues	Gingivitis, periodontitis, scurvy, advanced periodontitis in young individuals	Poor oral hygiene, vitamin C deficiency
Salivary glands	Sialadenosis, non-inflammatory enlargement of salivary gland	Peripheral autonaumic neuropathy
	Hyposalivation, xerostomia, altered salivary flow rate, buffering capacity, pH and composition of saliva. Necrotising sialometaplasia	Side effects of drugs such as antidepressants, vomiting, nutritional deficiency.
Alveolar Bone	Osteopenia, osteoporosis	Nutritional deficiency, infection of dental or periodontal origin causing quicker alveolar bone loss
Tongue	Glossodynia, altered taste, loss of taste (dysgeusia), hyposgeusia, burning sensation	Avitaminosis and trace metal deficiencies particularly zinc, somatoform disturbances and mucosal atrophy

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Conflicts of interest

There are no conflicts of interest.

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