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. 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. 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. Previously conducted studies have suggested that features of EDs are noticeable primarily within the oral cavity during the first 6 months of abnormal behavior. Hence, early and accurate diagnosis of the condition is imperative, considering the associated psycho-somatic and oral health consequences. EDs are now regarded as a social epidemic; AN and BN are

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

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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).[9]

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.[19] [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.[3]

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).[19] Box 2 defines the three diagnostic criteria for BN [Box 2].[11]

Table 1 depicts the various comorbidities associated with EDs.

Etiopathogenesis

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 snuggly woven network of psychological factors, personality traits, and environmental factors, like a peer and parental pressure, child maltreatment, social isolation, and cultural differences.[19]

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.[8]

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.[11]

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.[7] Dental erosion
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.\(^\text{[10]}\)

Patients with tooth erosion are more prone to dentinal sensitivity. The interplay between the intensity of the tooth erosion and
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Sensitivity has further reinforced the vomit-associated reflux effect as the primary cause.[5] Nevertheless, EDs cannot be considered as the sole etiology for hypersensitivity and may occur secondary to palatal/cervical erosion, or dental caries as periomylolysis.[13] 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.[19]

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.[13]

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 frequently described as a relapsing, bilateral, asymptomatic, non-inflammatory, non-neoplastic salivary gland enlargement, and does not affect the gland functioning.[24]

Lavender (1969) first suggested the association between chronic vomiting and bilateral parotid enlargement and demonstrated that bilateral parotid enlargement 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.[28] 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.[28] 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. Schoning et al. reported two cases of NS in BN subjects.

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. Serum amylase levels has attracted significant attention due to their diagnostic and regulatory role.

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.

Gustometric studies have revealed taste alterations and hypogeusia in AN and BN subjects, 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. However, these findings were refuted by other studies. Patients with ED may also present with dysphagia (difficulty in swallowing). Johansson et al. reported the occurrence of a throat lump (Globus sensation) in these individuals.

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.

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. Gingival recession occurs mainly in adult patients, either due to traumatic brushing or by constant acid attacks. However, cases where gingivitis and periodontitis occur in young adolescents, a differential diagnosis of eating disorders should always be given consideration. Periodontal health may further be affected by micronutrient deficit (e.g., zinc, iron, selenium, calcium, copper, and magnesium).

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. 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. Angular cheilitis is primarily due to chronic candidal infection, although, may also occur as a result of a coexisting staphylococcus infection.

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.\[3\] Furthermore, a viscous salivary flow rate with its low pH may facilitate the growth of acidic oral microbes.\[4\] 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.\[5\]

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.\[5\]

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.\[6\]

**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.\[7\] 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.\[8\] 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.\[9\] The specific course of therapy for EDs still needs to be accomplished, owing to the plethora of etiologic factors.\[9\] However, the associated complications like electrolyte imbalance with dehydration, hypotension, bradycardia, arrhythmias, and suicide tendencies may warrant urgent hospitalization.\[6\]

| 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, Poor oral hygiene, excessive consumption of carbonated drinks, sweets, caffeinated drinks or sports drinks for stamina. |
|                             | Caries                                                                       | Nutritional deficiency including iron and vitamin deficiency          |
| Oral mucosa                 | Mucosal atrophy, glossitis, oral ulcerations, erythematous lesions of the soft palate | Trauma caused by inserting foreign objects into the oral cavity to induce vomiting, Opportunistic infection by Candida albicans due to nutritional deficiencies, salivary dysfunction, secondary infection of mucosal lesions induced by trauma. |
|                             | Erythematous lesions of the soft palate and pharynx                         | Nutritional deficiency, candidal infection or concomitant candidal and staphylococcal flora |
|                             | Candidiasis                                                                  |                          |
|                             | Angular cheilitis                                                           |                          |
| 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 autonamic 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 (dygeusia), hyposgeusia, burning sensation | Avitaminosis and trace metal deficiencies particularly zinc, somatoform disturbances and mucosal atrophy |

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**Table 3: Summary of oral features in eating disorders**
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