Review Article

Oral implications of eating disorders: a review

Stuti Bhargavaa*, Mukta Bhagwandas Motwanib, Vinod Patnib

a Department of Oral Medicine and Radiology, Swargiya Dadasaheb Kalmegh Smruti Dental College and Hospital, Wanadongari-Wadhamna Road, Hingna, Nagpur 440016, b Vidhya Shikshan Prasark Mandal's Dental College and Research Centre, Hingna, Nagpur 440019, Maharashtra, India.

* Corresponding author: drstutibhargava@gmail.com

Revised edition: 20/09/2012. Accepted: 13/01/2013. Published online: 13/01/2013.

Abstract  Eating disorders (ED) are psychopathological conditions where patient demonstrates abnormal, distorted or chaotic eating behaviours and diet patterns which can deteriorate an individual's physical and emotional wellbeing. Manifestations of ED range from disruption of normal lifestyle to generalised weakness to even life threatening complications. Oral health care providers can be the first to notice the presence of previously undiagnosed eating disorders from the typical oral manifestations of the condition and instigate the multidisciplinary treatment required. However, there is a general lack of knowledge and awareness about the role of oral health care practitioners in the diagnosis, intervention and treatment of affected patients. This article reviews the recent literature on eating disorders and their subsequent oral manifestations. The authors have made an effort to highlight the information which will aid the oral healthcare professionals with diagnosis, treatment and rehabilitation of these disorders.

Keywords: Anorexia nervosa, binge-eating disorder, bulimia nervosa, pica.

Introduction

Eating disorders (ED) have psychopathological etiological factors where patient demonstrates abnormal diet patterns; manifesting through distorted or chaotic eating behaviours (Lo Russo et al., 2008). These can lead to poor quality of life by deteriorating an individual's physical and emotional well being. Other implications range from disruption of normal lifestyle to generalised weaknesses to even life threatening complications. Earlier findings suggested that females, adolescents and young adults in the Western countries were at the highest risk of developing these lifestyle disorders. However, recently, these disorders are showing a rise in numbers all over the world among both women and men, which can be attributed to cultural and peer pressures to attain an ideal body type under the influence of mass media and globalisation (Lucas et al., 1991; Carlat and Camargo, 1991; Patrick, 2002).

Oral health mirrors general health. Numerous systemic disorders show oral and maxillofacial manifestations which often are the first and definitive signs of these conditions. The oral health care practitioner’s role in the screening and diagnosis of such systemic diseases by their oral presentations are well established (Bouquot and Seime, 1997; DeBate et al., 2005; Little, 2002). The earliest manifestations of aberrant eating patterns and disorders have also been shown to appear in the oral cavity first, these include the deterioration of oral aesthetics, discomfort and pain in oral structures and impairment of oral functions (Lo Russo et al., 2008). With the rising numbers of these conditions, the possibility of a dental practitioner encountering an eating disordered individual is quite high. Oral health care
workers can be the first to notice the presence of previously undiagnosed eating disorders from the distinctive oral manifestations of the condition. But despite the fundamentally important role played by oral health practitioners in the early identification, referral, treatment planning and rehabilitation of ED patients; various studies have shown that the dental fraternity demonstrates limited knowledge of eating disorders and the subsequent oromaxillofacial and physical complications (DeBate et al., 2005; Harwood and Newton, 1995). A comprehensive review of literature on eating disorders highlighting the role of dentist in identification, management and complete rehabilitation of the patient is presented here.

Aetiology

The key point in pathogenesis of eating disorders is the distorted self perception of and resultant dissatisfaction with physical appearance of one’s body. This is triggered by a combination of biological, psychological and cultural factors.

Biological causes

These include genetic predisposition for development of ED (Klump et al., 2001). Gene expressions are altered by environmental factors without changing the underlying DNA sequence (Frieling et al., 2010). Eating behaviour is regulated by the hypothalamus-pituitary-adrenal axis. Irregularities in production, transmission of hormones and neurotransmitters such as serotonin, leptin and norepinephrine which are regulated from this axis, manifest in ED (Gendall et al., 1999; Licinio et al., 1996). Others include conditions such as brain calcifications (Conrad et al., 2008) elevated levels of auto-antibodies (Fetissov et al., 2005; Sinno et al., 2009) and lesions of temporal or frontal lobe (Houy et al., 2007; Uher and Treasure, 2005).

Psychological factors

Psychological stress and abnormalities commonly develop in individuals with high familial expectations, dysfunctional families, family history of psychoneurosis, personality traits such as a perfectionist, competitive personality, narcissistic, histrionic or avoidant personalities, emotional states of anxiety, depression and loneliness (Lucka and Cebella, 2004). Also individuals with psychological conditions like morbid fear of obesity, schizophrenia, obsessive compulsive disorders (Biederman et al., 2007), borderline personality disorders, attention deficit hyperactivity disorders (Cortese et al., 2007), body dysmorphic disorders (Gabbay et al., 2003); that is, having faulty self perception of body image, show more predilections for developing perturbed, chaotic eating patterns such as recurrent binge eating or food abstention. Most of these individuals have poor self-esteem; difficulty with self-regulation and try to control their problems by manipulating their diet (Lo Russo et al., 2008).

Socio-cultural factors

Traumatic personal experiences like physical, sexual or verbal abuse, childhood negligence, bullying and social isolation have a detrimental effect on individual’s physical, emotional health and psyche. Though traditionally associated with women, risk of development of eating disorders is commonly found both in men and women actively participating in performance arts and sports requiring weight regulation. Participants of sporting activities such as cheerleading, dancing, distance running, diving, figure skating, gymnastics, horse racing, rowing and swimming tend to regulate their diet for achieving lesser body weight whereas bodybuilders and wrestlers work to attain heavier bodies. Peer pressure, parental persuasion, cultural pressure for slimness and mass media influence for attaining ideal body type can lead to disruption of normal eating pattern (Committee on Sports Medicine Fitness, 1996; Glazer et al., 2008). Risk factors associated with development of eating disorders are summarised in Table 1.
Table 1  Risk factors associated with development of eating disorders. Individuals with a positive history of mentioned factors are at an elevated risk of developing chaotic eating patterns and subsequent eating disorder

| Biological: | Genetic predisposition, epigenetic mechanism, impaired fat metabolism, irregularities of hypothalamus-pituitary-adrenal axis, elevated levels of auto antibodies, Brain calcifications, lesions of temporal or frontal lobe of brain. |
| Psychological: | Psychoneurosis, morbid fear of obesity, depression, schizophrenia, poor self-esteem, anxiety, loneliness, obsessive compulsive disorders, border-line personality disorders, attention deficit hyperactivity disorders, narcissistic, histrionic or avoidant personalities, perfectionist personality body dysmorphic disorders. |
| Sociocultural 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. |

Types of eating disorders and their diagnostic criteria

The Diagnostic and Statistical Manual (DSM) classifies ED as anorexia nervosa (AN), bulimia nervosa (BN) and eating disorders not otherwise specified (EDNOS) (American Psychiatric Association, 1995, 2000); whereas the International Classification of Diseases (ICD) also adds vomiting associated with other psychological conditions and psychogenic loss of appetite in its classification (World Health Organisation, 1992). EDNOS is the variant of the disorder with limited symptoms; where the patient shows most of but not all of the diagnostic criteria. Both ICD and DSM cite similar AN diagnostic criteria for post-pubertal individuals; underweight (body weight maintained at less than 85% expected for given age and height), endocrine disorder typified by amenorrhea and a distorted body image. Other diagnostic features are self-induced weight loss achieved by food avoidance and vomiting or purging, amenorrhea for a minimum of three consecutive menstrual cycles and an intense fear of weight gain (Aranha et al., 2008; American Psychiatric Association, 1995; Frydrych et al., 2005; World Health Organisation, 1992).

The essential features to qualify for specific diagnosis of bulimia nervosa are persistent preoccupation with eating, binge eating, a morbid dread of weight gain and inappropriate compensatory methods to prevent weight gain occurring at least twice a week for three months. These methods include self-induced vomiting, excessive exercise and laxative abuse (American Psychiatric Association, 1995; World Health Organisation, 1992). DSM classification excludes the diagnosis of BN in the presence of AN. AN usually begins around puberty or in the 20s with a mean age of onset at 17 years and rarely after 40 years. Other findings include anaemia, thrombocytopenia, leucopenia, bradycardia, cold intolerance, hypothermia and hypotension. Digestive and excretory systems show delayed gastric emptying, poor motility, malabsorption, electrolyte abnormalities, and reduced glomerular filtration. Skin and hair changes include alopecia, loss of head hair, lanugo, hypertrichosis, carotenemia, and nail frailty (Little, 2002; Milosevic, 1999).
Oral manifestations of eating disorders

Oral manifestations occurring in ED are mainly due to metabolic impairment induced by nutritional deficiencies. The other reasons for oral manifestations of ED are neglect of personal hygiene and care activities (like brushing the teeth, bathing by psychologically disturbed individuals), modified dietary habits (cravings and excessive intake of particular food articles, certain drugs), and unhealthy compensatory methods for weight maintenance (such as induced vomiting). Oro-dental problems associated with ED can occur as early as 6 months following consistent disordered eating behaviours such as caloric restriction and vomiting (DeBate et al., 2005). Most common oral manifestations of eating disorders are summarised in Table 2.

Impaired oral functions and resulting symptoms

Oral cavity is multifunctional; it performs essential activities such as deglutition, mastication, speech and taste perception. Implications of ED result in impairment of these functions and symptoms such as dental sensitivity, oral pain, burning sensation, bleeding gums, tooth mobility and halitosis; causing discomfort and deterioration of oral and paraoral functions and aesthetics and further worsening the poor quality of life of ED sufferers.

Identification, intervention and treatment

Dentists may be the first health care workers to notice the early symptoms of a previously undiagnosed case of ED by its oral presentation and can play a pivotal role in deterring the development of a full-blown disorder through early identification, referral and treatment (DeBate et al., 2005; Schmidt and Treasure, 1997; Studen-Pavlovich and Elliot, 2001). The oral and physical manifestations of ED follow a consistent pattern. However, these must be differentiated from similar lesions (e.g. erosions) due to conditions other than ED. Biased perceptions with regard to patient susceptibility may inhibit accurate aetiological assessment of the underlying condition; thus stressing on fundamental importance of dental practitioners knowledge and skill to identify signs and symptoms of ED. Patients with ED are usually psychologically stressed with compromised general health and along with typical oral manifestations; they also show systemic manifestations such as fatigue, thinness, generalised weakness due to denial of hunger or ritualised exercising (Little, 2002; Milosevic, 1999). Interventions for patients exhibiting ED symptoms are particularly challenging and the topic requires sensitive approach by the clinician. Direct reference to the condition or use of terms such as “eating disorder,” “anorexia,” or “bulimia” can elicit a negative response as most of these patients respond in denial. Burkhart et al. (2005) suggested an interventional dialogue which can be used in the dental clinic set up. Once established as an eating disorder, compassionate behaviour, careful approach and judicious multispecialty referral is expected from dentists.

A most prudent first step of multidisciplinary treatment includes referral to the patient’s general physician or to a health professional specializing in treating ED. A patient with ED cannot be treated completely unless general health of the patient improves along with weight gain. Underlying depression, psychosis, weight loss and other systemic problems must be given priority for treatment which in severe cases may become life threatening. ED relapse occurs in two-thirds of treated patients within 18 months of recovery (Foster, 1998) resulting in probable re-emergence of oral problems or failure of conventional treatment modalities because of unresolved underlying aetiology.

The dental practitioner can provide pre-restorative care, protection of exposed dental faces, desensitization of exposed dentin, decreasing solubility of enamel and dentin (Kleier et al., 1984). The secondary prevention provided by the dental practitioner aims at decreasing the potential for further damage to the teeth and oral cavity as well as improving oral health. Acute conditions of pain, infection and symptom relief require immediate
Table 2  Common oral manifestations of eating disorders

<table>
<thead>
<tr>
<th>Oral Tissue</th>
<th>Manifestation</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Caries</td>
<td>Poor oral hygiene, excessive consumption of carbonated drinks, sweets, caffeinated drinks or sports drinks for stamina.</td>
</tr>
<tr>
<td><strong>Oral mucosa</strong></td>
<td>Mucosal atrophy, glossitis, oral ulcerations, erythematous lesions of the soft palate</td>
<td>Nutritional deficiency including iron and vitamin deficiency.</td>
</tr>
<tr>
<td></td>
<td>Erythematous lesions of the soft palate and pharynx</td>
<td>Trauma caused by inserting foreign objects into the oral cavity to induce vomiting.</td>
</tr>
<tr>
<td></td>
<td>Candidiasis</td>
<td>Opportunistic infection by Candida albicans due to nutritional deficiencies, salivary dysfunction, secondary infection of mucosal lesions induced by trauma.</td>
</tr>
<tr>
<td></td>
<td>Angular cheilitis</td>
<td>Nutritional deficiency, candidal infection or concomitant candidal and staphylococcal flora.</td>
</tr>
<tr>
<td><strong>Periodontal and gingival tissues</strong></td>
<td>Gingivitis, periodontitis, scurvy, advanced periodontitis in young individuals</td>
<td>Poor oral hygiene, vitamin C deficiency.</td>
</tr>
<tr>
<td><strong>Salivary glands</strong></td>
<td>Sialadenosis, non inflammatory enlargement of salivary gland</td>
<td>Peripheral autonomic neuropathy.</td>
</tr>
<tr>
<td></td>
<td>Hyposalivation, xerostomia, altered salivary flow rate, buffering capacity, pH and composition of saliva. Necrotising sialometaplasia</td>
<td>Side effects of drugs such as anti-depressants, vomiting, nutritional deficiency.</td>
</tr>
<tr>
<td><strong>Alveolar bone</strong></td>
<td>Osteopenia, osteoporosis</td>
<td>Nutritional deficiency, infection of dental or periodontal origin causing quicker alveolar bone loss.</td>
</tr>
<tr>
<td><strong>Tongue</strong></td>
<td>Glossodynia, taste impairment, dysgeusia, hyposgeusia, burning sensation</td>
<td>Trace metal deficiencies particularly zinc, somatoform disturbances and mucosal atrophy.</td>
</tr>
</tbody>
</table>

Treatment. Caries and periodontal disease could be managed as usual; however, timing of restorative intervention for eroded teeth is much debated, as some dentists believe that for restoration of the worn surfaces, vomiting should be controlled, preferably beforehand. There is no definite contraindication to restoring eroded surfaces other than the continued acid dissolution of tooth substances from around the restoration (Milosevic, 1999). Also, restoring aesthetics can be done in the recovery stage as in the active phase of disorder; patient may not comply with treatment or can be highly unsatisfied with the results due to disturbed psychological state. Use of composite restoratives that are not acid soluble should be preferred.
Table 3  Specialised instructions for dental care and maintenance of oral health for the ED patients are listed below

| I. For oral prophylaxis: | • Proper oral hygiene maintenance using soft bristled brush, nonabrasive fluoridated toothpaste and rinse for remineralisation; regular oral check-ups. 
• Patients must be made aware that the oral hygiene products they use are not acidulated. 
• Use of desensitizing toothpaste to decrease dentinal sensitivity. |
| II. For maintaining oral pH: | • Not to brush before an erosive challenge as dental pellicle can provides some protection against an erosive acidic challenge. 
• Not to brush teeth after vomiting; which may cause excessive enamel erosion. 
• Patients with purging type of behaviour should be advised to rinse their mouth with water or milk, antacid preparation, or to chew gum immediately after vomiting to decrease acidity in mouth. 
• Medication given for systemic conditions would not result in side effects of dry mouth or nausea, stoppage and substitution of offending medication 
• Neutral artificial saliva or sialagogues pastilles to be advised in case of dry mouth. |
| III. Modified diet patterns: | • Avoid acidic, citrus, carbonated or alcoholic drinks. 
• Wholesome nutritious diet along with healthy drinks such as milk to increase calcium uptake. 
• Referral to the dietician and regular intake of multivitamins and calcium supplements. |

over polyalkenoate (glass ionomer) restorative materials (Milosevic, 1999). Use of bonding porcelain and resin (dentine) bonded crowns (Milosevic, 1990; Milosevic and Jones, 1996), metal veneers (Darbar, 1994), metal onlays (Crawford and Aboush, 1993) and a combination of ‘double veneer’ (Bishop et al., 1996) is proven effective for satisfactory restoration of tooth destruction resulting from typical purging noticed in ED. Specialised instructions for dental care and maintenance of oral health for the ED patient are mentioned in Table 3.

Conclusion
Patients with oral problems as a corollary of ED may present first to the dentist. This presentation highlights the systemic and dental features of such disorders and suggests that the dentist cannot only assist in making the initial diagnosis but also provide timely intervention and referral for systemic and psychological treatment of the ED patient; along with providing dental care and support to the patient through treatment and recovery.

References
Biederman J, Ball SW, Monuteaux MC, Surman CB, Johnson JL, Zeilin S (2007). Are girls with ADHD at risk for eating disorders? Results from a controlled, five-
d nervosa: dental perspectives. Pract
Burkhart N, Roberts M, Alexander M, Dodds A
(2005). Communicating effectively with
patients suspected of having bulimia
nervosa. J Am Dent Assoc, 136(8): 1130-
1137.
bulimia nervosa in males. Am J
Psychiatry, 148(7): 831-843.
Committee on Sports Medicine Fitness (1996).
Promotion of health weight-control
practices in young athletes. Pediatrics,
97(5): 752-753.
Conrad R, Wegener I, Geiser F, Imbierowicz
nurture: calcification in the right thalamus
in a young man with anorexia nervosa and
Cortese S, Bernardina BD, Mouren MC (2007).
Attention-deficit/hyperactivity disorder
(ADHD) and binge eating. Nutr Rev, 65(9):
404-411.
Crawford PJ, Aboush YE (1993). The use of
adhesively retained gold onlays in the
management of dental erosion in a child: a
4-year case report. Br Dent J, 175(11-12):
414-416.
Darbar UR (1994). The treatment of palatal
erosive wear by using oxidized gold
veneers: a case report. Quintessence Int,
DeBate RD, Tedesco LA, Kerschbaum WE
(2005). Knowledge of oral and physical
manifestations of anorexia and bulimia
nervosa among dentists and dental
Fetissov SO, Harro J, Jaanisk M, Järv A,
Podar I, Allik J, Nilsson I, Sakhthivel P,
Autoantibodies against neuropeptides are
associated with psychological traits in
eating disorders. Proc Natl Acad Sci USA,
102(41): 14865-14870.
Foster DW (1998). Anorexia nervosa and
bulimia nervosa. In: Fauci AS, Braunwald
E, Isselbacher KJ, Wilson JD, Martin JB,
Kasper DL (eds.), Harrison’s Principles of
Internal Medicine, 14th edn. New York:
Frieling H, Römer KD, Scholz S, Mittelbach F,
Wilhelm J, De Zwaan M, Jacoby GE,
Kornhuber J, Hillemacher T, Bleich S
(2010). Epigenetic dysregulation of
dopaminergic genes in eating disorders.
Int J Eat Disord, 43(7): 577-583.
Frydrych AM, Davies GR, McDermott BM
(2005). Eating disorders and oral health: a
6-15.
Gabbay V, Asnis GM, Bello JA, Alonso CM,
Serras SJ, O'Dowd MA (2003). New onset
of body dysmorphic disorder following
frontotemporal lesion. Neurology, 61(1):
123-125.
Gendall KA, Kaye WH, Altemus M, McConaha
CW, La Via MC (1999). Leptin,
neuropeptide Y, and peptide YY in long-
term recovered eating disorder patients.
332-337.
of bulimia nervosa: Implications for the
health care team. Eur Eat Disord Rev, 3(2):
93-102.
Houy E, Debono B, Dechelotte P, Thibaut F
(2007). Anorexia nervosa associated with
frontal brain lesion. Int J Eat Disord,
40(8): 758-761.
Dental management of the chronic
618-621.
Klump KL, Kaye WH, Strober M (2001). The
evolving genetic foundations of eating
215-225.
Licinio J, Wong ML, Gold PW (1996). The
hypothalamic-pituitary-adrenal axis in
anorexia nervosa. Psychiatry Res, 62(1):
75-83.
Little JW (2002). Eating disorders: dental
Lo Russo L, Campisi G, Di Fede O, Di Liberto
manifestations of eating disorders: a
Lucas AR, Beard CM, O’Fallon WM, Kurland
of anorexia nervosa in Rochester, Minn.: a
population-based study. Am J Psychiatry,
148(7): 917-922.
Lucka I, Cebella A (2004). [Characteristics of
the forming personality in children
suffering from anorexia nervosa].
Milosevic A (1990). Use of porcelain veneers
to restore palatal tooth loss. Restorative


