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Review Article

Oral implications of eating disorders: a review

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Abstract Eating disorders (ED) are psychopathological conditions where patient demonstratesabnormal, 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 psycho-pathological 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 norepinepherine 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 amenorrhoea and a distorted body image. Other diagnostic features are self-induced weight loss achieved by food avoidance and vomiting or purging, amenorrhoea 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, leucopoenia, 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

Oral Tissue	Manifestation	Causes
Dentition	Enamel erosion, perimolysis (dental erosion on the palatal surfaces of teeth), sensitivity	Vomiting, salivary gland manifestations of ED affecting salivary flow rate, buffering capacity and pH of saliva resulting in erosion. Lemon pica.
	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 autonomic neuropathy.
	Hyposalivation, xerostomia, altered salivary flow rate, buffering capacity, pH and composition of saliva. Necrotising sialometaplasia	Side effects of drugs such as anti-depressants, vomiting, nutritional deficiency.
Alveolar bone	Osteopenia , osteoporosis	Nutritional deficiency, infection of dental or periodontal origin causing quicker alveolar bone loss.
Tongue	Glossodynia, taste impairment, dysgeusia, hyposgeusia, burning sensation	Trace metal deficiencies particularly zinc, somatoform disturbances and mucosal atrophy.

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

American Psychiatric Association (1995). *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Primary Care Version (DSM-IV-PC)*. Washington: American Psychiatric Association, pp. 119.

American Psychiatric Association (2000). Eating disorders. In: *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR).* Washington: American Psychiatric Association, pp. 583-597.

Aranha ACC, Eduardo CP, Cordás TA (2008). Eating disorders part I: Psychiatric diagnosis and dental implications. *J Contemp Dent Pract*, **9**(6): 73-81.

Biederman J, Ball SW, Monuteaux MC, Surman CB, Johnson JL , Zeitlin S (2007). Are girls with ADHD at risk for eating disorders? Results from a controlled, five-

year prospective study. *J Dev Behav Pediatr*, **28**(4): 302-307.

Bishop K, Bell M, Briggs P, Kelleher M (1996). Restoration of a worn dentition using a double-veneer technique. *Br Dent J*, **180**(1): 26-29.

Bouquot JE, Seime RJ (1997). Bulimia nervosa: dental perspectives. *Pract Periodontics Aesthet Dent*, **9**(6): 655-664.

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.

Carlat DJ, Camargo CA Jr (1991). Review of 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 K, Liedtke R (2008). Nature against nurture: calcification in the right thalamus in a young man with anorexia nervosa and obsessive-compulsive personality disorder. *CNS Spectr*, **13**(10): 906-910.

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*, **25**(3): 195-197.

DeBate RD, Tedesco LA, Kerschbaum WE (2005). Knowledge of oral and physical manifestations of anorexia and bulimia nervosa among dentists and dental hygienists. *J Dent Educ*, **69**(3): 346-354.

Fetissov SO, Harro J, Jaanisk M, Järv A, Podar I, Allik J, Nilsson I, Sakthivel P, Lefvert AK, Hökfelt T (2005). 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,* 14thedn. New York: McGraw-Hill, pp. 462-472.

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 review of the literature. *Aust Dent J*, **50**(1): 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. *Biol Psychiatry*, **46**(2): 292–299.

Glazer JL (2008). Eating disorders among male athletes. *Curr Sports Med Rep*, **7**(6): 332-337.

Harwood P, Newton T (1995). Dental aspects 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 right frontal brain lesion. *Int J Eat Disord*, **40**(8): 758-761.

Kleier DJ, Aragon SB, Averbach RE (1984). Dental management of the chronic vomiting patient. *J Am Dent Assoc*, **108**(4): 618-621.

Klump KL, Kaye WH, Strober M (2001). The evolving genetic foundations of eating disorders. *Psychiatr Clin North Am*, **24**(2): 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 implications. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*, **93**(2):138-143.

Lo Russo L, Campisi G, Di Fede O, Di Liberto C, Panzarella V, Lo Muzio L (2008). Oral manifestations of eating disorders: a critical review*. Oral Dis*, **14**(6): 479-484.

Lucas AR, Beard CM, O'Fallon WM, Kurland LT (1991). 50-year trends in the incidence 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]. *Psychiatr Pol*, **38**(6): 1011-1018.

Milosevic A (1990). Use of porcelain veneers to restore palatal tooth loss. *Restorative Dent,* **6**(3): 15-18.

Milosevic A, Jones C (1996). Use of resin bonded ceramic crowns in a bulimic patient with severe tooth erosion. *Quintessence Int,* **27**(2): 123-127.

Milosevic A (1999). Eating disorders and the dentist. *Br Dent J*, **186**(3):109-113.

Patrick, L (2002). Eating disorders: a review of the literature with emphasis on medical complications and clinical nutrition. *Altern Med Rev*, **7**(3): 184-202.

Schmidt U, Treasure J (1997). Eating disorders and the dental practitioner. *Eur J Prosthodont Restor Dent*, **5**(4): 161-167.

Sinno MH, Do Rego JC, Coëffier M, Bole-Feysot C, Ducrotté P, Gilbert D, Tron F, Costentin J, Hökfelt T, Déchelotte P, Fetissov SO (2009). Regulation of feeding and anxiety by alpha-MSH reactive autoantibodies. *Psycho-neuroendocrinology,* **34**(1): 140-149.

Studen-Pavlovich D, Elliott M (2001). Eating disorders in women's oral health. *Dent Clin North Am* 2001, **45**(3): 491-511.

Uher R, Treasure J (2005). Brain lesions and eating disorders. *J Neurol Neurosurg Psychiatry*, **76**(6): 852-857.

World Health Organisation (1992). *ICD-10: The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines.* Geneva: World Health Organisation, pp. 126-181.