

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

186,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Oral Implications of Eating Disorders

Aurea Lumbau and Giovanni Spano

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/66185>

Abstract

Eating disorders (EDs) are defined as persistent behavioural problems related to food and weight control, which significantly damage the physical and mental health with dramatic effects on the oral cavity. We briefly describe the effect on oral health and the principles of dental management.

Keywords: eating disorder, teeth, oral soft tissue, tooth erosion, mental disease

1. Introduction

Eating disorders (EDs) are classified as behavioural problems in food intake, and the main aim is body weight control. This condition is self-determined and not related to any known medical or psychiatric disease. All eating disorders bound to each other by several clinical features: the importance of body weight and body shape leading people affected to be obsessed by food checking and body checking. It is difficult to find out the true incidence of eating disorders due to the reluctance of the people affected to recognize it as a disease, thus avoiding consulting with a specialist, especially when early eating disorders are considered. There are numerous systemic manifestations caused by eating disorders; some are not pathognomonic, while others could lead the clinician to suspect the presence of a disease. Eating disorders are also characterized by intra-oral and extra-oral manifestations.

The most frequent intra-oral manifestations are dental erosion, trauma to the mucosa of the oral cavity and pharynx, dry mouth, a heightened risk of caries, periodontal problems, and lesions to the soft tissues secondary to the emesis, or effects induced indirectly by vomiting systemically [1].

2. Global view of the matter

Eating disorders (EDs) are psychiatric disorders that cause disability and chronicity. Few data are available to estimate their prevalence and correlates in the community. Current diagnostic classifications recognize main eating disorders, anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED) with symptomatic variants recorded as a global “not otherwise specified” class [2]. They have high prevalence in women [2, 3] and a comorbidity with other psychiatric disorders such as drug abuse, personality and mood disorders, anxiety. Systematic reviews [4] and recent studies [5] point out that women fall ill from AN and BN more than men in the ratio of 10:1. In addition, EDs are not only characterized by abnormal behaviours about food but also from deficits of emotional, cognitive and social factors that frequently lead to chronicity, relapse, reduced quality of life [6–9]. Prevalence of anorexia nervosa has been reported around 0.3% in American and European studies [10, 11]. It affects roughly 3,000,000 people in Italy and is the second-leading cause of death among female adolescents after road accidents [12]. The rate of diffusion of anorexia nervosa is keeping fairly constant; as opposed to Bulimia, that is continuously increasing [13–17]. The prevalence of bulimia nervosa in the young female population has a rate of 1%. Uncontrolled feeding also better known as binge eating disorder (BED) reaches about 1% in the general population, both male and female. As for the severity of eating disorders, according to a meta-analysis [18], anorexia nervosa was the mental disorder with the highest mortality rate; data from the National Centre for Epidemiology, Surveillance and Health Promotion, and several scientific studies [19] confirm that in the USA eating disorders still constitute the leading cause of death for mental illness. Yet, data are sparse, regarding binge disorder, with an estimated prevalence of around 3.3% among women and 0.8% among men in a large-scale population-based survey, conducted with telephone interviews in Austria [20, 21], and, respectively, 3.5 and 2.0% in the USA National Comorbidity Replication study (NCS-R) [2, 6, 7].

What are the factors that induce eating disorder?

Trouble about weight may be predictive: fear of gaining weight, dieting and negative body image. A high rate of eating disorders, mood disorders and certain anxiety disorders (panic disorder, generalized anxiety disorder and obsessive compulsive disorder) is found in family members of patients with EDs.

A high rate of families with substance abuse problems is present for BN. Studies also suggest a high rate of social phobia, generalized anxiety disorder, post-traumatic stress disorder [5]. A history of childhood obesity is a factor strongly associated with BN [22]. It is evident that youth depression and anxiety disorders play a significant role in influencing the onset or persistence of eating disorder symptoms; especially for depression [5]. Due to the complexity of these disorders, doctors recommend a multidimensional approach, which is the gold standard for the treatment of these patients. The best place for these treatments is the outpatient setting. Nevertheless, a number of patients (approximately 30%) do not respond to outpatient treatment, and needs more intensive care [23]. For this reason, a higher level of care is necessary.

All structures should be able to interact with each other in an organized service network of five levels: general practitioners and paediatricians, specialist outpatient services, hospital staff

or not, different types of day care services, therapeutic-rehabilitative residential, emergency [24]. Eating disorders have a high social impact but a low cost/benefit ratio.

The onset of eating disorders is often dated in adolescence or pre-adolescence. In the total population, prevalence and incidence of food disease are rare. But if we consider childhood, prevalence and incidence increase about 10 times, and in fact population aged between 12 and 16 years is considered 'at risk' [25]. So monitoring is necessary not only for treatment of overt diseases, but also for early diagnosis and prevention [24].

Eating disorders are also characterized by two complexity levels both mental and biological: we observe a frequent comorbidity with other psychiatric disorders such as anxiety, depression, obsessive-compulsive disorder, inevitable organic consequences (alterations of the cardiovascular system, bone, gastrointestinal, endocrine, gynaecological and neurological) determines from the beginning an integrated multi-professional approach. Finally, if the disease is an egosyntonic disorder (especially in case of AN), it is often difficult to start therapeutic alliance, forcing clinicians involved in care of a continuous redefinition and negotiation of therapeutic goals.

Evolution of the EDs is extremely variable

Normalization of weight, eating behaviour, absence of binge eating or compensatory behaviour is not sufficient to speak of healing. We must take into account other important aspects such as personality, family and extra-family relationships, emotional life, sexuality as well as the social and work integration [26]. The cure is possible in about 50% of cases. The other 50% have a difficult course with frequent relapses, chronic and a high risk of mortality [27–29]. Because of lack of awareness of the disease and a lack of motivation to treatment, only a small percentage of the people with anorexia and bulimia will appeal to deputies to the care services. This fact is due to a missed diagnosis general practitioners and paediatricians resulting in delayed treatment intervention and a worsening prognosis. The position of the Academy for Eating Disorders is that all eating disorders are mental illnesses and biologically determined and need the same level of treatment of other psychiatric disorders such as schizophrenia, bipolar disorder, major depression and obsessive-compulsive disorder. Despite this, eating disorders still receive little attention and insufficient treatment, compared to the high risk of complications and mortality [30].

2.1. Oral manifestation

Oral manifestations are mainly caused by nutritional deficiencies and consequent metabolic impairment, and lack of attention to personal oral hygiene and care.

Common intra-oral manifestations include erosion of dental tissues, trauma to the mucosa of the oral cavity and pharynx, dry mouth, high risk of caries, periodontal problems, and wound to the soft tissues caused to the direct actions of emesis, or indirectly because of effects induced systemically by vomiting. The extra-oral manifestations are mainly related to the pathological behavioural practice and include dysfunction and swelling of the parotid glands, TMJ disorders and cheilitis.

2.2. Dental erosion

Self-induced vomiting resulted in an increased frequency of erosion on the upper palatal, lower posterior buccal and occlusal surfaces. Some cases show erosions, both palatal and vestibular, and it is current opinion that these erosions are determined by two factors: intrinsic (gastric) acid results in palatally eroded sites and extrinsic (dietary) acids lead to labial erosion. This is known as perimolysis [31].

It may develop after frequent use of acidic sport drinks during physical activity, or from abnormal use of some caffeinated and/or carbonated drinks, to decrease the reflex hunger stimulus [32, 33] by increasing dilation of the stomach. Some patients will use vinegar and lemon juice (or slices of lemon) to eliminate/diminish the gustatory phase of the mechanism regulating hunger, which can also lead to erosion [34, 35]. Such erosion is characterized by a chemical rather than a bacterial dissolution and leads to uniform, polished and spoon-like surfaces, in contrast to abrasion, which is caused by mechanical wear [36]. Erosion can be accelerated by attrition [37, 38].

2.3. Dental caries

Whether the caries experience of eating disordered individuals is greater than that of the normal population remains unclear. Qualitative rather than quantitative differences, such as the cariogenicity of the oral flora, may occur and be dependent upon vomiting parameters. Sweetened drinks, candies or sugared chewing gum are frequently used by ED patients not only for energy, but also to reduce the sense of hunger; caries risk is high because of the frequency and the large quantity of eaten sugars, even if oral hygiene is generally acceptable in anorexic patients. Dental lesions and their complications may cause oral symptoms ranging from dental sensitivity to episodes of oral pain.

2.4. Saliva

The practice of self-induced vomiting, misuse of diuretics and laxatives may cause body dehydration with a negative impact on the salivary flow. Additionally, it may also influence salivary gland function. The saliva synthesis process (a dynamic process) also affects the blood and interstitial fluid, the metabolic state of the glands, electrolytes and protein synthesis that can be changed. Therefore, abnormalities such as alkalosis, acidosis and other general disturbances may affect the quantity and quality of the saliva. Salivary functions of buffering and lubrication are important in eating disorders. The salivary flow increases dramatically prior to vomiting because the medullary centre that controls vomiting is connected to salivary nuclei. Saliva concentration of bicarbonate and viscosity may change in patients with ED. Bicarbonate reduction and increased salivary viscosity act as a co-factor in dental erosion. The concentrations of other components like potassium, chloride, calcium, urea nitrogen and albumin can be normal. Episodic benign parotid enlargement has been described in bulimics by several workers. The swelling of the major salivary glands, parotids in particular, is a frequent manifestation of EDs and sometimes may be the presenting sign [39]. It is because of sialadenosis: a non-inflammatory enlargement of the salivary glands caused by a peripheral

autonomic neuropathy, which is responsible for disordered metabolism and secretion, resulting in acinar enlargement and functional impairment. Salivary gland inflammation can also produce variable size of calculi, which can occlude the salivary ducts and cause intense pain. The parotid gland tends to change the physiognomy of the face, giving a puffy look; this phenomenon is usually reversible in a few weeks with the abstention from compensatory practices (especially vomiting). Sialadenosis may also involve minor intraoral salivary glands [40].

2.5. Periodontal disease

The evidence on periodontal status is conflicting.

Patients affected by ED may have poor oral hygiene, which may lead to gingival inflammation and potentially predisposition to periodontitis. Adequate oral care habits are impeded by binge eating and purging episodes. Nutritional deficiencies [41], especially in vitamin C [42], may also predispose one to gingivitis.

The deficiency of vitamin C causes defective collagen synthesis, which can be associated with generalized swollen gingiva, spontaneous bleeding, ulcerations, tooth mobility and increased periodontitis [43].

2.6. Soft tissue lesions

Reduction in intake of vitamins and other nutrients, induce general metabolic alterations, iron deficiency anaemia, causing generalized mucosal atrophy. In particular, a deficiency of vitamins in the B group, especially B1, B6 and B12, has been classically associated with a decrease in epithelial cell turnover.

Angular cheilitis, candidosis, glossitis and oral mucosal ulceration are effects of nutritional deficiencies. Generally, this is particularly evident on the tongue; here, in association with erythema, it produces the clinical picture of atrophic glossitis. Mucosal atrophy may also cause diffuse oral burning sensation, which can be more intense on the tongue (glossodynia). Dysgeusia (altered taste sensation) and xerostomia (dry mouth) can be independent and disconnected from oral signs being of psychogenic origin and expressing somatization of underlying disorders. Erythematous mucosal lesions, especially on the soft palate in purging-type behaviour, may be related to the direct offending action of acid during vomiting (epithelial erosion), and sometimes to repetitive frictional trauma (palatal haematoma) caused by the object used to induce vomiting. Multiple nutritional deficiencies may also constitute a predisposing factor for oral opportunistic infections both directly [44] and through impairment of immunologic system function [45].

3. Management

There are numerous systemic manifestations caused by eating disorders. Several of these induce the physician to suspect a disease like the 'sign of Russell' (lesions of the dorsum of the

hands, arising from their repeated use to induce vomiting). As mentioned earlier, eating disorders are also characterized by intra-oral and extra-oral manifestations. The dentist may be the first person to become aware of the eating disorder by finding a pattern of teeth erosion due to the effects of chronic vomiting [46, 47].

On the basis of the pattern of dental erosion, dentist should discuss his findings with the patient's parents and arrange for referral to a physician for evaluation and management of a possible eating disorder. In these cases, the dentist's goal is to improve the patient's oral hygiene.

It is the duty of the dentist to teach oral hygiene concepts such as brushing technique, flossing and using fluoride toothpaste or mouthwash. The patient is instructed to use a baking soda mouth rinse after vomiting. For dentinal sensitivity is indicated a desensitizing toothpastes and fluoride varnish. Anorexia nervosa may be more difficult to find out for dentist. Patients with anorexia nervosa who also are bulimic may be identified by dental signs of bulimia.

Timing of intervention for eroded teeth is controversial. Caries and periodontal disease should be managed as usual? Dentists should wait before restoring worn surfaces?

There is no definite contraindication to restoring eroded surfaces other than the continued acid dissolution of tooth substance from around the restoration if vomit continues. Pain relief, reduced dentine sensitivity and improved aesthetic will motivate the patient.

During therapy, tooth wear should be monitored with study casts and photographs. On the other hand, dentist and patient need to be aware that frequently complex restorations may fail due to illness. In the worse cases, full coverage may be required in an attempt to save teeth with conservative tooth preparation and ceramic crowns. Patients with bulimia should not have treatment planned for permanent restorative procedures until the illness is during the active phase. Once the patient is stable and wants to have the teeth with severe erosion restored, this can be done. Endodontic and prosthetic treatment must be used for teeth with little clinical crown remaining. Patients with anorexia nervosa should regain their lost weight and be stabilized before starting complex restorative or prosthodontic treatment. The dentist can make several recommendations to the anorexic and bulimic. Tooth brushing after vomiting is generally regarded as inadvisable because the softened, demineralized surface is more susceptible to toothbrush abrasion. The treatment plan will be dependent on several parameters and it would be a discretionary decision of the dentist.

Dental restoration must be done with materials that can resist to acid attacks allowing preservation of occlusal plane. Glass ionomer cements are not indicated. Even a space of less than 1 mm is amenable to restoration with composite bonded to dentine. Any eroded and sclerosed dentine should be over etched (20–30 s) in order to enhance opening of dentinal tubules and intra-tubular formation of resin tags. Adhesive dentistry has advantages over conventional forms of treatment in that it is reversible and the latest generation of dentine bonding agents are durable. Space requirements are still minimal when using these adhesive techniques.

4. Conclusions

Patients whose teeth have been damaged as a consequence of an eating disorder are most likely to present first to a dentist who can assist in making the initial diagnosis and efficiently influence the progress of the medical and psychological management by providing support and dental care.

Oral manifestations of ED may appear in different stages along the disease progression. In fact, some oral manifestations may occur very early during disease onset (e.g. sialadenosis, palatal erythema, clinical unexplained oral symptoms); therefore, it can be very useful for an early diagnosis and prompt recovery. The dentist may be one of the few and the first clinicians contacted by the patient for incipient oral discomfort (i.e. dental sensitivity, oral pain and unusual oral sensations) [48].

In order to strengthen communication, the visit should be conducted confidentially using appropriate language and terminology: dentists should avoid judgement and pressure, observe the patients' body language and remain calm, reassuring and supportive.

It is well known that patients with ED have low self-esteem and dislike of their body.

Mouth is one of the main factors in the relationships, teeth and eyes are one of the most attractive parts in a face to the viewer. Being less attractive leads to a decrease in self-esteem, triggering a vicious circle in those who already have problems with the body. Therefore, the oral cavity should be further considered in both diagnosis and follow-up.

'Furthermore, the treatment of these manifestations may be important to the overall prognosis by preventing or reducing damages, which can modify eating habits and function, patient's self-image [49] and consequently self-esteem'.

4.1. How to face the problem

These pathologies involve different areas and produce malnutrition acting on the central nervous system (CNS) and the various organic districts; disorders of the body image; cognitive style; emotional style; family and interpersonal dynamics. Lines of action will move in parallel with each other and will be oriented towards the recovery of the physiological body weight, through learning behaviours of functional food and a diet adequate to the different social situations; definition of a therapeutic plan, with a combination of psychological, medical and pharmacological therapies; increased awareness of the patient's family and relapse prevention through continuous treatment.

'The integrated multi-professional approach to the EDs requires a transformation of "knowledge" and "know how" in a "know-how together". For this reason, the integrated multi-professional group is made up of different specialists working from the start in a shared project with common objectives and strategies tested by weekly team meetings and, when possible, by daily briefings. Intervention protocols should be subjected to continuous verification and monitoring, in liaison with similar experiences already. The staff also will have to be placed in a permanent training process, characterized by internal teams and external supervision. In

particular, within the team is of particular importance is the division of roles and responsibilities, so as to make the process of collaboration' [23].

In particular:

1. Task of the psychiatrist will be: diagnosis of any psychiatric comorbidity, psychopharmacological treatment, individual psychotherapeutic treatment or panel and the study of the dynamics of the multidisciplinary team;
2. The nutritionist/internist will be in charge of the diagnosis and treatment of medical complications, as well as the planning of medical care of nutrition;
3. The psychologist will cure the psychopathological assessment, research (evaluation of outcome and effectiveness of the treatment), individual treatment and family treatment;
4. The dietitian will be tasked to determine, plan and control meals, follow the food psychoeducation (individual and/or group), establish weighting targets and follow the patient assisted in the meals
5. Other professions where problems occur in other districts [23].

Author details

Aurea Lumbau* and Giovanni Spano

*Address all correspondence to: alumbau@uniss.it

Surgical Microsurgical and Medical Science Department, School of Dentistry, University of Sassari, Sassari, Italy

References

- [1] Lumbau A., Spano G., Meloni SM, Sanna F. Lugliè PF. Eating disorders: assessment of knowledge on a dentist's sample. *Int J. Child Health Nut.* 2015;4:50–3.
- [2] Preti A, De Girolamo G, Vilagut G, Alonso J, De Graaf R, Bruffaerts R, Demyttenaere K, Pinto-Meza A, Haro JM, Morosini P, ESEMeD-WMH investigators. The epidemiology of eating disorders in six European countries: results of the ESEMeD-WMH project. *J Psych Res.* 2009;43(14):1125–32. .
- [3] Swanson SA, Crow SJ, Le Grange D, Swendsen J, Merikangas KR. Prevalence and correlates of eating disorders in adolescents. *Arch Gen Psych.* 2011;68(7):714–23.

- [4] Stice E, Marti CN, Durant S. Risk factors for onset of eating disorders: evidence of multiple risk pathways from an 8-year prospective study. *Behav Res Ther.* 2011;49(10): 622–7.
- [5] Hautala L, Helenius H, Karukivi M, Maunula AM, Nieminen J, Aromaa M. The role of gender, affectivity and parenting in the course of disorder eating: a 4-year prospective case-control study among adolescents. *Int J Nurs Stud.* 2011;48(8):959–72.
- [6] Hudson JL, Pope Jr HG, Yurgelun-Todd D, Jonas JM, Frankenburg FR. A controlled study of lifetime prevalence of affective and other psychiatric disorders in bulimic outpatients. *American Journal of Psychiatry* 1987;144:1283–7.
- [7] Hudson JL, Hiripi E, Pope Jr HG, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psych.* 2007;61:348–58.
- [8] Jordan J, Joyce PR, Carter FA, Horn J, McIntosh VVW, Luty SE, McKenzie JM, Frampton CM, Mulder RT, Bulik CM. Specific and nonspecific comorbidity in anorexia nervosa. *Int J Eat Disord.* 2008;41(1):47–56.
- [9] Swinbourne JM, Touyz SW. The comorbidity of eating disorders and anxiety disorders: a review. *Eur Eat Disord Rev.* 2007;15(4):253–74.
- [10] Hoek HW, van Hoeken D. Review of the prevalence and incidence of eating disorders. *Int J Eat Disord.* 2003;34(4):383–96.
- [11] Favaro A, Ferrara S, Santonastaso P. The spectrum of eating disorders in young women: a prevalence study in a general population sample. *Psychosom Med.* 2004;65:701–8.
- [12] Renna C, La prevenzione dei DCA. Ministero della Salute, Dipartimento della Gioventù, Istituto superiore di Sanità; 2012. pp. 251–63. Roma.
- [13] Willi J, Giacometti G, Limacher B. Update on the epidemiology of anorexia nervosa in a defined region of Switzerland. *Am J Psychiatry.* 1990;147:1514–17.
- [14] Hall A, Hay P. Eating disorder patient referrals from a population region 1977–1986. *Psychol Med.* 1991;21:697–701.
- [15] Jorgensen J. The epidemiology of eating disorders in Fyn County Denmark, 1977–1986. *Acta Psych Scand.* 1992;85:30–4.
- [16] Hoek H, Bartelds A, Bosveld J, et al. Impact of urbanization on detection rates of eating disorders. *Am J Psychiatry.* 1995; 152:1272–8.
- [17] Turbull S, Ward A, et al. The demand for eating disorder care: an epidemiological study using the general practice research database. *British J. Psych.* 1996;169:705–12.
- [18] Harris EC, Barraclough B. Excess mortality of mental disorder. *Br J Psychiatry.* 1998;173:11–53. Review.

- [19] Birmingham CL, Su J, Hlynsky JA, Goldner EM, Gao M. The mortality rate from anorexia nervosa. *Int J Eat Disord.* 2005;38(2):143–6.
- [20] Kinzl JF, Traweger C, Trefalt E, Mangweth B, Biebl W. Binge eating disorder in males: a population-based investigation. *Eating and Weight Disorders* 1999;4:169–74
- [21] LaVange L, Stearns S, Lafata J, Koch G. Innovative strategies using SUDAAN or analysis of health surveys with complex samples. *Stat Meth Med Res.* 1996;5:311–29.
- [22] Pratt CA, Stevens J, Daniels S. Childhood obesity prevention and treatment: recommendations for future research. *Am J Prev Med.* 2008;35(3):249–52. doi: 10.1016/j.amepre.2008.05.025. Epub 2008 Jul 10.
- [23] Rapporti Istisan 13/49. 30 Ottobre 2013. ISSN 1123-3117. Roma.
- [24] De Virgilio G, Coclite D, Napoletano A, Barbina D, Dalla Ragione L, Spera G, Di Fiandra T. Consensus Conference on Eating Disorders (ED) in adolescents and young adults. Istituto Superiore di Sanità; 2013 (Rapporti ISTISAN 13/6). 26 Rapporti ISTISAN 13/49 27. Roma.
- [25] Jaffa T, Mcdermott B, Mansi GL Eating disorders in children and adolescents. Cortina editor. 2009. Milano.
- [26] De Giacomo P, Renna C, Santoni Rugiu A. Guide on eating disorders. Franco Angeli editor. 2005. Milano.
- [27] Sullivan PF. Course and outcome of anorexia and bulimia nervosa. In: Fairburn CG, Brownell KD (Ed.) 2nd ed., *Eating Disorders and Obesity*. New York: The Guilford Press; 2002. pp. 226–30.
- [28] Steinhausen HC. The outcome of anorexia nervosa in the 20th century. *Am J Psych.* 2002;159(8):1284–93.
- [29] Steinhausen HC, Weber S, The outcome of bulimia nervosa: finding from one quarter century of research. *Am J Psych.* 2009;166(12):1331–41.
- [30] Klump KL, Bulik CM, Kaye WH, Treasure J, Tyson E. Academy for eating disorders position paper: eating disorders are serious mental illnesses. *Int J Eat Disord.* 2009;42(2): 97–103.
- [31] Chadwick RG, Mitchell HL Conduct of an algorithm in quantifying simulated palatal surface tooth erosion. *J Oral Rehabil.* 2001;28(5):450–6.
- [32] Moazzez R, Smith BG, Bartlett DW. Oral pH and drinking habit during ingestion of a carbonated drink in a group of adolescents with dental erosion. *J Dent.* 2000;28(6):395–7.
- [33] Al-Dlaigan YH, Shaw L, Smith A. Dental erosion in a group of British 14-year-old, school children. Part I: prevalence and influence of differing socioeconomic backgrounds. *Br Dent J.* 2001;190(3):145–9.

- [34] Järvinen V, Rytömaa I, Meurman JH. Location of dental erosion in a referred population. *Caries Res.* 1992;26(5):391–6.
- [35] Del Signore A, Campisi B, Di Giacomo F. Characterization of balsamic vinegar by multivariate statistical analysis of trace element content. *J AOAC Int.* 1998;81(5):1087–95.
- [36] Ibarra G, Senna G, Cobb D, Denehy G. Restoration of enamel and dentin erosion due to gastroesophageal reflux disease: a case report. *Pract Proced Aesthet Dent.* 2001;13(4):297–304; quiz 306.
- [37] Bartlett DW, Evans DF, Smith BG. The relationship between gastro-oesophageal reflux disease and dental erosion. *J Oral Rehabil.* 1996;23:289–97.
- [38] Scheutzel P. Etiology of dental erosion: intrinsic factors. *Eur J Oral Sci.* 1996;104(pt 2):178–90.
- [39] Coleman H, Altini M, Nayler S, Richards A. Sialadenosis: a presenting sign in bulimia. *Head Neck.* 1998;20(8):758–62.
- [40] Aframian DJ. Anorexia/bulimia-related sialadenosis of palatal minor salivary glands. *J Oral Pathol Med.* 2005;34(6):383.
- [41] Prousky JE. Pellagra may be a rare secondary complication of anorexia nervosa: a systematic review of the literature. *Altern Med Rev.* 2003;8(2):180–5. Review.
- [42] Christopher K, Tammaro D, Wing EJ. Early scurvy complicating anorexia nervosa. *South Med J.* 2002;95(9):1065–6.
- [43] Touyz LZ. Oral scurvy and periodontal disease. *J Can Dent Assoc.* 1997;63(11):837–45. Review.
- [44] Paillaud E, Merlier I, Dupeyron C, Scherman E, Poupon J, Bories PN. Oral candidiasis and nutritional deficiencies in elderly hospitalised patients. *Br J Nutr.* 2004;92(5):861–7.
- [45] Chandra RK. Nutrition and the immune system from birth to old age. *Eur J Clin Nutr.* 2002;56(Suppl 3):S73–6. Review.
- [46] Milosevic A, Brodie DA, Slade PD. Dental erosion, oral hygiene, and nutrition in eating disorders. *Int J Eat Disord.* 1997;21:195–9;
- [47] Milosevic A. Eating disorders and the dentist. *Br Dent J.* 1999;186:109–13.
- [48] Burkhart N, Roberts M, Alexander M, Dodds A. Communicating effectively with patients suspected of having bulimia nervosa. *J Am Dent Assoc.* 2005;136(8):1130–7. Erratum in: *J Am Dent Assoc.* 2005 Nov;136(11):1517.
- [49] Bonilla ED, Luna O. Oral rehabilitation of a bulimic patient: a case report. *Quintessence Int.* 2001;32(6):469–75.

