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Eating disorders through the periodontal lens

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1 | INTRODUCTION

Eating disorders are mental disorders defined as persistent disturbances of eating or eating-related behaviors that result in the altered consumption or absorption of food that significantly impairs physical health or psychosocial functioning.[1] Eating disorders are classified into subthreshold disorders (other specified or nonspecified eating disorder) and full-threshold disorders, such as pica, rumination disorder, avoidant/restrictive food intake disorder, anorexia nervosa, bulimia nervosa, and binge-eating disorder. The last three are the most frequent ones among adolescents and adults. These disorders have serious psychiatric

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CONFLIC T OF INTEREST

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and somatic comorbidities. Depressive anxiety (including obsessive-compulsive disorder) or addictive disorders are frequently associated with eating disorders. Eating disorders also have severe consequences on somatic health, especially cardiovascular, gastrointestinal, metabolic, bone, and reproductive health.[2–5]

By the 1980s, case reports were published highlighting the oral comorbidities of eating disorders.[6,7] The first clinical study on the oral complications of anorexia nervosa was published more than 40 years ago, and it reported a high prevalence of dental erosion, caries, and saliva abnormalities in a cohort of 39 women between 14 and 42 years of age.[8] Although most of the studies have investigated the impact of eating disorders on the dentition, they have neglected the effects on the periodontal tissues. To the best of our knowledge, there are no reviews focused on the periodontal conditions in individuals with eating disorders. Previous systematic reviews have only dealt with mental health globally or dental caries and erosion in individuals with eating disorders. Therefore, the purpose of this narrative review was to summarize the existing evidence on eating disorders and dental manifestations, emerging evidence on eating disorders and periodontal manifestations, and findings directly identified by the authors who have a specialized practice in eating disorders in a hospital setting.

2 | EATING DISORDERS: DIAGNOSIS, EPIDEMIOLOGY, PATHOGENESIS, AND TREATMENTS

We will focus here on anorexia nervosa, bulimia nervosa, and binge-eating disorder. Self-evaluation is unduly influenced by body shape and weight in both anorexia nervosa and bulimia nervosa. Anorexia nervosa is characterized by a low weight and/or a significant weight loss. It can be subdivided into a restricting type (in which the weight loss is accomplished by reduction of food intake, excessive physical exercise, or prolonged fasting) and a binge-eating/purging type (in which patients use self-induced vomiting or laxatives, diuretics, appetite suppressants, or enemas). Bulimia nervosa is characterized by recurrent episodes of binge eating with purgative episodes to control weight gain. Weight is generally normal. Binge-eating disorder is characterized by recurrent episodes of binge eating, but without the inappropriate compensatory behaviors characteristic of bulimia nervosa. It is for this reason that binge-eating disorder sufferers are generally overweight or obese.[9] However, obesity, defined by the World Health Organization as abnormal or excessive fat accumulation that presents a risk to health with a body mass index over 30 kg/m^2, is not an eating disorder, and thus beyond the scope of this review. It is important to understand that the course of the eating disorders often involves switching from one form of eating disorder to another.[10]

Classical estimates of the prevalence of eating disorders according to *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition, criteria vary in western countries, usually from 0.8% for anorexia nervosa to 4% for binge-eating disorder in women, with a lower prevalence in men.[11,12] These prevalences seem to vary in different parts of the world.[13] The initial onset of eating disorders usually occurs during adolescence and young adulthood.[14] The highest risk groups for eating disorders are young girls, individuals with

a past history of eating disorders, individuals with a family history of eating disorders or obesity, and adolescents with concerns about their weight due to gastrointestinal disorders or psychologic problems. Professionals at risk for anorexia nervosa or bulimia nervosa but not binge-eating disorder are models, athletes in sports disciplines with weight categories or requiring weight control (particularly at a competitive level), and subjects suffering from pathologies involving diets (such as individuals with type 1 diabetes, familial hypercholesterolemia, or inflammatory bowel disease) (Appendix 1).

Certain individuals, because of predisposing factors (genetic, environmental), could develop different levels of vulnerability for eating disorders.[15] This vulnerability could surface due to precipitating elements that lead to the loss of “well-being” or “eudemonia” as a result of individual factors (mental or somatic) or interpersonal factors. The unsettled homeostasis is replaced by an eating disorder, a process that could contribute to prolonging the mental disorder. Vulnerability levels and precipitating factors vary in nature and intensity from one subject to another, thus explaining the variable clinical expression of the condition in terms of age at onset, clinical symptoms, intensity of the disorder, duration of evolution, therapeutic response, or evolution to chronic illness or other psychiatric disorders.

In order to treat the overall symptoms of the eating disorder, the therapeutic strategy has to be multidisciplinary and include psychologic, nutritional (treating individuals with malnutrition or those that are overweight), somatic, social, and familial-related aspects. The treatment plan is based on coordinating the various parties involved as part of a long-term program for the duration of the disorder. Guidelines for the treatment of anorexia nervosa, bulimia nervosa, and binge-eating disorder have been published by several national scientific organizations (Appendix 1). Treatment is initially organized, if possible, on an outpatient basis and adapted to the patient’s needs, comorbidities, and complications. Psychotherapy (individual or familial) and nutritional approaches are an important component of the treatment. Medications, including psychotropic (anxiolytic, neuroleptic, and antidepressants drugs) and various supplements can be prescribed as a complement to the psychotherapeutic treatment and nutritional approaches.[16] Treatment programs are more efficient if they are initiated soon after the onset of eating disorders. The recovery from eating disorder can vary from several months to years (around two in three after 5 years for anorexia nervosa and 12 years for bulimia nervosa), or it can be a long-term and chronic process (for one in five of eating disorders).[17–20] Eating disorders can have severe sequelae with high mortality rates (two to five times higher than the general population), especially for anorexia nervosa, which has the highest mortality rates of all psychiatric disorders.[11,21] Consequently, early detection of the onset of eating disorders is of utmost importance. Dental health professionals are frequently the first professionals who may suspect an eating disorder because of the unique clinical oral features.

3 | CLINICAL IMPACT OF EATING DISORDERS ON ORAL HEALTH CONDITIONS

In studies, where the diagnosis of eating disorder is not explicit—that is to say, anorexia nervosa, bulimia nervosa, or binge-eating disorder is not diagnosed—then it is designated as

individuals “with eating disorders.” This general term will be used for the remainder of this discussion.

3.1 | Teeth and temporomandibular joints

Erosive tooth wear is the most obvious oral manifestation of the purgative types of eating disorders.[22–26] Erosive tooth wear is a chemical-mechanical process resulting in a cumulative loss of hard dental tissue without bacterial involvement, and it is characterized by loss of the natural surface morphology and contour of the teeth.[27] Vomiting-associated eating disorders are one of the main causes of intrinsic erosion, along with gastroesophageal reflux disease, alcohol abuse, and pregnancy.[28] In people with eating disorders, the erosive lesions begin from the cingulum and expand to the entire lingual surface of the anterior teeth (Figures 1 and 2).[29] The erosive lesions become more severe and affect the dentin and palatal surfaces and beyond in parallel with the duration of the self-induced vomiting.[30] In addition, abrasion (ie, friction from toothbrushing and or the dentifrice) is usually observed concomitantly when traumatic toothbrushing follows the vomiting episodes.[31] A lateral sleeping position often leads to an asymmetric localization of the erosive tooth wear in people with eating disorders who suffer from gastrointestinal reflux disease (Figure 3). Acidic reflux has been causally associated with sleep bruxism in patients.[32] Bruxism-induced attrition of occlusal surfaces may contribute to the vicious circle of tooth wear in people with eating disorders.[31]

Several clinical indices have been developed to diagnose erosive tooth wear, including the basic erosive wear examination score, which is simple and reliable to use in daily practice and for research purposes (Appendix 2).[33] Thus, it has been reported that people with bulimia nervosa have a higher prevalence of a cumulative basic erosive wear examination score of 3 or more and a higher frequency of erosion than anorexia nervosa, caused most likely by the higher frequency of vomiting episodes in bulimia nervosa.[34] Two systemic reviews with meta-analyses have reported a significant higher risk of erosive tooth wear in people with eating disorders, with odds ratios of 12.4 (95% confidence interval 4.1–37.5) and 5.0 (95% confidence interval 3.31–7.58).[35,36] When vomiting was associated with eating disorders, the estimated risk of erosive tooth wear was even higher than in people with nonvomiting eating disorders. Regarding dental caries in people with eating disorder, the meta-analysis by Kisely et al found significantly higher scores for decayed missing filled surfaces, with a mean difference of 3.07 (95% confidence interval 0.66–5.48).[36] Nocive dietary habits, mainly a high consumption of carbohydrates and multiple episodes of food intake, are plausible etiologies.[37,38]

When the chewing surfaces of the teeth have been worn, a loss of stability of the interarch contacts and a loss of vertical dimension ensue, which may lead to or contribute to temporomandibular disorders.[39] Case-control studies have shown that people with eating disorders, especially those who report vomiting and/or binge-eating behaviors, present with a higher sensitivity to muscle palpation, prevalence of intensive gum chewing, and higher levels of cranio-facial pain than healthy people do.[40,41] Nearly 60% of people with eating disorders reported some form of facial pain currently or in the recent past.[42] Interestingly, an association has been reported between gastrointestinal reflux disease and chronic, painful

temporomandibular disorders mediated by anxiety and somatization.[43] The presence of chronic facial pain may interfere with the overall treatment of individuals with eating disorders and should be systematically measured and managed.

3.2 | Oral mucosa and periodontium

An unbalanced diet (vitamin deficiencies, high carbohydrate consumption), traumatic habits, hyposalivation, and poor oral hygiene may negatively affect the health of the oral mucosa and periodontal tissues. People with eating disorders exhibit a higher prevalence of dry lips, labial erythema, exfoliative cheilitis, palatal tissue discoloration (orange-yellow palate), hemorrhagic lesions, lip-cheek biting, burning tongue, and periodontal diseases than healthy patients do.[44,45] Oral mucosal ulcerative lesions are usually related to self-induced vomiting habits.[46] Thus, during the clinical examination of a person at risk for eating disorders, dentists must systematically check for palatal ulceration induced by the finger or toothbrush that provokes the vomiting (Figure 4).[47]

Several deficiencies are common in restrictive types of eating disorders, such as in calcium, potassium, and vitamins. Very low blood levels of vitamin C explain the occurrence of gingival bleeding that is characteristic of scurvy, which has been reported in people with anorexia nervosa.[48] It is noteworthy that carbohydrates may form a higher proportion of total energy intake in people with eating disorders than in the general population.[49,50] A high consumption of sugar has been associated with gingival inflammation in adolescents and young adults.[51–54] In people with eating disorders, there are still conflicting results in the literature regarding plaque control and gingival inflammation (Table 1). Studies have reported lower[55] as well as higher[34,49,50,56] plaque indices and gingival bleeding in people with eating disorders than in controls. However, a high frequency of toothbrushing (ie, three times or more per day) has been repeatedly reported in people with eating disorders.[34,37,57] Findings regarding the occurrence of periodontal pockets of more than 3 mm reveal no difference between the anorexia nervosa/bulimia nervosa population and the general population.[58] However, a higher percentage of sites with a clinical attachment level of 3 mm or more have been repeatedly shown in people with eating disorders, particularly in those suffering from anorexia nervosa. Finally, it can be assumed that people with eating disorders are at higher risk than the general population for gingival recession, but not for periodontitis.[34,49,50,56] In a cohort study (n = 23, age range 14.4–27.2 years), 43% of patients with eating disorders presented with at least one site of gingival recession.[59] One recent cohort study found more periodontitis in a group of 33 outpatients with anorexia nervosa or bulimia nervosa than in sex and age-matched controls; however, the definition of periodontitis cases included the presence of recession or a probing depth greater than 3 mm and was based on partial mouth recording (six teeth).[56]

Based on our personal experience, we find that the periodontal impact of eating disorders is primarily triggered by abrasion of the gingiva related to compulsive toothbrushing (ie, extreme and frequent brushing), resulting in multiple areas of gingival recession (Figure 5). An unbalanced diet and thin periodontal phenotypes are important predisposing factors. We also realize that the gingival inflammation in people with eating disorders is aggravated by the negative morphology of the cervical region where erosive tooth wear is present.

The compromised cervical anatomy suppressing the deflection of food, leads to food, dental plaque, and, ultimately, calculus accumulation (Figure 6). After restorative treatment, we would observe the resolution of gingival inflammation and recession. In addition, the repeated vomiting episodes promote gastrointestinal reflux, which aggravates dental erosion and initiates gingival recession on the palatal surfaces of the maxillary teeth (Figure 7).[58,60]

It should be highlighted that in people with eating disorders the cervical erosive tooth wear often prevents use of the cemen-to-enamel junction for measuring the height of the recession and for calculating the clinical attachment level. Any other reference point used should be clearly identified in the periodontal chart.

4 | BIOLOGICAL IMPACT OF THE EATING DISORDERS ON ORAL HEALTH CONDITIONS

4.1 | Oro-digestive microbiota

Few studies have examined oral microbiological data in subjects with eating disorders to determine whether their unbalanced diet and acidic oral environment change the levels of cariogenic and periodontal pathogenic bacteria. Two studies by the same group observed no difference in the levels of salivary counts of *Streptococcus mutans* and *Lactobacillus* species in anorexia nervosa or bulimia nervosa patients compared with controls.[49,50] However, higher levels of salivary *Streptococcus sobrinus*, *S. mutans*, and *Lactobacillus* species have been reported in people with eating disorders.[37,55,61] Only a few studies have investigated the oral mycobiota of patients with eating disorders, and those studies found a high prevalence of fungi (based on microbial culture) and a significant fungal diversity (based on culture-independent molecular techniques).[61–63] To date, there are no oral microbiome data based on next-generation sequencing in individuals with eating disorders. Nevertheless, emerging evidence from studies on the gut microbiome suggests that people with anorexia nervosa have a decreased intra-individual bacterial richness, an increased Bacteroidetes-to-Firmicutes abundance ratio and significant changes in the relative abundances of several digestive bacteria, compared with healthy women.[64,65]

4.2 | Salivary flow rate and composition

Swelling of the parotid salivary glands is a clinical sign of vomiting-associated eating disorders.[57] The salivary glands remain functional in most of the cases. The involvement of the submandibular glands is rare. These signs can remain present for months after recovery from the eating disorder. A patient with this type of clinical sign may be suffering from a current or past eating disorder. Therefore, it is important to consider this presentation when conducting the interview with these patients. Hyposalivation, measured by an unstimulated salivary flow rate, was repeatedly noticed in individuals with eating disorders across comparative studies.[37,55,56] The volume of unstimulated whole saliva was reduced in patients with eating disorders compared with healthy individuals, although it was primarily due to medications.[60,66,67] However, no differences in the stimulated salivary flow rate between people with and without eating disorder have also been reported.[37,49,50,55] Hyposalivation could be involved in progressive halitosis, plaque accumulation, gingival inflammation, dental caries, and erosion.[68] In addition, significant

increases in the concentration of several salivary proteins, enzymes, and minerals has been observed in people with eating disorders: total proteins,[58] albumin, aspartate and alanine aminotransferases, collagenase, inorganic phosphate, chloride, and magnesium.[67,69] Despite starvation and the development of anorexia nervosa, key salivary enzymes show physiologic activity. This indicates a partial adaptation by the body to severe conditions during malnutrition.

4.3 | Systemic biomarkers of periodontal breakdown

A study evaluated the relationship between routine blood parameters and oral health outcomes (dental erosion, reduced periodontium) in women with anorexia nervosa or bulimia nervosa. After adjustments for age and duration of the disease, high serum ferritin levels were associated with a generalized reduced periodontium.[70] The main role of ferritin is iron storage, and it can act as a positive acute-phase reactant. Ferritin is considered a marker of starvation. Given this positive outcome, additional studies are warranted to determine the potential use of serum markers as indicators of periodontal breakdown in individuals with eating disorders.

5 | NEED FOR IMPROVED DENTAL SCREENING AND DENTAL CARE IN PEOPLE WITH EATING DISORDERS

Dentists and dental hygienists are in a good position to recognize the aforementioned oral symptoms of eating disorders.[71–74] They can play a major role in the early diagnosis and treatment of patients with eating disorders.[75–77] However, limitations in the dental curriculum on this topic have been identified.[78,79] A study investigating the knowledge, clinical experience, and professional attitude of dentists regarding people with eating disorders found that the main source of knowledge and information on this topic was obtained from common media (television, web). The majority of dentists (76%) reported a need for more undergraduate education and continuing education related to the management of eating disorders.[80] In addition, gender and gender-related health beliefs have been identified as important factors in a dentist's capacity to assist in the prevention of eating disorders. Female dentists seemed to have greater knowledge of the oral and physical manifestations of eating disorders, which explained their superior ability to assess the oral cues associated with these diseases and their superior ability to provide oral care advice and referral compared with male dentists.[80–82] In the same vein, a survey found that the more family members in the health professions that dental students had the better they felt able to communicate with patients and providers from other disciplines about eating disorders.[83] A cross-sectional study, aiming to determine the knowledge among dentists and dental hygienists concerning the oral and physical manifestations of eating disorders, found that more dental hygienists than dentists correctly identified the oral manifestations and physical cues of eating disorders.[84] However, even among dental hygienists, several barriers were identified, such as their perceived ability, individual knowledge of oral issues, and perception pertaining to the severity of the eating disorders.[85] Innovative training programs (web-based, "EAT" framework for evaluating, assessing, and treating of eating disorders based on motivational interviewing principles) have been developed and evaluated within the dental and dental hygiene student populations with very good results in terms of

improvement in self-efficacy, knowledge of oral manifestations, and treatment options, as well in attitudes toward the secondary prevention of eating disorders.[86–89]

Therefore, inclusion of a dentist trained in the examination and treatment of patients with eating disorders should be considered in multidisciplinary teams.[90] Given that the majority of non-dental professionals specialized in treating eating disorders (psychiatrist, physician, dietician, etc) are not satisfied with the level of oral health education during their formal education, collaborations between oral and non-oral health professionals are strongly encouraged.[91]

6 | PROMOTION OF ORAL HYGIENE AND PREVENTION OF OR AL DISEASES IN PEOPLE WITH EATING DISORDERS

The initial phase of dental treatment for individuals with eating disorders is focused on discouraging obsessive-compulsive toothbrushing behavior, which is destructive to the oral tissues, and on improving oral hygiene proficiency. Toothbrushing is usually intense and frequent in people with anorexia nervosa and bulimia nervosa and is used as a way to hide their embarrassment after an episode of vomiting, or even as a way to provoke vomiting.[47] It is essential to advise them to limit toothbrushing to a maximum of three times daily. They should rinse their mouth with an acid-neutralizing solution or simply use still water instead of brushing after vomiting. The gastric acids, with a pH of 1.5, have a severe impact on the teeth, and so the patient should be encouraged to delay brushing for at least 1 hour after vomiting so that his or her toothbrushing behavior is no longer compulsive. Additionally, patients at risk for dental erosion should always use an additional fluoride source, such as a toothpaste and/or rinse containing fluoride combined with stannous ions.[92] Appointments fully dedicated to oral hygiene with oral and written recommendations are mandatory for patients with eating disorders (Appendix 3). The aim is to prevent dental erosion, dental caries, gingivitis, and gingival recession. The use of an electric toothbrush should be recommended for plaque control, and a system with an overpressure monitoring system is preferred. Our personal opinion, based on clinical practice, is that it is often more efficacious to emphasize an atraumatic brushing method rather than on the frequency. Several studies on eating disorders reported a mean frequency of toothbrushing above three times a day. The gingival phenotype should also be evaluated to identify the risk for gingival recession and to adapt the oral hygiene instructions. Dentists should instruct patients how to use a tongue cleaner and stress the importance of brushing the tongue thoroughly to remove acid residue that collects on the papillae after vomiting.[93]

7 | DENTAL AND PERIODONTAL TREATMENT IN PATIENTS WITH EATING DISORDERS

7.1 | Restorative and prosthodontic treatment in people with eating disorders

Usually, esthetic damage in an emergency situation remains the primary reason for dental consultation in this cohort of patients. Frequently, people with eating disorders do not speak willingly about their disorder to their dentist.[94]

Thus, the first step is to establish good communication with the patient, in a nonthreatening manner, to gather information to evaluate for a diagnosis of an eating disorder.[75] Emergency dental care is usually followed by a staged treatment of various phases that integrates medical care, psychotherapy, nutritional counseling, and dental management.[95]

Various treatment options may be implemented in a second step to help repair the damaged dentition according to the unique clinical presentation of each patient (ie, personalized dental care). The extent of the restorative treatment plan depends on the patient's needs; namely, the level of discomfort and the extent, severity, and depth of the lesions. A temporary treatment plan to stabilize the damaged dentition is usually recommended, since a favorable prognosis for permanent treatment cannot be attained before recovery from the binge- and-purge episodes.[96] Mild cases, not involving a great loss of hard tissue but painful due to enamel loss and dentin exposure (hypersensitivity), need an ultraconservative approach, such as the application of desensitizing agents, and direct restorations with composite or glass ionomer cements, to reduce the hypersensitivity.[29]

The third step of active treatment may require indirect composite restorations using chairside computer-aided design/computer-aided manufacturing processes or porcelain veneers/crowns to improve the esthetic and functional results. In severe cases—where the erosive tooth wear involves the posterior teeth resulting in loss of the vertical dimension—a full-mouth reconstruction, including occlusal rehabilitation with crowns, veneers, or on/overlays, is required (Figure 8).[97–100] The total cost of the restorative/prosthetic treatment should be taken into account, since individuals with eating disorders may face difficult social and financial situations. In particular, studies have reported a high proportion of unemployment in individuals with eating disorders due to psychiatric problems.[47,101]

7.2 | Nonsurgical and surgical periodontal treatment in people with eating disorders

Control of local and systemic risk factors should be considered as part of the periodontal treatment, especially in people with eating disorders that have physical and or emotional comorbidities. The worst periodontal conditions in individuals with eating disorders are frequently associated with a thin gingival phenotype, a compulsive (i.e. frequent and traumatic) toothbrushing habit, and multiple areas of dental erosion. The classical therapeutic approach includes a phase of initial therapy with personal and professional plaque control, a reevaluation, and a phase of periodontal plastic surgery aimed at root coverage and to increase the width of the gingiva. Periodontal plastic surgery in people with eating disorders may be indicated after a careful evaluation of the stage of progression of the disease. To date, no study has evaluated periodontal plastic surgery procedures in individuals with eating disorders; that is, neither patient-related outcomes nor periodontal clinical outcomes (delayed wound healing, percentage of root coverage, etc) have been evaluated. However, individuals with eating disorders can be more sensitive to the auditory, visual, and contact stimuli of the oral surgical procedures under local anesthesia, potentially impacting the outcomes. A questionnaire-based study showed that people with eating disorders had greater levels of dental fear and anxiety than controls did.[102] In addition, a study among patients with eating disorders indicated that third molar surgery caused an exacerbation or a relapse of their disease.[103] Consequently, delaying surgical intervention is recommended

if third molars are asymptomatic in people with eating disorders. If surgery is necessary, the surgeon and other members of the psychotherapy team should establish clear guidelines regarding behavior and postoperative nutrition and monitor the patient's nutritional status. Furthermore, dental professionals should be aware that severe forms of anorexia nervosa are accompanied by a calcium deficiency that requires the intake of oral bisphosphonates, which increase the risk of medication-related osteonecrosis of the jaws.

8 | DENTAL AND PERIODONTAL FOLLOW-UP IN PEOPLE WITH EATING DISORDERS

Numerous studies found a reduced frequency of dental follow-up in individuals with eating disorders (outpatients) compared with the general population.[37,104] Barriers to communication between people with eating disorders and their dentists have been surveyed. It is surprising that only one-third of individuals with eating disorders considered their dentist to be the most helpful source of oral health information.[105,106] It is important to avoid inducing feelings of guilt on the patient and to initiate a positive face-to-face discussion with the patient. Although people with eating disorders may only visit their dental health professional for urgent dental care, a regular recall prophylaxis should be encouraged and maintained.[107] However, oral health professionals should be aware that caring for people with eating disorders is highly demanding from a psychologic perspective and time consuming. Standards for esthetic outcomes of the dental or periodontal treatment are difficult to achieve. Functional and patient-related outcome measures, such as self-esteem and comfort, should be the primary objectives of oral treatment.

9 | CONCLUSIONS AND IMPLICATIONS FOR FUTURE WORK

The role of nutrition on the development of oral diseases, such as dental caries, erosive tooth wear, and periodontal diseases, puts oral health professionals at the forefront for the screening of eating disorders.[108] Oral health professionals play a valuable and unique role with respect to eating disorders and assessment of their oral and physical manifestations, patient communication, referral, case management, and restorative care. Consequently, there is a need to better prepare oral health professionals for comprehensive care of the patient with eating disorders. Oral health professionals must work in close collaboration with the treatment teams caring for individuals with eating disorders, namely psychiatrists, psychologists, nutritionists, and dieticians.

This report demonstrates that the level of knowledge of the oral conditions of individuals with eating disorders must be improved. Furthermore, biological studies on the oral microbiome and on salivary and systemic biomarkers of dental/periodontal destruction (such as ferritin) are needed. From a clinical standpoint, no study has yet tested the association between the level of gingival abrasion and the occurrence of gingival recession. Further, there is a real paucity of data on dental/periodontal complications in people with binge-eating disorder, although it is the most frequent eating disorder worldwide. Finally, there is an urgent need to include conceptual, procedural, and specific skills-based objectives for management of people with eating disorders in the dental/dental hygiene educational

curriculum.[109] Improving the level of oral care in people with eating disorders may contribute to better overall outcomes in nutritional and psychotherapeutic approaches.

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APPENDIX 1: American, British, and French National Guidelines for the Management of People with Eating Disorders

Joint guidelines from the French Federation Anorexia Bulimia (FFAB) and Haute Autorité de Santé (HAS) can be found here:

https://www.has-sante.fr/upload/docs/application/pdf/2012-06/summary_anorexia_nervosa_1_identification.pdf

https://www.has-sante.fr/upload/docs/application/pdf/2013-05/anorexia_nervosa_guidelines_2013-05-15_16-34-42_589.pdf

https://www.has-sante.fr/jcms/c_2581436/fr/boulimie-et-hyperphagie-boulimique-reperage-et-elements-generaux-de-prise-en-charge

The National Institute for Health and Care Excellence Guideline 69 (Eating disorders: recognition and treatment) can be found here: https://www.nice.org.uk/guidance/ng69

The *Practice Guideline for the Treatment of Patients with Eating Disorders*, third edition, can be found here:

https://psychiatryonline.org/pb/assets/raw/sitewide/practice_guidelines/guidelines/eatingdisorders.pdf

APPENDIX 2: The Basic Erosive Wear Examination[33]

Basic erosive wear examination score	Criteria for grading
0	No erosive tooth wear
1	Initial loss of surface structure
2	Distinct defect, hard tissue loss <50% of the surface area
3	Hard tissue loss ≥50% of the surface area

For scores of 2 and 3 dentine is often involved. The highest score per sextant is recorded and the cumulative score of all sextants gives a level of risk from none to high.

APPENDIX 3: Checklist of Oral Hygiene Routine for People with Eating Disorders

- Delay brushing after vomiting for at least 1 hour
- Limit toothbrushing to three times daily
- Rinse the mouth with acid-neutralizing solution or simply still water
- Use a toothpaste and/or rinses containing fluoride (combined with stannous ions)
- Use an electric toothbrush with an overpressure monitoring system
- Use a tongue cleaner to remove acid residue as soon as possible after vomiting

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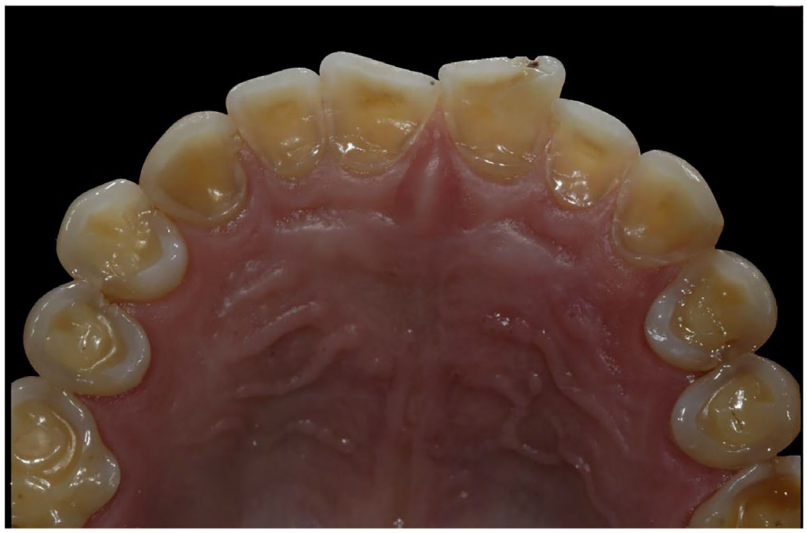

FIGURE 1.
Generalized and advanced erosive tooth wear in a woman (38 years old), with vomiting episodes for more than 5 years related to anorexia nervosa. Anterior lingual and occlusal localizations of the erosive tooth wear are typical of vomiting-associated eating disorders

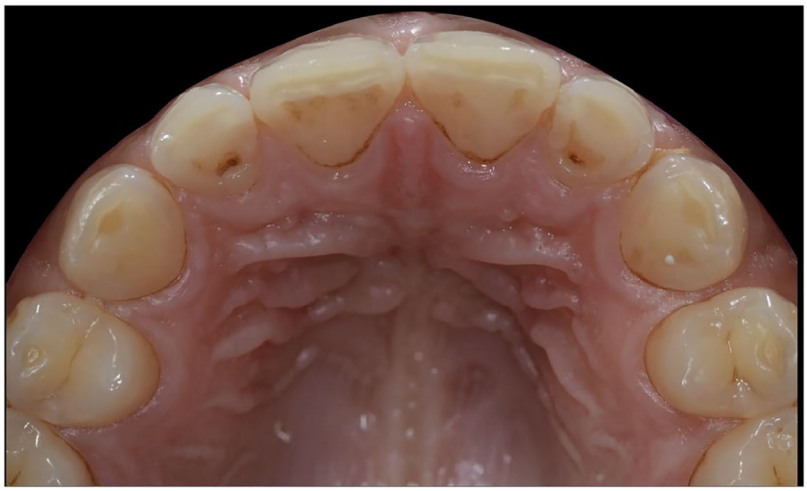

FIGURE 2.
Early stage of erosive tooth wear in a woman (25 years old) with anorexia nervosa

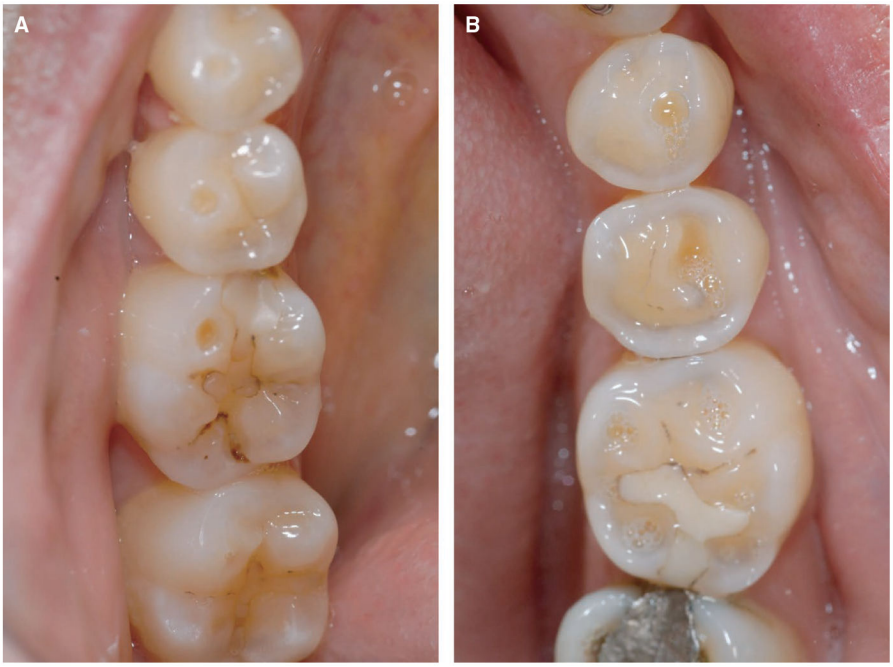

FIGURE 3.

Comparison of differences in mandibular erosive tooth wear caused by the combination of a nocturnal gastroesophageal reflux disease and a lateral sleeping position in a patient with eating disorder: A, left side, showing less advanced wear; B, right side

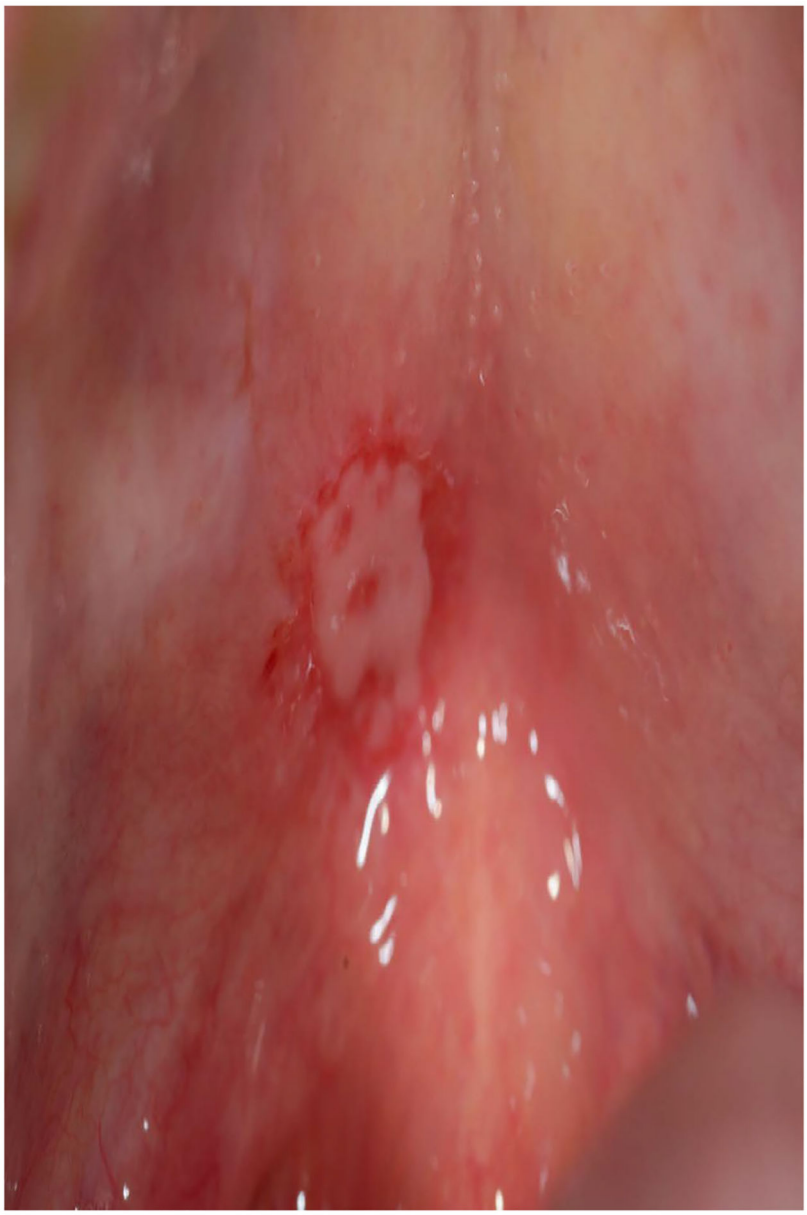

FIGURE 4.
Oral mucosa ulceration of the soft palate (velo). This episodic injury was induced by the patient's finger to provoke vomiting

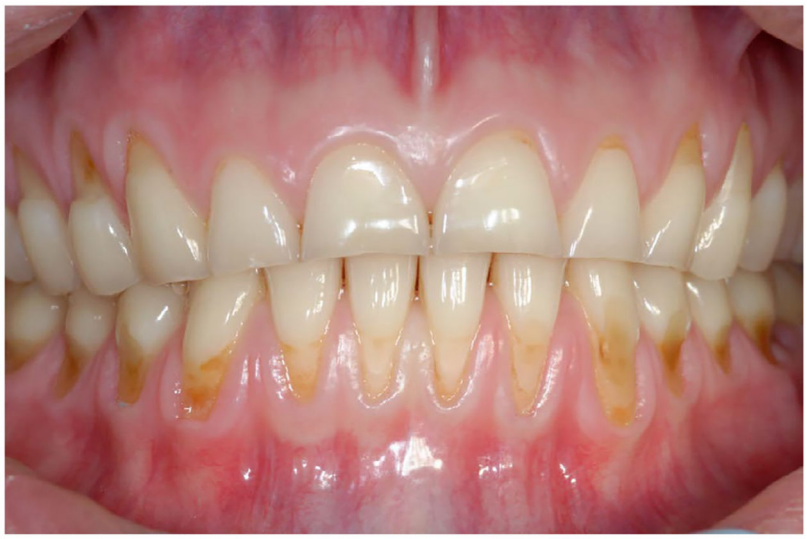

FIGURE 5.
Generalized buccal gingival recessions in a patient with anorexia nervosa. A compulsive (excessive and too frequent) toothbrushing was identified to initiate/favor the progression of the gingival recessions. The acid oral environment may be a modifier/amplifier in the pathogenesis of gingival recession in vomiting-associated eating disorders

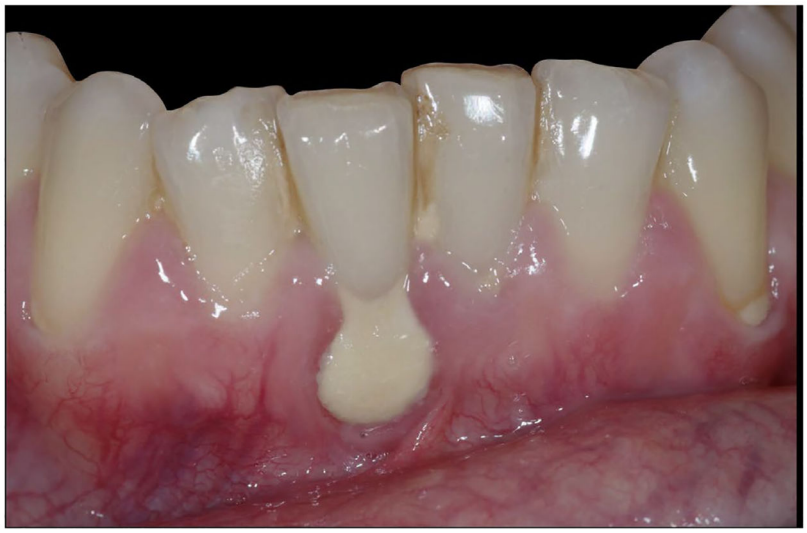

FIGURE 6.

Calculus accumulation at the site of the deep buccal gingival recession in a young woman with anorexia nervosa under enteral food without oral mastication. Gingival inflammation (plaque-associated gingivitis) is directly in relation with this local risk factor

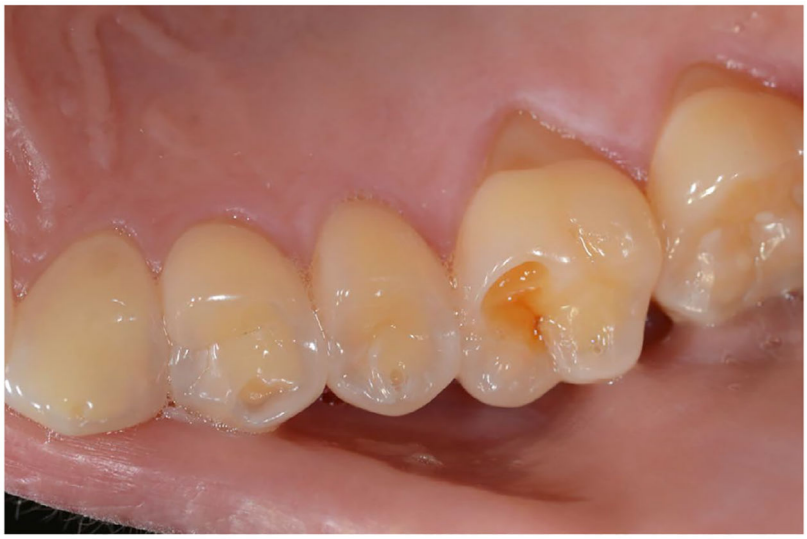

FIGURE 7.
Lingual localization of molar gingival recession that is frequently observed in patients with vomiting-associated eating disorders. Notice the erosive tooth wear on the cusp points of the premolars and molars

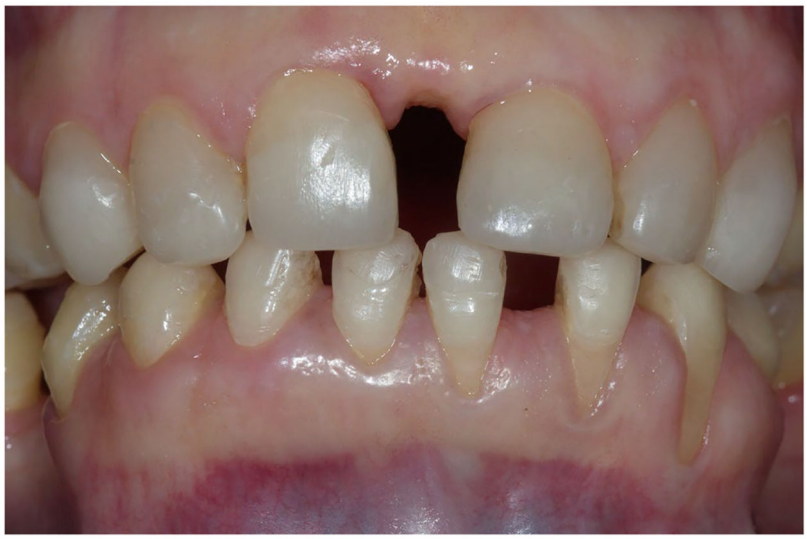

FIGURE 8.

Clinical case of generalized erosive tooth wear restored with direct composite veneers and crowns. Notice the associated gingival recessions induced by a compulsive toothbrushing in a 29-year-old patient become blind and consecutively developing bulimia nervosa. In this case, the patient used to toothbrush after each vomiting episode

TABLE 1

Clinical and biological periodontal outcomes in people with eating disorders

Author(s) Study design/ country	Materials and methods	Study population	Significant clinical periodontal outcomes in people with eating disorder	Significant and nonsignificant biological outcomes in people with eating disorder
Liew et al (1991)[49] Case-control study Australia	**Oral clinical examination** Parotid enlargement, decayed missing filling tooth, facial and lingual plaque index; full-mouth periodontal examination on 6 sites per tooth: bleeding on probing, periodontal pocket depth, gingival recession; community periodontal index of treatment needs **Biological samples and analysis** Stimulated saliva collection, bacterial culture under anaerobic conditions	**n = 30** 15 female inpatients with restricting anorexia nervosa (no vomiting) Mean age 20.1 ± 8.3 y Eating disorder diagnosis: Eating Attitude Test and the Eating Disorder Inventory 15 women without anorexia nervosa and matched for age	**Oral hygiene** More lingual and facial sites with plaque index >0 **Periodontal data** 16.9% of sites with bleeding on probing versus 6.5% for controls 7.9% of sites with gingival recession of 1 mm versus 1.8% for controls; 1.7% of sites with gingival recession of 2 mm versus 0.2% for controls; and 0.6% of sites with gingival recession >2 mm versus 0.0% for controls Lower mean number of healthy sextants (community periodontal index of treatment needs score of 0)	**Salivary biochemical data** Mean stimulated salivary flow rate of 0.7 ± 0.4 mL/min in anorexia nervosa versus 0.9 ± 0.6 mL/min in controls, no significant difference Mean salivary pH of 7.1 ± 0.4 in anorexia nervosa versus 7.6 ± 0.3 in controls, $P < 0.01$ **Salivary bacteriologic data** Counts of *Streptococcus mutans* of $4.1 \times 10^6 \pm 5.7 \times 10^7$ in anorexia nervosa versus $4.2 \times 10^6 \pm 1.5 \times 10^6$ in controls, no significant difference Counts of *Lactobacillus* spp of $6.0 \times 10^5 \pm 1.7 \times 10^6$ versus $5.2 \times 10^4 \pm 1.3 \times 10^5$ in controls, no significant difference
Touyz et al (1993)[50] Case-control study Australia	**Oral clinical examination** Parotid gland enlargement, decayed missing filling tooth, erosion, facial and lingual plaque index[110] recorded for 6 representative teeth; community periodontal index of treatment needs **Biological samples and analysis** Stimulated saliva collection, bacterial culture under anaerobic conditions	**n = 45** 15 female inpatients with restricting anorexia nervosa (no vomiting) and 15 female inpatients with bulimia nervosa Eating disorder diagnosis: *Diagnostic and Statistical Manual of Mental Disorders*, third edition, Revised criteria 15 women without anorexia nervosa/ bulimia nervosa and matched for age Same population as in Liew et al[49] for anorexia nervosa and controls, with bulimia nervosa added (mean age 19.1 ± 3.8 y)	**Oral hygiene** Less lingual and facial sites with plaque index of 0 (51% of facial surfaces with plaque index of 0 in anorexia nervosa, 55% in bulimia nervosa, and 72% in controls; 29% of lingual surfaces with plaque index of 0 in anorexia nervosa, 27% in bulimia nervosa, and 51% in controls **Periodontal data** 16.9% of sites with bleeding on probing in anorexia nervosa versus 9.4% in bulimia nervosa versus 6.5% in controls 10.2% of sites with gingival recession of 1–3 mm in anorexia nervosa versus 3% in bulimia nervosa versus 2% in controls Lower numbers of healthy sextants (community periodontal index of treatment needs score of 0) and higher with bleeding (community periodontal index of treatment needs score of 1) in anorexia nervosa versus bulimia nervosa (score 0: 1.9 ± 1.5, 2.0 ± 1.3, respectively; score 1: 2.7 ± 1.7, 2.0 ± 1.3, respectively) and versus controls (score 0: 3.2 ± 1.0; score 1: 1.7 ± 1.1)	**Salivary biochemical data** Mean stimulated salivary flow rate of 0.7 ± 0.4 mL/min in anorexia nervosa versus 1.0 ± 0.6 mL/min in bulimia nervosa versus 0.9 ± 0.6 mL/min in controls, no significant difference Mean salivary pH of 7.1 ± 0.4 in anorexia nervosa, 7.1 ± 1.7 in bulimia nervosa, and 7.6 ± 0.3 in controls, $P < 0.001$ **Salivary bacteriologic data** Counts of *Streptococcus mutans* of $4.1 \times 10^6 \pm 5.7 \times 10^7$ in anorexia nervosa, $6.9 \times 10^5 \pm 2.2 \times 10^5$ in bulimia nervosa versus $4.2 \times 10^6 \pm 1.5 \times 10^6$ in controls, no significant difference Counts of *Lactobacillus* spp of $6.0 \times 10^5 \pm 1.7 \times 10^6$ in anorexia nervosa versus $6.0 \times 10^5 \pm 1.7 \times 10^6$ in bulimia nervosa versus $5.2 \times 10^4 \pm 1.3 \times 10^5$ in controls, no significant difference
Ohm et al (1999)[37] Case-control study Sweden	**Structured interview** Medical, oral, and dietary habits **Oral clinical, radiographic, and photographic examination** Decayed missing filling surface and erosions **Biological samples and analysis** Unstimulated saliva and stimulated saliva collection,	**n = 133** 81 outpatients (79 women and 2 men), 3 with anorexia nervosa, 7 with anorexia nervosa and bulimia nervosa, 46 with bulimia nervosa, 25 with eating disorders not otherwise specified 26/81 did not experience self-induced vomiting, median age 25 y (range 17–47 y)	**Dental attendance** 65/81 (80.2%) in eating-disorder patients versus 44/52 (84.6%) in controls were regular dental attenders (1 visit in the last 2 years) **Oral hygiene habits** 7/81 (8.6%) of eating-disorder patients brushed their teeth three times or more per day versus 3/52 (5.8%) in controls	**Salivary biochemical data** Unstimulated salivary flow rates <0.2 mL/ min: 50% in eating-disorder patients versus 35% in controls, $P < 0.001$; unstimulated salivary flow rates <0.1 mL/min: 27% in eating-disorder patients versus 2% in controls, $P < 0.001$ Stimulated salivary flow rates <0.7 mL/min: 12% of eating-disorder patients versus 6% of

Periodontol 2000. Author manuscript; available in PMC 2022 October 01.

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	commercial kit Cariotest (GE America, Alsip, IL, USA)	Eating disorder diagnosis: *Diagnostic and Statistical Manual of Mental Disorders*, third edition, Revised criteria 52 healthy volunteers (48 women and 4 men), median age 24 y (range 19–41 y)		controls, no significant difference Stimulated buffer capacity pH <4.5: 35% of eating-disorder patients versus 10% of controls, $P < 0.05$ **Salivary bacteriologic data** Counts of *Streptococcus mutans* >100 000 CFU/mL: 30% of eating-disorder patients, 12% of controls, $P < 0.001$ Counts of *Lactobacillus* spp >100 000 CFU/mL: 42% of eating-disorder patients, 19% of controls, $P < 0.01$
Lifante-Oliva et al (2008)[57] Cohort study Spain	**Oral clinical examination** Decayed missing filling tooth, erosions, plaque index,[110] periodontal pocket depth **Biological samples and analysis** Unstimulated and stimulated saliva collection, commercial kit Cariotest (GE America, Alsip, IL, USA)	**n = 17** 7 female inpatients with anorexia nervosa and 10 with bulimia nervosa (*Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, Revised criteria) Mean age 20.12 ± 5.6 y	**Oral hygiene habits** The majority of the eating-disorder patients reported brushing ≥3 times a day **Oral hygiene** 1/7 (14.3%) in anorexia nervosa versus 1/10 (10%) in bulimia nervosa with calculus **Periodontal data** 2/7 (28.6%) in anorexia nervosa versus 0 in bulimia nervosa with gingival bleeding 1/7 (14.3%) in anorexia nervosa versus 4/10 (40%) in bulimia nervosa with gingival recession	**Salivary biochemical data** Unstimulated salivary flow rates ≤0.1 mL/min: 0% in anorexia nervosa versus 50% in bulimia nervosa Stimulated saliva ≤0.5 mL/min: 0% in anorexia nervosa versus 30% in bulimia nervosa Acid pH in 1/7 (14.3%) of anorexia nervosa versus 2/10 (20%) of bulimia nervosa
Shaughnessy et al (2008)[59] Cohort study USA	**Oral clinical and radiographic examination** Decayed missing filling tooth, dental erosions, simplified oral hygiene index,[111] modified gingival index,[112] periodontal pocket depth	**n = 23** Adolescent and young female patients attending a hospital eating-disorder program with restricting anorexia nervosa Median age 17.6 y (range 15–30 y) Eating disorder diagnosis: *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition criteria	**Oral hygiene** 5/23 (21.7%) of the patients with oral hygiene index-simplified score of >0 **Periodontal data** 4/23 (17.4%) of the patients with modified gingival index of score >0 43% of the patients with gingival recession of 1 mm on ≥3 surfaces	
Johansson et al (2012)[55] Case-control study Sweden	**Structured interview** Questionnaires on oral health status and self-perceived oral health **Oral and radiographic examination** Visible plaque index,[113] gingival bleeding index, decayed missing filling tooth, decayed missing filling surface, erosions, cervical defects **Biological samples and analysis** Unstimulated and stimulated saliva collection, commercial kits Dentobuff (Orion Diagnostica, Espoo, Finland), Dentocult LB and SM (Orion Diagnostica, Espoo, Finland)	**n = 108** 54 outpatients (50 women and 4 men), 14 with anorexia nervosa, 8 with bulimia nervosa, and 32 with eating disorders not otherwise specified Mean age 21.5 y (range 10–50 y) Eating disorder diagnosis: made by professional team at the clinic 54 sex and age-matched controls without risk of eating disorder (based on symptom index of Eating Disorder Inventory-2)	**Oral hygiene** Median visible plaque index of 7.1% (0–51) in eating-disorder patients versus 11.3% (0–39) in controls, $P = 0.001$ **Periodontal data** Median gingival bleeding index of 1.0% (0–38) in eating-disorder patients versus 7.1% (0–30) in controls, $P = 0.01$	**Salivary biochemical data** Unstimulated salivary flow rate ≤0.1 mL/min: 39% in eating disorder versus 21% in controls, $P = 0.04$ Stimulated salivary flow rate ≤0.7 mL/min: 11% eating disorder versus 6% controls, no significant difference **Salivary bacteriologic data** Counts of *Streptococcus mutans* >100 000 CFU/mL: 75% of eating disorder versus 50% of controls, $P = 0.02$ Counts of *Lactobacillus* spp >100 000 CFU/mL: 42% of eating disorder versus 25% of controls, no significant difference

Periodontol 2000. Author manuscript; available in PMC 2022 October 01.

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Szupiany et al (2015)[62] Cohort study Poland	**Oral clinical and radiographic examination** Approximal plaque index (4 grades scale) **Biological samples and analysis** Mucosa smear and gingival fluid collection, bacterial and fungi culture under aerobic and anaerobic conditions	**n = 40** 30 adult female and 10 adult male patients with eating disorder diagnosis: F4.xx, F5x.x, F6x.x, according to International Classification of Diseases-10 criteria	**Oral hygiene** Mean plaque index of 60.1% 45% of the eating-disorder patients exhibited an approximal plaque index >70% (poor oral hygiene), 22.5% an approximal plaque index between 40% and 69% (average oral hygiene), 27.5% an approximal plaque index between 25% and 39% (rather good oral hygiene), and only 5% an approximal plaque index <25% (optimal oral hygiene)	**Salivary bacteriologic data** Fungal infections in 66.5% of the patients All fungal infections were caused by the *Candida* spp fungus Aerobic bacterial infection 26.8% of the patients, and anaerobic bacterial infection 6.7% of the patients *Actinomyces* spp (*A. israeli*, *A. meyeri*, *A. naeslundii*), *Fusobacterium* spp, *Bacteroides* spp, and *Enterobacter cloacae* were identified
Lourenço et al (2018)[56] Case-control study Portugal	**Structured interview** Questionnaires on oral health status **Oral clinical examination** Decayed missing filling tooth, erosions, periodontal status assessed by the index teeth (probing of the buccal surface of the first and second maxillary molars, maxillary right central incisor and mandibular left central incisor, and the lingual surface of the first and second mandibular molars), and oral mucosa **Biological samples and analysis** Unstimulated saliva collection, commercial kit Schirmer Tear Strips (ContaCare, Gujarat, India)	**n = 66** 33 female outpatients, 18 with anorexia nervosa (10/18 with vomiting) and 15 with bulimia nervosa (6/15 with vomiting) Mean age 28.21 ± 10.11 y Eating disorder diagnosis: *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition, criteria 33 age-matched women without previous history or risk of suffering of eating disorder identified using the Eating Disorder Examination Questionnaire Mean age 23.24 ± 3.33 y	**Periodontal data** 2/32 (6.25%) eating-disorder patients with gingivitis defined as visual signs of generalized gingival inflammation, with bleeding and pain after probing versus 0/33 (0%) in controls, no difference 18/32 (56.25%) eating-disorder patients with periodontitis defined as gingival recession or periodontal pocket depth >3 mm versus 2/33 (6%) in controls Mean periodontitis index 0.60 ± 0.498 versus 0.06 ± 0.242 in controls, $P < 0.001$	**Salivary biochemical data** Unstimulated salivary flow rate of 24.00 ± 9.966 mm in eating disorder versus 32.97 ± 4.727 mm in controls, $P < 0.001$
Pallier et al (2019)[34] Case-control study France	**Structured interview** Questionnaires on oral health behaviors **Oral clinical examination** Decayed missing filling tooth, basic erosive wear examination, full-mouth periodontal examination on 6 sites per tooth: plaque index,[114] bleeding on probing, periodontal pocket depth, gingival recession	**n = 140** 70 female inpatients (36 with anorexia nervosa and 34 with bulimia nervosa) Mean age 32.1 ± 9.1 y Eating disorder diagnosis: verified with the Mini-International Neuropsychiatric Interview questionnaire Control group: 70 women matched on age Mean age 30.2 ± 4.7 y	**Dental attendance** 75% of eating-disorder patients reported having visited the dentist at least once a year versus 51.4% in controls, $P < 0.01$ **Oral hygiene habits** 42.9% of the eating-disorder patients brushed their teeth ≥3 times per day versus 7.1% in controls, $P < 0.01$ 2.8% in anorexia nervosa brushed their teeth <2 times per day versus 17.7% in bulimia nervosa, $P < 0.01$ **Oral hygiene** Mean plaque index (percentage of sites) of 71.5 ± 26.8% in eating disorder versus 53.0 ± 20.4% in controls, $P < 0.01$ Mean plaque index (percentage of sites) of 78.8 ± 19.7% in anorexia nervosa versus 63.7 ± 31.1% in bulimia nervosa, $P = 0.02$ **Periodontal data** Mean bleeding on probing of 30.2 ± 26.3% in eating disorder versus 21.8 ± 18.7% in controls, $P = 0.03$ Mean bleeding on probing of 41.3 ± 27.2% in	

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			anorexia nervosa versus 18.5 ± 19.5% in bulimia nervosa, $P < 0.01$ Percentage of sites with gingival recession >2 mm of 2.3 ± 4.1% in eating disorder versus 0.0 ± 0.1% in controls, $P < 0.01$ Percentage of sites with periodontal pocket depth >3 mm of 0.5 ± 1.7% in eating disorder versus 3.1 ± 7.3% in controls, $P < 0.01$ Percentage of sites with clinical attachment level >2 mm of 33.9 ± 18.6% versus 22.9 ± 15.8% in bulimia nervosa, $P < 0.01$	
Vieira Esteves et al (2019)[47] Cohort study Brazil	**Structured interview** Questionnaire on xerostomia **Oral clinical examination** Decayed missing filling tooth, erosions, simplified oral hygiene index,[111] oral mucosa alterations **Biological samples and analysis** Unstimulated saliva collection	**n = 26** 9 women with anorexia nervosa (binge/purging type) and 17 women with bulimia nervosa 64% of the cohort with vomiting 9 inpatients and 17 outpatients Mean age 31 y (range 20–39 y) 64% with vomiting Eating disorder diagnosis: *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition criteria	**Oral hygiene** Mean value of simplified oral hygiene index of 1.13 (range 0–4) Candidiasis was found in 2/26 (7.7%) eating-disorder patients	**Salivary biochemical data** Mean unstimulated salivary flow rate of 0.3 mL/min (range 0.005–0.8 mL/min) Mean pH value of 6.71 (range 5.54–9)