Oral Complications of Eating Disorder

Subjects: Dentistry, Oral Surgery & Medicine

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Eating disorders (ED) patients were found to present related incidences of oral complications. Studies have reported that the possible course of an ED and comorbidities may be an imbalance in the oral environment. The results showed an association between biological (malnutrition, etc.), behavioral (binge eating episodes, vomiting, acidic diet, poor oral hygiene), and pharmacotherapeutic (addiction, hyposalivation) factors that may threaten oral health. Early diagnosis of the past and present symptoms is essential to eliminate and take control of destructive behaviors. Oral changes need to be tackled with medical insight, and additionally, the perception of dietary interactions is recommended.

eating disorders nutrition oral complications oral hygiene

1. Introduction

Medical professionals, including general practitioners (GPs), dieticians, and dentists, may see eating disorder (ED) patients in their practices. Patients attending medical or dental appointments usually show up for a routine check-up, and EDs can be diagnosed by accident. Those affected by eating disorders are young and often unaware of the seriousness of the complications that can occur as a result of a lack of diagnosis, untreated disease, and dietary errors. Romanos (2012) points out that oral symptoms can appear asymptomatically [1]. Implementing treatment standards, and providing professional medical and psychological help, can happen faster if there is knowledge on and awareness of dietary traps. This increases the chance of not only preventing dangerous health complications manifested in the oral cavity but also improving the prognosis of disease treatment.

2. Eating Disorder Characteristics

2.1. Types of Eating Disorders

The most important classifications that describe diagnostic criteria are the American Psychiatric Association (APA) guidelines, provided in the Diagnostic and Statistical Manual of Mental Disorders (DSM 5th edition, updated in 2013), and the International Classification of Diseases (ICD-11), approved by the World Health Organization (updated 2019) [2][3]. According to ICD-11, this group of diseases falls under behavioral syndromes associated with physiological disorders and physical factors. According to the APA, eating disorders are characterized by ongoing abnormal behaviors related to feeding and eating, followed by changes in food intake or impaired food absorption, and this in turn causes disorders of physical health or social functioning. The symptoms of the disorder allow a

clear diagnosis since the diagnosis of a particular disease entity excludes another. Division of EDs according to DSM-5 and ICD-11 subtypes is presented in **Figure 1**, [2][4].

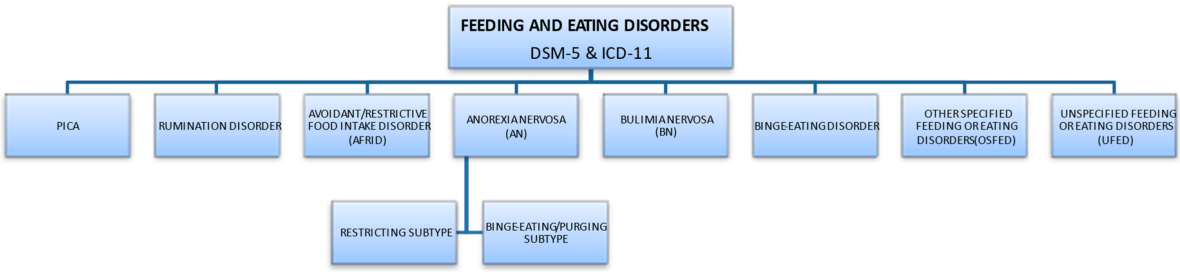

Figure 1. Division of EDs according to DSM-5 and ICD-11 subtypes.

2.2. ED Risk Factors

Eating disorders are characterized by multiple risk factors, which can be biological, psychological, social, and cultural determinants. Individual theoretical models were considered, referring to socio-cultural determinants (ideal body model, gender role), aspects of personal vulnerability (character and behavioral features, including genetic), family and interpersonal background (disorders occurring in the family, e.g., lack of hierarchy, unclear roles, existence of strong ties, turbulent way of solving problems, perfectionism or overprotection of the family, influences of the environment from the same age group), and influence of traumatic life events (e.g., physical, sexual violence) [5][6]. Risk factors predisposing to the disorder in childhood trigger the first onset of the disease in adolescence [7].

Eating disorders often begin with the selective eating of certain foods or following of specific diets, such as vegetarian diets, that seem easy to accept socially [8]. The image of idealized figures created by social media can also lead to behaviors that promote eating disorders [9]. A contributing factor is perfectionism, an ideal that is an important reference point for the individual [10][11][12]. The following risk groups can be recognized: athletes—including more often female athletes, such as those in cycling, judo, gymnastics, and athletics—and people working in modeling, dance training, the military, catering, and show business [13][14]. Biological backgrounds, e.g., disorders of the serotonergic system, are considered in genetic analysis or as correlations with metabolic and immunological features (including glycemic, lipid) and analysis of the fetal period [7]. Meta-analyses indicate the potential importance of single-nucleotide polymorphisms (SNPs) including serotonin receptor gene and serotonin transporter gene (5-HTR2A, 5-HTT), catechol-O-methyltransferase (COMT) polymorphism (Val158Met), and brain-derived neurotrophic factor (BDNF) polymorphism (Val66Met) [15][16]. The heritability in families with mono-dizygotic twins is estimated to be between 48–74% [17]. Genome-wide association studies (GWASs) have identified a chromosome 12 (12q13.2) locus that is relevant to the inheritance of the disease [18].

Researchers' attention is drawn to changes in both the hunger and satiety centers as well as to the associated regulation of appetite and metabolism. This includes anorexigenic (appetite-reducing) and orexigenic (appetite-stimulating) substances secreted centrally in the central nervous system and peripherally (by adiponectin and

enterohormones). Substances that regulate appetite in the short term (e.g., cholecystokinin and other proteins) and long term (hormones leptin, insulin) and enterohormones that interact with the brain–gut axis (e.g., ghrelin, obestatin, neuropeptide B, vaspin phoenixin, spexin, kisspeptin) are involved in the processes of regulating appetite and metabolism [19][20][21][22].

2.3. Epidemiology

Current data show that about 2.9 million people worldwide suffer from anorexia nervosa (AN), with the prevalence in European countries being at the levels of 1–4% (women) and 0.3–0.7% (men) [23][24]. Regarding bulimia nervosa (BN), up to 3% of females and more than 1% of males suffer from this disorder during their lifetime [25]. According to current epidemiological data, EDs are characterized by a long-term course and a high mortality rate [6]. Thus, EDs are an important issue for modern medicine, especially since the incidence rate in Western-civilization countries has been almost declining since the 1970s and the full-blown course is being observed in patients at an increasingly younger age [6][25][26][27].

3. Oral Complications of Eating Disorders

3.1. Oral Hygiene

Until recently, studies have reported conflicting results regarding oral hygiene and periodontal health conditions in ED patients. On the one hand, the ED sufferer exhibits personality traits supposed to lead to overzealous toothbrushing. On the other hand, they suffer from depressive comorbidity with low interest in oral hygiene practices and a higher risk of periodontal diseases. Indeed, periodontal diseases include reversible and irreversible clinical forms of periodontal tissue destruction, known as plaque-induced gingivitis and periodontitis, respectively. The evidence-based pathogenesis of periodontal diseases emphasizes the role of malnutrition [28][29], substance abuse in particular tobacco smoking and alcohol consumption [30], and anxiety [31]. Unbalanced diets with a high consumption of carbohydrates [32][33] and deficiency in vitamins and minerals are frequent in EDs [34]. In addition, mood and anxiety disorders are commonly associated with EDs [35][36]. Hence, ED patients exhibit dietary habits and comorbidities at high risk of periodontal disease. According to the joint classification of the American Academy of Periodontology and the European Federation of Periodontology, plaque-induced gingivitis is an inflammatory response of the gingival tissues resulting from bacterial plaque accumulation located at and below the gingival margin [37]. Managing gingivitis is a primary preventive strategy for periodontitis. Periodontitis is a chronic multifactorial inflammatory disease associated with dysbiotic plaque biofilms and characterized by the progressive destruction of the tooth-supporting apparatus [38]. Its primary features include the loss of periodontal tissue support, manifested through clinical attachment loss (CAL) and radiographically assessed alveolar bone loss; the presence of periodontal pocketing; and gingival bleeding. Without treatment, periodontitis leads to tooth loss.

Case-control studies comparing oral health conditions in EDs with controls have evaluated plaque control with conflicting results. Two studies in the 1990s using the Plaque Index (PI) from Silness and Loe [39], which is a partial recording system prone to bias, found no difference in plaque accumulation between ED and non-ED subjects [40]

[41]. The oral hygiene level was better in ED outpatients than in non-ED controls in two studies again using non-full-mouth recorded data [42][43]. Poorer plaque control in ED inpatients compared to non-ED controls was shown in two studies [44][45]. In a study with a subgroup analysis, according to the ED diagnosis, plaque control was worse in patients with AN compared to patients with BN and controls with a Plaque Control Record (PCR), measured at 79%, 64%, and 53%, $p < 0.01$, respectively [44]. Hyposalivation induced by the disease and the psychotropic medications [46] may make plaque control difficult and the lack of salivary mechanical and biological antimicrobial actions can favor plaque accumulation [47][48]. However, in studies, the toothbrushing frequency in people with EDs was not different [49] or even higher than in non-ED controls [44][50].

3.2. Periodontal Health

Several observational studies have assessed the periodontal status of ED patients, mostly those suffering from AN and BN. Gingivitis case definitions used were often heterogeneous among studies based on partial-mouth examination approaches with the Gingival Index (GI) [51], leading to no difference [41] and less gingival inflammation [42][43] in ED patients. However, the two studies with full-mouth bleeding scores showed a higher occurrence of plaque-induced gingivitis in ED patients [44][45]. In addition, two well-designed case-control studies found that ED patients present significantly fewer healthy (no gingival bleeding after probing) sextants, measured by the Community Periodontal Index and Treatment Needs (CPITN) [52], compared with non-ED controls, but found no difference in the number of sextants with periodontitis (probing pocket depth > 3mm) [45][53]. These results are in accordance with those of Pallier and collaborators, who observed a higher CAL, with significantly more sites exhibiting gingival recessions in the ED group in comparison with the control group, but no difference in the number of sites with probing pocket depth [44]. To sum up, ED sufferers are not at risk of periodontitis but are at high risk of plaque-induced gingivitis and gingival recessions.

Gingival recession is defined as the apical shift of the gingival margin with respect to the cementoenamel junction; it is associated with attachment loss and with the exposure of the root surface to the oral environment [54]. People with AN and BN have an increased prevalence of gingival recession compared to subjects without EDs of the same age [44][45]. Gingival recessions have multiple maxillary and mandibular localizations. A whitish appearance of the free gingival margin of the recession is frequent as a sign of chemically induced tissue damage by intrinsic and extrinsic acidity. Atypical localizations such as the palatine surfaces of the upper molars are characteristic of ED patients [55]. Toothbrushing frequency is one of the main risk factors for gingival recession along with improper toothbrushing duration and force [54]. This could explain the more frequent generalized gingival recessions observed in ED patients.

Induced by both tooth wear and gingival recessions, dentinal hypersensitivity is also more frequently reported by patients suffering from EDs than controls [40][50].

3.3. Oral Mucosal Health

Oral mucosal lesions are often observed in EDs. Factors such as malnutrition and associated deficiencies of vitamins or micro- and macronutrients, dehydration, or pathological behaviors such as provoking vomiting, overbiting, and other parafunctions predispose to their occurrence [56]. Stress levels and addictions like smoking are also important [49].

When analyzing the published results, it is important to pay attention to the profile of the study group. Some differences will exist due to the fact that previous authors analyzed subjects with varying proportions of AN, BN, and EDNOS cases; disease durations; and ages. Panico et al. (2018) [57] and Lesar et al. (2022) [58] observed oral lesions in up to 94% of patients. Their locations can be on the lips, which are dry in as many as 76.2% [59], 93.2% [49], or 69% [42] of subjects. In addition, exfoliative cheilitis, angular cheilitis, and labial erythema are very frequently diagnosed [1][49][50][57][58][60]. The mucosa is anemic, thin, and prone to injury. As a result, it is pale, and more often, ulcerations and hemorrhagic lesions appear on it, the occurrence of which are significantly influenced by the provocation of vomiting and the highly acidic content of vomit [49][57][58][59][60].

Patients are more likely to report mouth irritation, diagnosed as Burning Mouth Syndrome (BMS) [1][50][60]. This disease can be determined by the dentist on the basis of subjective symptoms reported by the patient as it is usually not accompanied by any objective local changes in the oral mucosa. Johansson et al. (2012) [42] showed that the chance of experiencing episodes of burning mouth is 14.2 times higher in patients with eating disorders than in healthy individuals. This is suggested to be associated with mucosal atrophy, xerostomia, and the general disruption of oral homeostasis. Individuals with eating disorders have also been observed to have more common symptoms of cheek and lip biting, as well as impressions on the tongue and linea alba, associated with increased stress levels [49][57][58][60]. A weakened immune system and dysbiosis resulting from a breach in tissue continuity will promote a variety of bacterial, viral, and fungal infections, with the typical clinical presentation of these pathogens [61][62][63][64].

3.4. Dental Health

3.4.1. Dental Caries

Eating patterns in EDs that may affect the onset of dental caries include, on the one hand, avoiding entire food groups and certain macronutrients without a medical reason, participating in fad diets to lose weight, and intentionally skipping meals, which may result in nutritional deficiency [65][66]. The low intake of nutrients such as proteins; vitamins A, C, and D; and minerals may enhance their susceptibility to demineralization in carious processes [67]. On the other hand, alternative eating behaviors, such as slowing down the pace of eating, the consumption of large amounts of high-caloric and high-carbohydrate food during binge eating, and engaging in making yourself vomit to control body weight, may contribute to the retention of food debris, the formation of dental plaque on the tooth surface, the lowering of the pH of the oral cavity environment, and the promotion of demineralization, and thus favor dental caries [65].

Additional factors that may promote dental caries in ED patients are alterations in the composition of saliva [68]; a decrease in saliva's buffer capacity and secretion rate [49][50][69][70], which may be associated with the structural

changes in the salivary glands [42][71]; self-induced vomiting or starvation; and the side effects of psychotropic drugs (i.e., antidepressants, appetite suppressants), diuretics or laxatives [46]. Undoubtedly, poor oral hygiene in patients with eating disorders, presumably also resulting from psychological reasons, may be a contributing factor to dental caries [44][72].

Numerous studies have examined the association between dental caries and EDs, applying commonly used caries assessment measures such as caries prevalence or caries severity based on the clinical or (less often) radiographic recordings and expressed by the sum of decayed (D), missing (M), and filled (F) teeth (DMFT index) or tooth surfaces (DMFS index) [42][44][49][50][71][72][73][74][75] or the International Caries Detection and Assessment System (ICDAS-II) system) [76]. However, the results obtained are contradictory and vary between 37% and 80% depending on the type of ED and the qualification criteria adopted for diagnosis [71][72][75][76][77][78]. Some authors demonstrated significantly higher values in terms of the DMFT index and its D and M components in comparison with control groups [44][50][72].

Relationships between the caries index and vomiting frequency are also inconsistent although it is clear that gastric acid along with high-carbohydrate food or beverages may initiate the carious process [79].

It is important to emphasize that there is no single ED-associated factor, but rather, all the above-discussed contributing risk factors may have inputs in ED. The following factors should be considered: cariogenic diet, oral hygiene level, remineralization exposure, and taking certain types of medication [78].

3.4.2. Erosive Tooth Wear

Erosive tooth wear (dental erosion), the irreversible loss of tooth structure due to chemical dissolution by acids not of bacterial origin, is one of the major oral complications of eating disorders [80]. Erosive tooth wear (ETW) is caused by the frequent contact of teeth with acid from either the stomach (gastric acid) or drinking and eating acidic drinks and foods, particularly outside meal times [81]. EDs, particularly BN, can potentially increase the risk for ETW since they may be associated with bingeing on acidic foods and/or drinks followed by vomiting after every meal [42][82][83][84][85]. Some of these patients use these beverages both in the place of normal meals as well as during intense exercise to lose calories [84]. These characteristics are associated with the frequent contact of the teeth with gastric or dietary acids over an extended period of time, with the consequent wearing away of the dental hard tissue through acid demineralization, initially affecting the enamel, and with the progression to an advanced stage, dentin is exposed (**Figure 2** and **Figure 3**). The exposure of dentin may result in dentin hypersensitivity in response to external stimuli of a cold, hot, tactile, or osmotic nature. The acid of gastric juice brought up due to vomiting, the pH of which can be as low as 1, causes the wear of the palatal surfaces of upper incisors (**Figure 2**), and with lesion progression, the lingual surfaces of premolars and molars become affected, and in more advanced stages, the process extends to the occlusal surfaces of molars and to the facial surfaces of all teeth [85][86]. Erosion due to dietary acid, with pH ranging from 2.7 to 3.8, has no specific distribution pattern, but depends on factors such as the method of application. Thus, EDs in combination with vomiting are associated with an increased

occurrence, severity, and risk of dental erosion [85]. The reported prevalence of ETW among eating disorder patients, particularly those with BN, varies among countries and ranges from 42 to 98% [85].

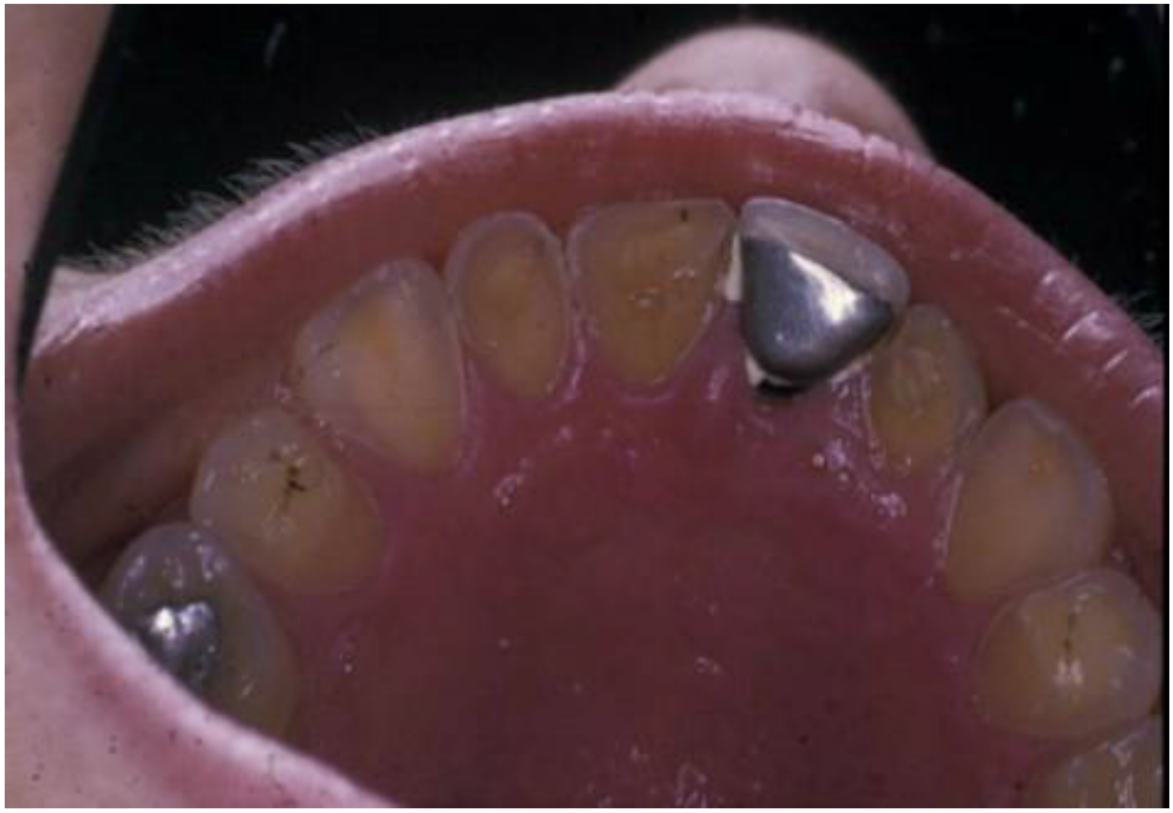

Figure 2. Palatal surfaces of maxillary teeth affected by erosive tooth wear (Courtsey Amaechi BT, UTHSA, San Antonio, TX, USA).

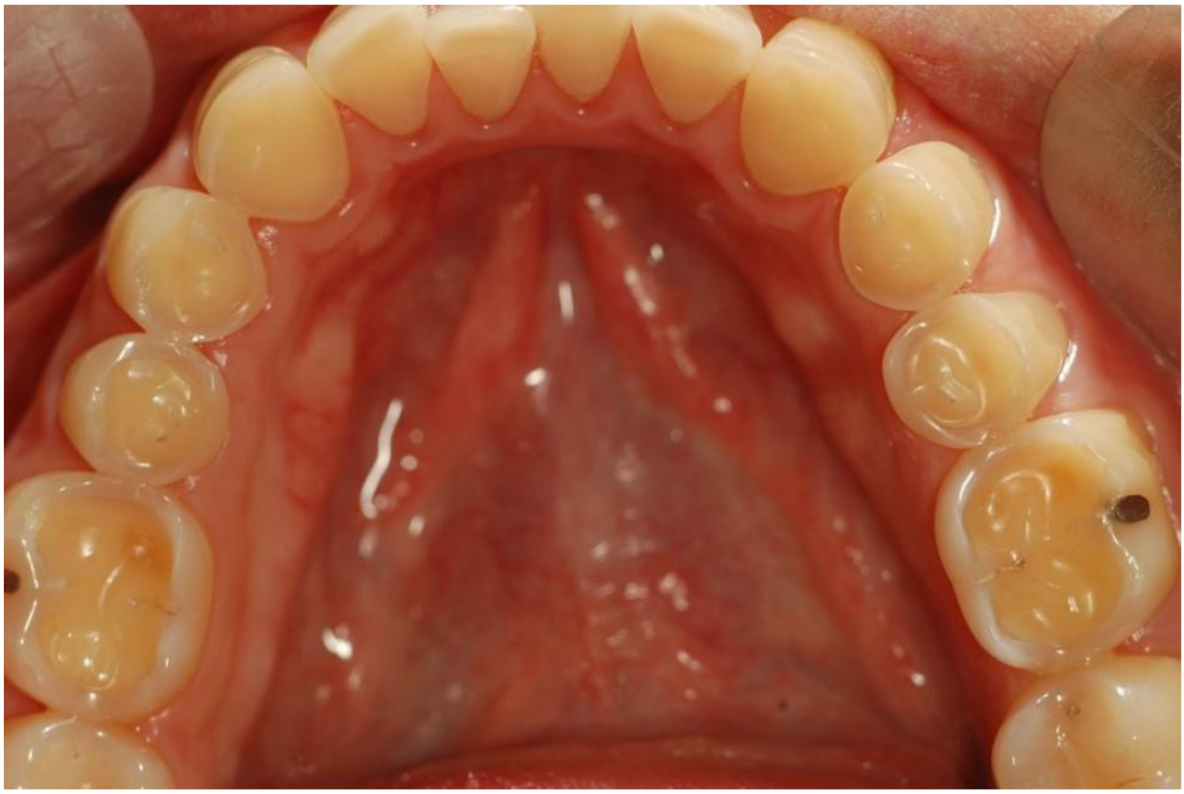

Figure 3. Occlusal surfaces of mandibular teeth affected by erosive teeth wear (Courtsey Amaechi BT, UTHSA, San Antonio, TX, USA).

Main ED's symptoms and oral effects are presented in **Figure 4**.

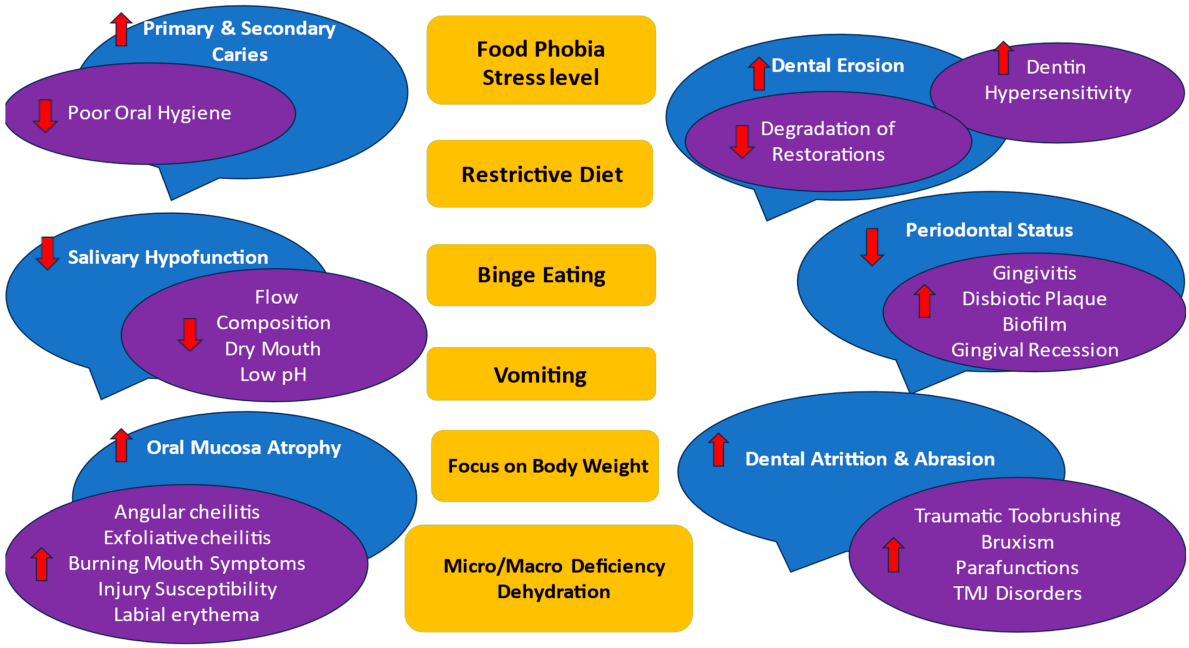

Figure 4. EDs' main symptoms (in yellow) and oral effects (in blue and purple). The following factors may influence any oral complications: subtype of AN, BN, and EDNOS according to nutritional behaviors; vomiting frequency;

disease duration; age and sex of the patient; general health status; pharmacotherapy and their side effects to salivation; and psychosocial profile; as well as cariogenic/acidic diet, individual oral hygiene, and remineralization exposure. Abbreviations: ↑↓ means the ED symptoms increase the oral effect/decrease the oral effect.

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