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Review Article

Eating Disorders in Late-life

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ABSTRACT: Eating disorders are a heterogeneous group of complex psychiatric disorders characterized by abnormal eating behaviours that lead to a high rate of morbidity, or even death, if underestimated and untreated. The main disorders enlisted in the chapter of the Diagnostic and Statistic Manual of Mental Disorders-5 dedicated to "Feeding and Eating Disorders" are: anorexia nervosa, bulimia nervosa and binge eating disorder. Even though these abnormal behaviours are mostly diagnosed during childhood, interesting cases of late-life eating disorders have been reported in literature. In this review, these eating disorders are discussed, with particular attention to the diagnosis and management of those cases occurring in late-life.

Key words: eating disorders, anorexia nervosa, bulimia nervosa, binge eating disorders, eating disorders in late-life.

Eating disorders are a heterogeneous group of complex psychiatric disorders characterized by abnormal eating behaviours that lead to a high rate of morbidity, or even death, if underestimated and untreated [1]. The Diagnostic and Statistical Manual for Mental Disorders-5 (DSM-5) [2] distinguishes, in the chapter "Feeding and Eating disorders", the following illnesses: Anorexia nervosa (AN), Bulimia nervosa (BN), Binge eating disorder (BED) and "Eating disorders not otherwise specified" (EDNOS) that do not meet the criteria for anorexia nervosa, bulimia nervosa and binge eating disorder. The same chapter includes other eating disorders (Avoidant/Restrictive Food Intake Disorder, Elimination Disorders, Pica and Rumination Disorder) that, due to their low frequency among adults, will not be discussed in this review.

Even though anorexia nervosa, bulimia nervosa and binge eating disorder are mostly diagnosed during childhood, interesting cases of late-life eating disorders have been reported in literature. In this review, AN, BN and BED are discussed, with particular attention to the diagnosis and management of those cases occurring in late-life.

Methods

All studies that evaluated eating disorders in the old age were taken into consideration. More specifically, the review included only: (a) manuscripts written in English; (b) original articles; (c) prospective or retrospective observational (analytical or descriptive), experimental or quasi-experimental studies, case series. Editorials and letters to the Editor were excluded from the search. Literature search was performed using PubMed. The following MeSH terms, words and combinations of words, were used in constructing the systematic search: (a) eating disorders; (b) anorexia nervosa; (c) bulimia nervosa; (d) binge eating disorder; (e) eating disorders in the elderly; (f) eating disorders in the older age; (g) late-onset eating disorder; (h) eating disorders in midlife.

Finally, reference lists of key studies were searched manually to retrieve other interesting studies not previously identified through the designed search strategy. The search included only articles published between 1930 and 2013.

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Anorexia nervosa

AN is characterized by: restriction of energy intake leading to significantly low body weight that is less than minimally expected (<85% predicted); intense fear of weight gain; undue emphasis on body shape. Two frequently overlapping subtypes of AN exist: restrictive and binge-eating/purging types [2]. The overall incidence of AN ranges from 5 to 5.4 per 100.000 people/year [3] with a peak incidence of 490 per 100.000 people/year in women aged 15-19 years [4] and a peak incidence of 15.7 per 100.000 people/year in men aged 10-24 years [5]. Furthermore, lifetime prevalence of AN ranges from 2.4% to 4.3% in women [6] and from 0.24% to 0.3% in men [7-8]. It is universally recognized that AN is associated with a variety of medical complications and high mortality rates [9]: the standardized mortality ratio for lifetime anorexia nervosa amounts to 4.37 (95% CI=2.4-7.3) [10]. The most frequent medical complications pertain: cardiopulmonary system (mitral valve prolapse, hypotension, ventricular arrhythmias), electrolyte balance (hypophosphatemia, hypokalemia, hypomagnesemia), gastrointestinal system (gastric dilatation, gastroesophageal reflux, constipation), musculoskeletal apparatus (osteopenia, trabecular bone fractures, weakness, rabdomyolysis), reproductive (amenorrea, infertility) and endocrine (diabetes insipidus, hypoglycemia) systems, oral health (in case of binge-purge subtype) [11]. The aetiopathogenesis of AN is still uncertain. Various risk factors, such as early childhood eating and gastrointestinal problems, history of sexual abuse, depressive and anxiety disorders [12] seem to play a role in the pathophysiology of this disorder.

Clinical observations have reported associations between personality disorders and AN. In particular, women suffering from obsessive-compulsive personality disorder showed an elevated risk of developing AN (OR 4.48, 95% CI 1.22-18.5) [13]. Several neuroimaging studies suggest neurodevelopmental alterations in AN. According to some evidence, this disorder could result from the interaction between predisposing factors and compensatory responses to chronic stress [14]. A recent Voxed Based Morphometry MRI study performed on AN patients demonstrated: (a) increased gray matter volume in an area of the ventral system, the girus rectus/medial orbito frontal cortex, associated with the feeling of pleasantness; (b) increased antero-ventral insula gray matter volumes on the right side; (c) reduced dorsal striatum volume; (d) reduced white matter in right temporal and parietal areas. These brain modifications could lead to an altered brain circuitry associated to pleasantness and reward [15]. Moreover, preclinical studies have examined two main characteristics of AN: reduction in food intake (diet restriction) and hyperactivity. Diet restriction has been related to reduced dopamine levels in hypothalamus, hippocampus, and dorsal striatum. Tyrosine and dopaminergic antagonists normalized anorexia-like behaviours in animal models of AN, but did not restore body weight. However, recent neuroimaging studies demonstrated an increased binding of dopaminergic receptors in the striatum, thus supporting the opposite theory, stating the decrease of intrasynaptic dopamine. This finding could explain the efficacy of atypical antipsychotics, going beyond weight gain, on the symptoms of AN [16].

Even though to date no evidence clearly supports the efficacy of any specific form of psychotherapy, according to the international treatment guidelines, psychotherapy is considered the treatment of choice for patients with anorexia [17]. In support of what above mentioned, a recent multicentre, randomized controlled efficacy trial in adults with AN (ANTOP study) study demonstrated that non-pharmacological outpatient treatment of AN patients with optimized treatment as usual (psychotherapy and structured care from a family doctor in combination), focal psychodynamic therapy, or enhanced Cognitive-Behaviour Therapy (CBT) seemed to be equally efficacious in determining a significant weight gain and an improvement of the overall psychopathology [18].

As to pharmacotherapy, the use of antidepressants appears reasonable in relation to the high rates of comorbidity with depression [19]. Unfortunately, the effectiveness of antidepressants [both tricyclics (TCA) and Serotonin Re-uptake Inhibitors (SSRIs)] on weight recovery has only a low level of evidence [20-23].

Bulimia nervosa

According to the DSM-5 criteria [2], BN is characterized by recurrent episodes of binge eating (eating a larger amount of food than most people would eat in the same time with a lack of control during the episodes) as well as by recurrent inappropriate compensatory behaviours to prevent weight gain (i.e. self-induced vomiting, misuse of laxatives, diuretics or other medications, fasting or excessive exercise). Binge and purging episodes are present at least twice per week for 3 months. The self-evaluation of BN patients is unduly influenced by body shape and weight [2]. Two categories of BN exist: purging and not purging type. The peak incidence of BN in women (aged 16-20 years) ranges from 300 to 438 per 100.000 people/year [4]. The lifetime prevalence of BN ranges from 1.7% to 2.9% [24, 6]. The standardized mortality ratio is 2.33 (95% CI=0.3-8.4) [10]. Similarly to AN, BN have been associated with several risk factors, such as early childhood eating and gastrointestinal problems, elevated weight and shape concerns, negative self-

evaluation, sexual abuse and other adverse experiences, as well as with general psychiatric morbidity [12].

Studies pertaining personality disorders demonstrated a certain association between borderline personality disorders and BN (OR 3.26, 95% CI 1.61-6.62). It has been demonstrated that high levels of impulsivity and inattention relate to severe neuropsychological deficits and eating disorder symptoms, thus representing an additive risk factor for BN [25].

Recent neuroimaging studies performed on BN patients demonstrated the existence of a hypo-responsive reward system to food stimuli (taste reward in particular). Furthermore, patients with BN presented impaired brain activation in the inhibitory control network during the performance of general response-inhibition tasks. These finding suggest that BN patients may binge to compensate a hypo-responsive reward system; the impaired brain activation in the inhibitory control network, in fact, may facilitate the loss of control over food intake in these patients [26].

This hypothesis is also supported by the evidence of atrophy of the caudate nucleus, a region involved in the reward mechanisms, in the processes of self-regulation and in the genesis of binge-eating behaviours in BN patients [27].

It is still unclear whether the structural brain abnormalities observed in patients with AN and BN are reversible after long-term recovery [28].

As AN, even BN is burdened with a variety of cardiopulmonary (atrial arrhythmias, ventricular arrhythmias), electrolytes (hypokalemia, hypochloremia, metabolic alkalosis), gastrointestinal (gastroesophageal reflux, Barrett esophagus, constipation, diarrhea), musculoskeletal (weakness), gynecologic (irregular menses) and oral (gingivitis, dental caries, sialadenosis) complications. As a result, the management of the disorder should be multidisciplinary [11].

The form of psychotherapy with the highest level of evidence for the treatment of BN is CBT [29]. In this regard, it has been demonstrated that CBT leaded to a more rapid correction of abnormal eating behaviours with the advantage of being relatively cheap and more acceptable for the patients than family therapy [30]. In addition, CBT has been demonstrated to be superior also to psychoanalytic psychotherapy, despite the latter entails a great number of treatment sessions and higher costs [31]. Even though standard psychological treatments have been successful in treating several main features of BN, emotional regulation or impulsivity appear to be relatively stable tracts. Due to the similarity, in terms of impulsivity, between impulse control disorders and BN, a pilot study demonstrated that video game therapy determined an improvement in the emotional regulation, in the heart rate variability and in the respiratory rate, also determining a reduction of impulsivity measures in BN patients [32].

As to pharmacological treatment, evidence strongly support the efficacy of antidepressants (fluoxetine in particular) on core bulimic symptoms (binge eating and purging) and associated psychological features in the short-term [33]. Other studies demonstrated a certain efficacy of trazodone, fluvoxamine, desipramine [34-35] and of the anticonvulsant topiramate [36]

The association of pharmacotherapy and psychotherapy or self-help seems to guarantee the best results, even if the limits pertaining the variety of the study designs facing this topic, with the consequential difficulties in replication, must be considered [37].

Binge eating disorder

The core psychopathology of BED is represented by binge eating episodes that are characterized by an unusual large food intake within a discrete period of time while experiencing loss of control. Differently to BN, BED is not associated to inappropriate compensatory behaviours performed to counteract the potential weight-gaining effects of bingeing and, as a consequence, it is commonly associated to overweight and obesity [2]. BED is considered to be the most frequent eating disorder, with a lifetime prevalence ranging from 1% to 3% [38].

Several studies have showed a genetic influence in the development of BED; this disorder, in fact, has been demonstrated to present a high estimated heritability [39-40]. Different acquired risk factors have been associated to the disorder, in particular depression, anxiety, anger, impulse control difficulties, poorer body image, lower self-esteem, greater body dissatisfaction and recent stress [41-43]. Moreover, impulsive personality traits and borderline personality disorder were found to be strongly associated with BED [44]. The risk of death for individuals suffering from BED is mostly related to the complications of obesity. Moreover, obese individuals with binge eating disorder frequently experience impairments in mood and quality of life [45] and, as a vicious circle, major depression was found to be associated to an increased risk for premature death, primarily due to cardiovascular complications. Previous studies, in fact, have demonstrated that depressed patients are more likely to present metabolic syndrome (visceral obesity, dyslipidemia, insulin resistance, and hypertension), through an unclear mechanism [46].

From a neurobiological point of view, BED individuals present a constitutional decrease in basal dopamine levels, resulting in a hypothetical reward deficiency state. This may lead to an increased risk for self-medication (whether conscious or subconscious) with highly palatable foods. However, the prolonged dopamine

receptor stimulation due to long-term consumption of sugars/fats could result in the down-regulation of dopamine receptors, thus leading to tolerance, with the consequential necessity of a greater exposure to rewarding stimuli (escalation of consumption) [47-48]. The lack of cognitive control (that could be the basis of the binge behaviour) has been evaluated also trough neuroimaging studies that demonstrated, in BED patients, a relative hypoactivity, during cognitive performances, in the brain areas involved in self-regulation and impulse control (ventromedial prefrontal cortex, inferior frontal gyrus, and insula) [49]. As to therapy, psychological approaches, in particular modified CBT, interpersonal therapy and dialectical behaviour therapy, have been studied with positive results [50]. In addition, recent studies supported the role of the mindfulness approaches in the improvement of the control of abnormal eating behaviours in obese and BED patients determining, in particular, a reduction of food intake and a healthier food selection, with a consequent weight loss [51-52].

Concerning pharmacotherapy, SSRIs are successful in transiently reducing binge-eating and body weight [53-55]; the SNRIs (Serotonine- Norepinephrine Reuptake Inhibitors) duloxetine was found to be effective in the improvement of abnormal eating behaviours and in the reduction of the illness severity, with an unclear effect on body weight [50-56]. Promising results have been obtained with topiramate [57] and bupropion; the latter, even if in a retrospective study, has been demonstrated to be effective in promoting weight loss in depressed patients with BED, with better outcomes in those patients presenting an higher body mass index [58]. On the other hand, the positive clinical effects of this drug in terms of eating disorder features and associated depressive symptoms are still debated [59]. Finally, in non-responders or severe cases, bariatric surgery, always followed by psychological/pharmacological support, could be useful [60].

Midlife and late-life eating disorders

Despite the fact that eating disorders are more frequently diagnosed during adolescence, it is widely accepted (with some scepticism) that eating disorders may be present at any age. The question is whether these presentations represent a continuation of a lifelong disorder or a late onset disease. Usually, in fact, the typical age at onset of eating disorders is between 16 and 25 years [61]. Even the definition of late onset eating disorder is unclear considering the literature data; there is, in fact, a substantial heterogeneity of the criteria used to identify the typical age-range at onset [62-63]. Anyhow, an onset after the age of 40 is generally considered late onset [56]. The reported prevalence of late-life eating disorders ranges from 1.8% [65] to 3.8% [62]. The most common eating disorder across aging is BED [66]. Eating disorders in the older adults can be divided into to groups: early onset (which continues or recurs in old age) and late onset (which develops for the first time later in life). The clinical core of eating disorders in the older adults are consistent with the DSM-5 criteria [2]. Late-life eating disorders present some differences if compared to eating disorders occurred "in the typical age": the studies analyzing the risk factors demonstrated less family history of major psychiatric disorders [67] and comorbidity [68] but more marital conflicts, separation or divorce [69]. A recent study on late-life eating disorders showed that the individuals affected by these illnesses reported significantly fewer weekly vomiting episodes and fewer self-harming behaviours, but a more frequent history of past or current obesity than early onset individuals [70]. However, the rates of purging (laxative abuse) was found to be higher among patients with late-life eating disorder, probably because mature people are more likely to know the existence of laxative drugs and the access to these substances is easier for them [71]. It has been demonstrated that a high percentage of individuals presenting late-life eating disorders reported comorbid depression and anxiety [72]. General complications of late-life eating disorders are the same of early (typical) onset, even though some case series reported less severe eating disorder symptoms in terms of weight, fear of food and body image [73], as well as less substance abuse, self-harming behaviours and suicide attempts [74]. Obsessive-Compulsive personality disorder was found to be associated with late-life eating disorders as well as it has been associated with early-typical onset of eating disorders [75]. In addition, even if the medical complications of late-life eating disorders are the same underlined for early onset eating disorders, the risk of death for cardiovascular, metabolic, gastric and bone disorders is considerably higher [63]. Special attention should be given to BED patients for the cardiovascular risk related to obesity. Even though they lack of empirical support, several psychodinamic mechanisms have been postulated to explain eating disorders in the older adults. Some Authors argued that women in their late-life become more and more devalued by the society, with a growing sense of envy, loss and preoccupation with body image [76]. Other underlying processes include the need of control and attention. Futhermore, it has been suggested that eating disorders in the older adults have the same meaning as an indirect suicide [77]. Other studies hypothesized that a protective self-presentation style could be considered as a risk factors for abnormal eating behaviours through socio-cultural mediators, such as idealization of appearance and thinness [78]. More recent studies have underlined the negative effect of the "thin-

ideal" transmitted by magazines, television and internet [79]. In addition, according to the feminist theories, special attention should be given to the gender-role attitudes. In fact, the women idealizing the "Superwoman" construct (outstanding realization, ultra-thin body) are more likely to present an abnormal eating behaviour [80]. Among the risk factors for late-life eating disorders, body dissatisfaction seems to play a certain role; in fact, women in their midlife frequently report to be more dissatisfied with their actual body than they were when they were younger, often desiring to be thinner [81]. Several factors seem to exert a "protective" role from body dissatisfaction: being mother, having a stable partner, having a secure career. These factors could reduce the attention given to "appearance", body image and aging by the women presenting the above mentioned characteristics [82]. On the contrary, the signs of aging (wrinkles, hair loss, changes to body fat distribution) [83] and the emphasis given to appearance, self-care and body mass index, represent risk factors for body dissatisfaction [84]. The fear of aging has been positively related to disordered/disinhibited eating and drive for thinness in middle-aged women. Literature data on adult males regarding body dissatisfaction are poor. Even though men are generally considered to be less interested in body appearance [85], they can attribute importance to the goals of losing weight and increasing muscle tone, with a strict correlation between male's body image and sense of self [86]. Concerning women, some critical stages of life must be considered as stressors that could facilitate the occurrence of an eating disorder. Among these: engaged period, marriage, pregnancy, childbirth, menopause [87], separation or divorce [88]. Pregnancy and menopause are the most discussed topics in literature. As it is well known, the social, psychological and biological changes occurring during pregnancy could influence the perceptions of the body determining the remission [89] or the exacerbation of pre-existing abnormal eating behaviours or favoring the onset of an eating disorder [90-91]. A relatively recent study reported a high incidence of BED in the first half of pregnancy, also demonstrating a certain association between BED, anxiety, depression, low life satisfaction, deficient social support and weight-related factors [92].

Also menopause, probably for the hormonal imbalance, has been associated to the development of eating disorders. It is in fact well known that sex hormones exert a fundamental role in the control of eating behaviours. Estrogens, in particular, reduce food intake, whereas progesterone and testosterone increase it, promoting bulimia and abdominal obesity [93].

Concerning the treatment, no clinical trials on the treatment of eating disorders in the older adults exist. Nevertheless, once diagnosis is made, treatment should be as "aggressive" as for early onset eating disorders, obviously taking into consideration the problems pertaining the management of a disorder occurring in an older patient (e.g. dose adjustment, drug choice, general health condition, comorbidity, use of multiple medications for various medical conditions).

Conclusion

Due to the high risk of complications, late-life eating disorders should not be neglected.

The risk of underestimation is increased by the fact that the older adults may be embarrassed for having a "teenage" pathology. Older individuals with eating disorders, as underlined, may present with different clinical pictures if compared to younger patients; special attention should be given to an unexplained weight loss in a woman in her late-life, as well as to depressed or anxious women (which represent high-risk populations for eating disorders), in order to reduce morbidity and improve the patients' quality of life. In conclusion, clinicians must consider late-life eating disorders when, in their clinical practice, they are called to perform a holistic assessment of the older adults.

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