4. Definition and Classification of Eating Disorders

Key Questions:

4.1. How are eating disorders defined and classified? What are the shared and specific clinical features of each type?

4.2. Etiopathogeny of eating disorders: What are the main risk factors?

4.3. What are the most frequent comorbidities of eating disorders?

4.1. How are eating disorders defined and classified?
What are the shared and specific clinical features of each type?

Eating disorders are a group of mental disorders characterised by disordered eating behaviour and the development of behaviours aimed at managing weight. These behaviours lead to physical problems and deteriorated psychosocial functioning of the patient. Current classifications of eating disorders include AN, BN and other less specific disorders known as EDNOS (See chapter 7, “Diagnosis”).

The first descriptions of AN date back to the 17th century, when Morton determined that the origin of this disorder, in contrast to other states of malnutrition, was a disturbance of the nervous system accompanied by sadness and pre-occupation. In the 19th century, it was described as an individual psychopathological picture similar to the one observed today, though it was believed to be a mood disease. Although the history of BN is much younger, the number of affected patients has been growing significantly in the past few years, possibly due to its less dramatic evolution and the ease with which affected individuals go undetected.

AN is an eating disorder that manifests itself as an uncontrollable desire to be thin, accompanied by the voluntary practice of procedures to achieve this goal: a strict, restrictive diet and purging behaviour (self-induced vomiting, laxative abuse, use of diuretics, etc). Despite gradual weight loss, patients present an extreme fear of becoming obese. They present body image distortion and an extreme pre-occupation with diet, figure and weight, and thus engage in food avoidance behaviour by means of compensatory actions to compensate for what they have ingested (extreme physical hyperactivity, purging behaviour, etc). Patients are not usually aware of the disease or the risks their behaviour entails. Their attention is focused on ponderal loss, leading to deficient nutritional states and ultimately to life-threatening risks. Usually there are previous personality traits that tend towards conformism, the need for approval, hyperresponsibility, perfectionism and poor response to internal needs.
BN is an eating disorder characterised by binge-eating episodes (voracious and uncontrolled eating), in which a large amount of food is consumed in a short period of time and usually in secret. Affected individuals attempt to counteract the effects of over-eating by means of self-induced vomiting and/or other purging methods (misuse of laxatives and diuretics, etc.) and physical hyperactivity. These individuals present pathological concern about weight and figure. BN does not necessarily lead to weight changes. Patients can present normal, low or excess weight. BN tends to be a hidden disorder given that it goes easily undetected and patients deal with feelings of shame and guilt. Patients usually seek help when the problem has progressed to advanced stages.

EDNOS are usually incomplete AN or BN pictures that do not constitute a complete picture. They are not, however, less serious. EDNOS include disorders such as the frequent use of inappropriate compensatory behaviour (after eating, chewing and spitting out small amounts of food) and recurrent compulsive eating episodes without compensatory behaviour. BED is a disorder that is currently in the study phase to determine if it is a disorder different from other EDNOS or simply a light form of BN. The main difference with BN is the absence of compensatory mechanisms, which eventually lead the patient to inevitably become overweight or obese.

What are the shared clinical features of AN and BN?

At a psychopathological level, AN and BN share an excessive pre-occupation with image and weight, which reaches irrational extremes in AN (not in BN). At a physical level, malnourishment and its potential complications are always present in AN and possible in the case of BN. There are also mixtures of anorexic and bulimic behaviour that are hard to differentiate, although ponderal loss and secondary malnutrition point to AN.

What are the specific clinical features of AN?

- Refusal to maintain normal body weight or to gain ponderal weight and distortion of body image: patients with AN are focused on their body weight, on the dread of gaining weight or becoming fat (a fear that is compounded as the patient loses weight) and on the desire to lose weight. An altered body experience is a core factor in the concept of eating disorders. Dissatisfaction with one’s own body image is the main reason for weight loss, especially if it is associated with low self-esteem. It is a phobic fear of becoming fat and losing control over food. These ideas lead to behaviours aimed at achieving ponderal loss.
– Other psychopathological disturbances: symptoms such as depressive mood, apathy, difficulty concentrating, anxiety, irritability, social alienation, loss of sexual drive, brooding and/or obsessive rituals regarding food are usually present.

– Physiological disturbances: as a consequence of ponderal loss there is malnutrition, leading to secondary disturbances, especially hormonal and metabolic.

– Amenorrhoea (primary or secondary): a characteristic symptom of the disease, it can appear in up to 70% of cases when there is significant ponderal loss. 20% of patients present amenorrhoea without prior detectable weight loss. This is due to hypogonadotrophic hypogonadism originated by a hypothalamic dysfunction that is considered to be primarily produced by a reduction of calorie intake and weight loss.

– Physical hyperactivity: Is usually present from the beginning of the disease. These individuals present two types of hyperactivity: deliberate physical exercise aimed at burning calories and losing weight, which can be practiced alone, presents obsessive characteristics and occurs only in a minority of patients, and involuntary hyperactivity secondary to malnutrition, which is an automatic response that manifests as persistent restlessness similar to that observed in laboratory animals subjected to hypocaloric intake.

What are the specific clinical features of BN?

– Loss of control over eating behaviour, which serves to understand recurrent binge-eating episodes. During these episodes, patients eat large amounts of food in a short period of time. The duration of these episodes can vary (approximately 2 hours), but is always within a 2-hour period; in fact, eating small amounts of food throughout the day is not considered a binge-eating episode, even though the episode does not have to occur in one place solely (for example, an individual may begin the episode in a restaurant and end it at home). Overall, even though the types of food eaten during the episode vary, in most cases it is sweets and high-calorie foods that are ingested, such as ice-cream or cake; quantity can also vary but may amount to several kilograms. Binge-eating episodes can occur at any time of the day but are more frequent at mid-afternoon and onward. They may be triggered by dysphoric moods, interpersonal difficulties, intense hunger or after restrictive diets or feelings related with weight, body figure or food. Episodes are accompanied by a feeling of losing control and can temporarily reduce dysphoria, but are always followed by feelings of guilt, self-contempt or depressive mood.

– Presence of compensatory mechanisms aimed at avoiding weight gain: 80% to 90% of patients engage in self-induced vomiting after the binge-eating episode. The immediate effect is relief of physical discomfort and decreased fear of gaining weight. The most frequent way of self-inducing vomiting is by inserting the hand to trigger the nauseous reflex. With time, it becomes easier to induce vomiting, and may be done by simply compressing the abdomen. Other mechanisms used to avoid weight gain are the misuse of laxatives and diuretics, the use of other anorexigenic drugs, excessive exercise or fasting. Both laxatives and diuretics cause dehydration and the resulting feeling of ponderal loss, but when their use is interrupted reflex fluid retention occurs and thus, their use is perpetuated.
- Persistent preoccupation with weight and figure: it is morbid dread of becoming fat. Most BN symptoms are secondary to these beliefs and their modification is probably essential to achieve complete resolution of the disorder.

4.2. Ethiopathogeny of eating disorders: what are the main risk factors?

Like other mental disorders, eating disorders have a multiple and somewhat uncertain etiology (yet unclear). According to studies, its ethiopathogeny involves several biological-genetic and vulnerability factors, psychological characteristics, sociocultural aspects and stressors. The specific impact of each one of them is yet to be determined.

Eating disorders tend to begin in adolescence, although we are seeing a gradual increase in the frequency of cases beginning in adulthood and childhood. These disorders usually affect the female population (for every 9 cases of eating disorders in women, there is 1 in men, approximately).

At present, several risk factors have been determined for these disorders. The designs of some of the studies used have been cross-sectional and enable us to establish associations and not causal relationships.

Biological factors

Research on biological factors has focused mainly on genetic factors and neurobiological disturbances.

Studies conducted on families show a higher frequency of eating disorders among relatives of individuals with eating disorders than among control subjects, leading to the conclusion that there must be family vulnerability to these disorders.

Case-control studies using molecular genetics have found a positive association between the presence of certain polymorphisms and a greater vulnerability to developing AN. The most studied are the serotonergic system (5-HT; regulates appetite, stress response, sexual behaviour, obsessive symptomatology, mood, etc), the dopaminergic system and neurotrophins (especially BDNF, NTRK2 and NTRK3).

Equally positive results have also been obtained in chromosomes 1, 2 and 13. However, these results are not yet conclusive. Genes seems to account for 60% to 70% of vulnerability in the case of AN. In the case of BN, susceptibility is found in chromosome 10. There is discrepancy regarding the early appearance of menarche as a risk factor in girls.
Sociocultural factors

The studies identified the following sociocultural factors as risk factors for eating disorders: overprotective, rigid and demanding, conflictive and poorly cohesive family models, destructured families (divorced parents), family history of mood disorders and obsessive-compulsive symptomatology, eating disorders (especially in mothers), atypical dieting and/or eating behaviour in the family (parents concerned about weight), obesity (especially in mothers), alcoholism (especially in fathers), inconsistent eating habits during childhood, careers and/or activities during childhood-adolescence that place too much emphasis on slimness and/or weight.

Psychological factors

The following psychological factors have been associated with eating disorders: mood disorders, personality disorders, obsessive-compulsive disorders, impulse control disorder, following an anomalous and restrictive diet and pre-occupation with the body, personal history of eating difficulties, extreme rigidity, perfectionism, social alienation and low self-esteem.

Potentially stressful life events

Regarding the potentially stressful life events associated with eating disorders, the following stand out: sexual and/or physical abuse during childhood, criticism towards one’s body and a history of life crisis.

According to a review of eating disorder risk factors, some of the previously mentioned factors have shown consistent results in the prediction of eating disorders, both in longitudinal and cross-sectional studies: gender, ethnicity (except Asians), eating problems and gastrointestinal disorders at early ages of childhood, sexual abuse and other adverse life experiences, low self-esteem, general psychiatric morbidity, high weight and bodily dissatisfaction and dieting.

Of all the different explanatory models of AN, Garner’s (1993) suggests that AN is the result of the interaction of three types of factors: predisposing, precipitating and perpetuating. Predisposing factors confer susceptibility to AN.

Some of these are determined by a genetic component, such as the female sex. Other predisposing factors are individual, family and cultural. Precipitating factors, such as dissatisfaction with body weight and shape, interact with predisposing factors in such a way that they condition affected individuals to the point where they decide they must lose weight and restrict eating. Once AN has initiated, gradual weight loss leads to complications derived from malnutrition. The disorder’s multidimensional consequences (physical, psychological and social) are both perpetuating factors of the disorder and boosters of predisposing and precipitating factors.
Current literature suggests that eating disorders are partially determined by both sociocultural and biological-genetic factors (the latter would explain 60%-70%)\textsuperscript{99-101}. However, a part of the variance is not explained by any of these factors, leading to the performance of studies that assess the relevance of non-shared environmental factors which would explain why twins, who have been raised in a similar family environment, can differ in terms of eating behaviour, pathological in some cases and normal in others. Amongst these factors, the following would be included: parents treating each sibling differently, the subjects’ personality and temperament, the subjects’ relational style, experienced stressful situations and specific differential characteristics\textsuperscript{81, 86}. Published scientific evidence shows that non-shared environmental factors are more relevant than shared factors. In this respect, 24% to 42% of variance in AN\textsuperscript{102} and 17% to 46% of variance in BN\textsuperscript{90} would be explained by the influence of non-shared environmental factors.

4.3. What are the most frequent comorbidities in eating disorders?

Comorbidity in eating disorders is common, both of mental and organic origin. The next section describes the most frequent associations. The therapeutic approach required in these cases will be addressed in the chapter dealing with treatment.

Substance-related disorders

Abuse of illegal substances and chemical dependence is common in eating disorders, especially in BN and similar clinical pictures. In the beginning the use of stimulants (amphetamines and cocaine) is related with attempts to decrease appetite, but later it is linked with impulsivity-associated BN pictures. Up to 40% of diagnosed patients (AN or BN) admit to abusing or having a dependence on alcohol or illegal substances\textsuperscript{103-105}.

Anxiety disorders

Anxiety is so present in eating disorders it is hard to decide if it is a specific clinical component of these disorders or if it is a comorbid condition. Eating disorders present specific phobia clinical pictures (phobia of certain foods, social phobia, etc.) and others such as claustrophobia or unrelated simple phobias. Panic attacks or anxiety crises present a similar situation. In some cases they are linked to eating disorders and in others they are truly comorbid. Studies report very varied anxiety prevalences that range between 10% and 40%, depending on the measurement tools and inclusion criteria used\textsuperscript{106-109}.

Obsessive-compulsive disorder (OCD)

Patients with eating disorders, especially AN, present significant prevalence of obsessive personality traits (See personality disorders). Many of these patients are rigid, strict, organized, responsible, constant, intransigent and intolerant, personality traits that predispose and
accompany eating disorders. However, in a considerable number of patients, up to 40% of them diagnosed with AN, obsessive-compulsive disorder that meets comorbidity criteria is present.\textsuperscript{110-112}

**Personality disorders**

Approximately 30% of eating disorder cases present personality disorders.\textsuperscript{113-115} There is a high prevalence of patients who meet BN criteria and present an associated personality disorder, especially borderline and histrionic.

**Mood disorders**

Depression is closely linked with eating disorders. It is hard to think of AN or BN without a depressive clinical picture. As in the case of anxiety, in some cases depression predisposes and in others it is linked with the clinical manifestation of eating disorders or presents itself as a comorbid condition. Prevalence of depression ranges between 40% and 80% and occurs more frequently in BN.\textsuperscript{116, 117}

**Impulse control disorder**

Some of the behavioural disturbances of eating disorders entail a loss of self-control (overeating, purging behaviour, self-aggression, etc.). Other impulse control disorders occur with eating disorders, such as kleptomania or trichotillomania.\textsuperscript{118, 119}

**Diabetes mellitus**

DM is present in the genesis and evolution of eating disorders and also in treatment, which will have to be adjusted to this physical condition. Studies show very varied prevalence rates. Between 0.5% and 7% of cases of AN and BN present Type 2 DM. This percentage reaches up to 20% in EDNOS. Up to 9% of obese diabetics present an eating disorder.\textsuperscript{120-122}

Type 1 DM is a risk factor for eating disorders (three times more risk in BN and two times more risk in subclinical EDNOS\textsuperscript{120} than the population without type 1 DM). Type 2 DM is a risk factor for engaging in inadequate eating behaviours. When type 1 DM and an eating disorder coincide (OR: 4.8; 95% CI: 3.0 to 7.8) physical complication such as retinopathy increase. In another 4-year longitudinal study (N=91) on a cohort of patients with both pathologies (ED and type 1 DM), 60% were associated with retinopathy.\textsuperscript{123} In a further study conducted by Nielsen, 2002, mortality in patients with type DM at 10 years follow-up was 2.2 per 1,000 inhabitants/year; in the AN population it was 7.3 and in the population with AN associated with type 1 DM it was 34.4.
Obesity

Obesity as a risk factor is linked to AN and BN. It is also a mid-long term habitual state of BED and, in this case, obesity directly influences diagnosis and treatment. Up to 6% of obese children present BED124-126.

Malabsorption syndromes

Especially in AN, malabsorption syndromes, gluten-intolerance or lactose-intolerance are risk factors for eating disorders, course and prognosis modifiers and pathologies that must be taken into account when planning a treatment diet aimed at ponderal recovery. There are no prevalence studies and the literature only yields descriptions and studies of alienated cases127, 128.

Thyroid diseases

Thyroid diseases, both hyper and hypothyroidism, are relevant in the onset, course, prognosis and treatment of eating disorders. There are no prevalence studies, only case description studies129-132.