Eating disorders are common psychiatric conditions in young women and have a substantial impact on physical health and psychosocial well-being. Early identification is essential to improve outcomes.

Eating disorders clinical features and diagnosis

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Eating disorders are widely thought of as modern problems arising as a consequence of a Western culture that values, especially for women, being thin. Yet history suggests that these disorders are not a new phenomenon — there have been reports of young women restricting their food intake as early as the Middle Ages (the so-called Fasting Saints). Anorexia nervosa (described below) has also been identified in diverse contemporary cultures, including African and East Asian. It is worth noting that not only young women suffer from eating disorders — an estimated one in 2,000 men in the UK suffer from anorexia nervosa at some time in their lives.

Although core clinical features of eating disorders, such as low weight, endure across history and geography, the underlying beliefs often vary. For example, in non-Western cultures beliefs relating to the pursuit of thinness are uncommon. The notion that these disorders can be explained by cultural influences alone is further challenged by the substantial genetic contribution to risk. Indeed, the causes are complex and involve interactions between genetic, neurobiological and psychological factors.

Anorexia nervosa (AN) was described first in the late 19th century. Derived from Greek, it literally means "without appetite" with an underlying "nervous" or psychiatric cause. Bulimia nervosa (BN) was used later to describe individuals with a pattern of binge eating followed by purging behaviours. It originates from the Greek word boulimia (bous meaning "ox" and limos meaning "hunger") and implies a ravenous hunger.

Classification

Eating disorders comprise a spectrum of syndromes and are characterised by several key features, including a clear disturbance of eating habits or other behaviours that affect an individual’s weight. These disturbances are often associated with an over-evaluation of, and preoccupation with, body shape or weight and result in clinically significant impairment of psychosocial functioning or physical health. Importantly, to be classified as an eating disorder, these changes cannot be attributable to any general medical disorder or other psychiatric condition.

The main diagnostic criteria for AN and BN, according to the International Classification of Diseases 10th Revision (ICD-10), are shown in Box 1. A third diagnostic category, atypical eating disorders, is given for individuals who do not meet the full ICD-10 diagnostic criteria of either AN or BN (eg, a key symptom such as amenorrhoea may be absent in presence of a body-mass index [BMI] less than or equal to 17.5). In reality, patients often move between these diagnoses suggesting some overlap of the underlying causes.

The American Diagnostic and Statistical Manual of Mental Disorders 4th Edition (DSM-IV) has a similar
classification system which also allows for specifying prominent types of abnormal behaviour. For example, AN can be classified as either restricting type or binge-eating and purging type. DSM-IV recognises an additional diagnosis: binge eating disorder (BED), which is characterised by periods of uncontrolled eating. It is differentiated from BN by the lack of compensatory behaviours (eg, dieting). Hence, individuals with BED are often obese.

Epidemiology

In Western society, AN has a prevalence of about 0.3% and the estimated number of new cases per year is eight per 100,000 people. Over 90% of those affected are female and prevalence among women in their late teens is estimated at around 1%. The peak age of onset for AN is 16 years.10

BN is more common — the prevalence in women is estimated to be 1% (and over 5% of those with partial syndromes [ie, significant BN symptoms but not meeting the full diagnostic criteria] are included). Similar to AN, it predominantly affects females (90%) but has a slightly later mean age of onset of 18 years.

Atypical eating disorders are the most common, representing as much as 50% of those visiting an eating disorder clinic;1 although the prevalence within the community is unknown.

Causes

As with other psychiatric conditions, no single cause of eating disorders has been identified; they are thought to arise through interactions of multiple genetic and environmental risk factors, affecting both biological and psychological systems. These factors contribute to development and maintenance of an eating disorder, the latter being of greatest importance to treatment.1,14

Family studies have shown a genetic contribution to the development of both AN and BN (as high as 50–80% in some studies), but the exact degree of heredibility is unknown.

Much attention has been given to polymorphisms of genes relating to 5-hydroxytryptamine (5-HT) and associated neurotransmitter system abnormalities. This system is known to be strongly involved in the regulation of eating, mood and obsessional traits.15 Restricting food intake reduces the availability of tryptophan (and thus 5-HT) and may serve to reinforce further food restriction by reducing anxiety.14

Importantly, starvation also dampens emotional experience and it may be valued as a coping strategy following stressful life events or difficulties. Life events or difficulties can be identified before onset of an eating disorder in approximately 70% of cases.15

Criticism in early life by family members regarding eating habits, body shape or weight can contribute to development of BN.16 People who participate in activities that routinely promote thinness (eg, ballet dancing, modelling) would also appear to be at a greater risk of developing eating disorders.

Established risk factors for developing eating disorders include female gender, adolescence and young adulthood, being of white ethnicity and living in a Western society. Although there is no evidence that a Western cultural overvaluation of thinness plays a causative role in AN, it may be an important factor contributing to maintaining the disorder. A number of studies have shown that BN is more common in immigrant populations living in Western countries.17

Emerging trends relating to personality show that eating disorders are associated with low self-esteem and difficulties in expressing negative emotions and resolving conflict. Perfectionism has also been found to be a specific risk factor for eating disorders.16

Diagnosis

Eating disorders can be difficult to identify. Early symptoms are often non-specific (eg, lethargy or dizziness) and are only brought to the attention of healthcare professionals when prompted by worried family members or friends. Sufferers can be in denial about their condition or be unwilling to seek help. Often, aspects of the disorder are highly valued by the individual. Also, feelings of shame can lead to secrecy. Consequently, more than half of cases are believed to be undiagnosed. However, early identification in primary care should be a priority since early intervention leads to improved treatment outcomes.18

Box 1: Diagnostic criteria for eating disorders

The following are diagnostic criteria for eating disorders according to the International Classification of Diseases 10th Revision (ICD-10).17

Anorexia nervosa

Characterised by weight loss, induced or sustained. Definite diagnosis requires:

- Body weight maintained at least 15% below that expected or body mass index ≤17.5
- Weight loss is self-induced by avoidance of “fattening foods” and one or more of: self-induced vomiting; self-induced purging; excessive exercise; use of appetite suppressants and/or diuretics
- Body-image distortion whereby dread of becoming fat persists as an intrusive, overvalued idea, with self-imposed low weight threshold
- Widespread endocrine disorder involving hypothalamic-pituitary-gonadal axis (eg, amenorrhoea for women and loss of sexual interest for men)
- If onset before puberty, normal development is delayed or ceased (eg, for girls the breasts do not develop and for boys the genitals remain undeveloped)

Bulimia nervosa

Characterised by repeated bouts of overeating and excessive preoccupation with control of body weight, resulting in extreme measures to mitigate the “fattening” effects of ingested food. Definite diagnosis requires:

- Persistent preoccupation with eating and irresistible craving for food; individual succumbs to episodes of overeating in which large amounts of food are consumed in short periods
- Attempts to counteract the “fattening” effects of food by one or more of: self-induced vomiting; laxative abuse, alternating periods of starvation; use of drugs such as appetite suppressants, thyroid preparations or diuretics (patients with type 1 diabetes may choose to neglect their insulin treatment)
- Morbid dread of becoming fat and sharply defined weight threshold set well below the pre-morbid weight that constitutes the optimum or healthy weight
Diagnosis is based on a detailed assessment of past and current eating and weight-control behaviours, thoughts about weight and shape and associated clinical features (see Box 1, p326). This can be supported with collateral information from family members and friends, although some individuals do not wish for others to be involved in their care and decline consent to share information. An appropriate risk assessment should be undertaken because, in some situations, it may be necessary to breach confidentiality and involve others to ensure an individual’s safety.

Questions that can be used to identify people who require further investigation include:

- Do you think you have an eating problem?
- Do you worry excessively about your weight?

Box 2 describes the kinds of people for whom clinicians should adopt a lower threshold for suspecting an eating disorder.

**Signs and symptoms** Physical examination may be completely normal, especially in the early stages of an eating disorder. An accurate BMI should be calculated and compared with normal reference ranges (see Box 3, p329). BMI centile charts should be used for children and adolescents under 18 years of age (failure of expected growth or a value below the 2.4 centile of the reference population indicates underweight). Clinicians should always consider that patients may take measures to mask their true weight (eg, carrying weights in pockets or drinking extra fluids).

By definition, individuals with AN will have a BMI less than or equal to 17.5, whereas those with BN are usually of normal weight or overweight and only occasionally underweight.

Other signs and symptoms are usually secondary to either starvation or purging behaviours.

Signs of starvation can include bradycardia, orthostatic hypotension, hypothermia, dizziness, syncope, headaches, cold intolerance, delayed gastric emptying, constipation, loss of muscle tissue and subcutaneous fat, amenorrhoea, dry skin, lanugo (fine downy hair on upper body and face) and fractures secondary to osteoporosis.

Purging can cause symptoms such as: dizziness, syncope, palpitations, fits, pins and needles, cramps (all secondary to hypoglycaemia and/or electrolyte disturbances); bloating, fullness after eating, gastro-oesophageal reflux disease, blood in vomit (because of oesophageal tears or peptic ulcer disease), sore throat, dental erosion, knuckle calluses (Russell’s sign), salivary gland enlargement (many of which are secondary to self-induced vomiting); atomic colon and rectal prolapse (secondary to laxative misuse).

Psychosocial problems can include a preoccupation with body weight, body shape and food, anxiety, irritability, overactivity, social withdrawal, interpersonal and occupational difficulties, sleep disturbance and poor concentration.

**Investigations** BMI is only a proxy measure of medical risk and is easily falsified. Therefore, laboratory investigations are essential to determine the extent of complications; however, findings may be within normal reference ranges despite severe disease.

Results of thyroid function tests and the erythrocyte sedimentation rate are usually sufficient to exclude medical causes for eating and weight disturbance (such as hyperthyroidism or inflammatory bowel disease, respectively).

Abnormalities that can occur in a person with an eating disorder include: hypoglycaemia; hypokalaemia (because of vomiting and laxative/diuretic misuse); hypochloraemia (due to vomiting); hyponatraemia (from laxative misuse and water loading); anaemia; low white cell count; hypercholesterolaemia; raised liver function test results; and, with severe, end-stage starvation, low platelets and low albumin.

Urea and creatinine are usually low in AN due to reduced dietary intake. Therefore, blood levels in the upper normal reference range can indicate significant renal impairment. Low phosphate levels are associated with refeeding syndrome (see accompanying article, p330). Sick euthyroid syndrome and reduced concentrations of leutenising hormone, follicle stimulating hormone and oestradiol are among the body’s usual adaptations to starvation.

An electrocardiogram is indicated if a patient’s BMI is less than 15 or there is hypokalaemia. Findings may include bradycardia, prolongation of the QTc interval and nonspecific ST-T changes.

A dual-energy X-ray absorptiometry (DEXA) scan to determine bone density, and possibly identify osteoporosis, is indicated if amenorrhoea has been present for a year or more.

**Comorbidities** Psychiatric comorbidity is common in patients with eating disorders and can often make
diagnosis and treatment difficult. Depression is the most common with an estimated lifetime risk of up to 80% for patients with AN. However, diagnosis of depression at low weight is complicated because low mood, lack of energy, poor motivation, reduced enjoyment and poor concentration are also symptoms of starvation. Depressive symptoms before weight loss and their persistence after weight gain are suggestive of true comorbidity requiring treatment.

Problems with illicit substance misuse are also found, particularly in patients with BN (some studies showing up to 70% comorbidity). Anxiety disorders, including social phobia and obsessive-compulsive disorder, personality disorders and deliberate self harm are also common.19

Endocrine and gastrointestinal disorders are associated with an increased risk of developing an eating disorder. This type of comorbidity worsens prognosis for both the eating disorder and comorbid condition and may present significant challenges for treatment. For example, up to one third of females with type 1 diabetes mellitus omit insulin to control weight — this may be the only compensatory weight loss strategy. By prioritising a drive for thinness over health, these individuals are extremely vulnerable to early, severe complications of diabetes. In such cases, management needs to integrate treatment for both the diabetes and the eating disorder.19

Complications and prognosis

Eating disorders can have a substantial impact on individuals’ physical, psychological and social health and development.

Generally, individuals with AN will have more serious medical complications than those suffering from BN (or atypical eating disorders) because of persistent low body weight. Physical morbidity is high and can include cardiovascular and renal complications. AN can also adversely affect growth and development, especially when occurring in childhood or early adolescence, and may result in osteoporosis and osteopenia.

Long-term follow-up studies of those with AN show that around 27–58% of patients have a good outcome, 13–25% have an intermediate outcome, 11–42% have a poor outcome and 1–11% of patients die.16

Approximately two fifths continue to have poor psychosocial functioning at 18-year follow-up and a quarter are unable to engage in employment.20 Mortality rates have been found to be upwards of 15% in individuals with AN (six times greater than peers). Death usually occurs as a result of suicide or ventricular arrhythmias.

A longer duration of illness at presentation is an indicator of poor prognosis — the greatest chance of recovery is in the first six years. Hence, early identification in primary care should be a priority. Other poor prognostic indicators include childhood obesity, low self-esteem, poor social and family relationships, mood and personality disturbances and substance misuse.19

The prognosis of BN is less studied but, if untreated, it has a chronic and fluctuating course. At five to 10 years follow-up after treatment, 50% of patients are fully recovered and 50% still have some form of eating disorder. For those who achieve remission, the relapse rate is around 30%, with risk declining four years after presentation. For BN, poor prognosis is associated with comorbid mood disorder or substance misuse, impulsivity, premorbid or family history of obesity, a history of AN and poor social adjustment. BN is not associated with an overall increased risk of death.21

References


20. References