Behavioural eating disorders

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Abstract

The eating disorders, anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED), manifest through distorted or chaotic eating and in the case of AN and BN are characterised by a morbid preoccupation with weight and shape. Whilst recent changes in diagnostic criteria have changed the landscape to some extent, eating disorders and partial syndromes, including avoidant/restrictive food intake disorder (ARFID), remain relatively common and early recognition and intervention is helpful. Aetiology is multifactorial, with high heritability. Prognosis overall is good but treatment can be long and intensive, significantly impacting families. An integrated multidisciplinary approach is essential, working collaboratively with families and young people. Psychological interventions focus on the eating disorder, supported by medical monitoring and dietetic guidance. Although working with families is the backbone of treatment for AN, young people also need opportunities for confidential discussion. For BN, family or individual approaches may be equally effective. Evidence for effectiveness of psychopharmacological agents is limited in both AN and BN. Psychological and pharmacological approaches may both be of benefit for BED. Cases of ARFID require individualised approaches, often involving anxiety reduction. Paediatric expertise is of particular value in the assessment and management of acute malnutrition and complications secondary to disordered eating behaviours, in the early stages of re-feeding, and in the monitoring and management of long-term complications such as growth retardation, pubertal delay and osteopenia. This article offers an overview of eating disorders in children offering advice for clinicians who will undoubtedly encounter them in clinical practice.

Keywords adolescent; anorexia nervosa; bulimia nervosa; child; eating disorders

Introduction

The eating disorders, anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED), manifest through distorted or chaotic eating and in the case of AN and BN are characterised by a morbid preoccupation with weight and shape (Tables 1–3).

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Dasha Nicholls MBBS MRCPsych MD is a Consultant in Child and Adolescent Psychiatry and Honorary Senior Lecturer, Feeding and Eating Disorders Service, Department of Child and Adolescent Mental Health, Great Ormond Street Hospital for Children NHS Trust, London, UK. Conflicts of interest: none declared. These behaviours and the associated cognitions differentiate eating disorders from other psychological problems associated with abnormal eating, including feeding disorders.

Debate about feeding and eating disorders classification has been prominent in recent years due to the process of revising the two major classification systems for mental disorders, the Diagnostic and Statistical Manual for Mental (DSM) Disorder and the International Classification of Diseases (ICD). This remains only partly resolved as DSM-5 was published in 2013, whilst ICD-11 is not expected to be published until 2018. The main challenge for the DSM-5 revisions was addressing evidence that the majority of those presenting with clinically significant eating disorders did not fulfil diagnostic criteria for AN or BN, and were therefore classified as have an eating disorder not otherwise specified (EDNOS). Changes to the diagnostic criteria addressed this by broadening the definition of AN and BN. DSM-5 also identified BED, previously incorporated in EDNOS, as a separate diagnosis.

Additional changes in DSM-5 reframed feeding problems as food intake disorders, and removed age related criteria (previously feeding disorders required onset before age 6 years). These presentations are now classified as the new diagnosis of Avoidant/Restrictive Food Intake Disorder (ARFID) (Table 4). In addition to recognition and diagnosis, paediatric expertise is vital in management of malnutrition and other acute medical complications, and of long-term complications such as the impact on growth, development and bone density.

Epidemiology

Within the Western world, eating disorders are seen regardless of class, culture and ethnic group. Increasingly eating disorders are recognised as a significant problem in non-western cultures too. It appears to be increasing in frequency. Even prior to DSM-5 revisions the number of young people in the UK directly affected by eating disorders increased significantly between 2000 and 2009. The incidence rates (per 100,000) for all eating disorders were: aged 10–14, 64.5 (female) and 17.5 (male); aged 15–19, 164.5 (female) and 17.4 (male).

Eating disorders are common

The prevalence of AN is around 0.3-0.5%, with a peak age of onset between 15 and 18, cases steadily increasing from age 10 and occurring in children as young as 7. High-risk populations (athletes, models, ballet dancers) have higher prevalence rates. BN tends to occur later. The prevalence is just under 1%, with a slightly later mean age of onset with cases reported from about 12 years. It is rare before puberty and is much less likely to come to clinical attention. Prevalence rates for BED range from around 2 -3% although unlike AN and BN, peak incidence is after adolescence. BED is probably under-recognised, and in young people may look more like loss of control over eating than true bingeing.

Eating disorders are significantly more common in girls and young women than in boys

Female gender is the strongest risk factor for eating disorders, but this can lead to under-recognition in boys. In AN there is marked increase in female-to-male ratio following puberty, leading to an overall ratio of around 11:1. For BN the ratio is around 30:1, whilst BED is thought to be much closer to equal. Presentation is

Diagnostic features of anorexia nervosa (adapted from DSM-5 and ICD-10 criteria)

- . AN is characterised by distorted body image and excessive dieting that leads to severe weight loss with a pathological fear of becoming fat
- · Weight lost or maintained at less than 85% of expected weight for height and age, or failure to make weight gain during a growth period
- · Fear of gaining weight or becoming fat, even though underweight
- Disturbance in the way one's body weight and shape is experienced (body image distortion), undue influence of body weight or shape on selfevaluation, or denial of the seriousness of low body weight
- Weight loss is achieved by restriction of food intake and specific avoidance of 'fattening foods' and/or: self-induced vomiting, self-induced
 purging, excessive exercise, use of appetite suppressants/diuretics
- If bingeing or purging behaviours are absent, this is known as restrictive anorexia nervosa; if present, as binge purge anorexia
- NB: Amenorrhoea has been removed by DSM-5 as a diagnostic criterion

Table 1

Diagnostic features of bulimia nervosa (adapted from DSM-V and ICD-10 criteria)

- Persistent preoccupation with eating and recurrent episodes (over a period of months) of binge eating, which are characterised by: eating a large
 amount of food in a short period of time AND a sense of lack of control while eating
- Attempts to counteract the 'fattening' effects of food by use of compensatory behaviours such as: self-induced vomiting, purgative abuse, alternating periods of starvation or excessive exercise, use of drugs such as appetite suppressants, diuretics, thyroid preparations or, in diabetics, misuse of insulin
- Psychopathology consisting of a morbid dread of fatness and setting of a target weight way below what might be considered healthy
- Bulimia nervosa may follow on from a period of anorexia nervosa, but would only be diagnosed if the patient is no longer significantly underweight
- · Bingeing and associated compensatory behaviours occurring on average weekly for at least 3 months

Table 2

Criteria for Binge eating disorder (BED) - summarised from DSM-5

- Recurrent episodes of binge eating*
- Binges associated with at least three of:
 - o Eating faster than usual
 - o Eating alone due to embarrassment by volume of food
 - Still eating large amounts despite not being hungry
 - $\circ \quad \hbox{Eating until uncomfortably full} \\$
 - o After eating feeling depressed, guilty or ashamed
- Distress about the bingeing
- Bingeing occurs at least once a week (on average) for 3 months
- Unlike bulimia nervosa there are no recurrent and inappropriate compensatory behaviours, and the bingeing does not occurring only during episodes of AN or BN
- * binge eating
 - Eating an amount of food larger than most people would eat in that time and in those circumstances
 - o A sense or feeling of a lack of control over the eating

Table 3

similar in both sexes, except for a male tendency to be concerned over shape rather than weight.

Pathology and pathogenesis

Biological, psychological and sociocultural factors all have a role in aetiology, which include predisposing factors (risks),

precipitating factors (triggers) and perpetuating (maintaining) factors. Such a formulation is useful as a working hypothesis to guide treatment interventions. Having some understanding of how the eating disorder has come about in the young person's life will likely be helpful to them and their parents once it comes to relapse prevention stage. However, symptom management and targeting maintaining factors are the initial priority. For example, if weight-related teasing is identified as a trigger, addressing bullying will not in itself address the eating disorder. By contrast, if perfectionism or athleticism are predisposing and maintaining risk factors, these may need to be addressed during the recovery process.

There is considerable evidence for genetic contributions to individual symptoms, attitudes and behaviours, such as self-induced vomiting, or perfectionism traits, which increase risk within individuals. Puberty may also activate some aspects of genetic heritability. Family studies, twin studies and adoption studies, have provided heritability estimates of 60–75% for AN and 30–80% for BN.

Understanding the aetiology of eating disorders has been subject to definite 'fashions'. Recent interest in the neurobiological aspects is thanks largely to advances in neuroimaging, and molecular genetics. This is not to devalue sociocultural theories, which may be more relevant in understanding changing epidemiology as well as individual risk within families. Culbert et al. (2015) confirmed a number of sociocultural influences as important: media exposure and pressures for thinness and nonspecific personality factors including negative emotionality/ neuroticism and perfectionism. Current data support the theory

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