Assessment and treatment of eating disorders in children and adolescents

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ABSTRACT

Feeding and eating disorders (FEDs) are serious mental health disorders that cause impairments in physical health, development, cognition and psychosocial function and can go undetected for months or years. They are characterised by disturbed eating behaviour associated with concerns about weight and shape or by disinterest in food, phobic avoidance or avoidance due to sensory aspects of food. Restrictive forms of FEDs lead to significant weight loss requiring intervention. Without specific knowledge of these conditions, they can evade detection, delaying time to diagnosis and treatment and potentially influencing outcome. This review article focuses on the key factors involved in the psychiatric assessment and treatment of four feeding or eating disorders (EDs): anorexia nervosa, avoidant-restrictive food intake disorder, bulimia nervosa and binge eating disorder. They have been chosen for discussion as they are most likely to be encountered in both a psychiatric and paediatric setting. It emphasises the importance of a family-focused, developmentally appropriate and multidisciplinary approach to care. It does not address aspects of medical assessment and treatment. Other feeding or EDs not included in this article are pica, rumination disorder, other specified feeding and eating disorder and unspecified feeding and eating disorder.

INTRODUCTION

Feeding and eating disorders (FEDs) are associated with impairments in physical health and social, emotional and cognitive development, which in adolescence can impact identity formation and self-esteem. If untreated, the outcome is extremely poor. Early intervention appears more promising than interventions for established and chronic illness.[1] Of key importance, FEDs require both paediatric and mental health expertise across the spectrum of presentations.

DIAGNOSIS AND CLASSIFICATION

DSM-5's reclassification of FEDs in 2013 means that more young people meet diagnostic criteria for a specific feeding disorder (FD) or eating disorder (ED) than previously. The criteria needed to make a diagnosis of anorexia nervosa (AN) or bulimia nervosa (BN) have been broadened and new diagnoses such as binge eating disorders (BED), night eating syndrome, purging disorder and avoidant-restrictive food intake disorder (ARFID) included. The International Classification of Diseases (ICD) 10 criteria are currently under revision. However, currently FDs in ICD 10 are classified separately from EDs, a separation reflected in clinical practice in the UK. The scope of many ED services, clinical guidelines, training and clinical commissioning networks explicitly excludes FD/ARFID, a distinction that has unhelpful implications for research as well as for service planning. Obesity is not classified as an FED.

Diagnostic criteria for the specific disorders are given below. Diagnosis of FD or ED is based on clinical assessment using a combination of self-report (where appropriate), parent report and clinical observation.

EPIDEMIOLOGY

The incidence of ED has remained relatively stable over the last few decades with a possible increase in the number of 15–19 year olds diagnosed with AN. BED, EDs among boys, and subthreshold or atypical cases also appear to be on the increase,[2] but this rise may reflect increased awareness among health professionals. Further research is needed to determine whether the same increase is occurring in childhood onset AN. The increase in obesity in the same time period is likely to be a factor in these changes since obesity is a risk factor for ED, and both BN and BED are more likely to occur in overweight populations.

Overall, estimated lifetime prevalence in the community of clinically significant ED in adolescents is around 13%, of which only 0.8–1.7% meet criteria for AN, 0.8–2.6% for BN, BED 2.3–3% and the remainder have subthreshold or atypical ED.[3 4] This profile looks very different from presentations at clinical services, where AN and atypical AN are most common.

As ARFID is a relatively new diagnostic entity, there are limited prevalence studies. In a tertiary setting, prevalence rates have been reported as ranging from 5% to 23%.

EDs are about 10 times more common in girls than in boys, although in childhood onset anorexia and ARFID the proportion of male sufferers is relatively higher. Typically, ARFID presents in middle childhood, AN emerges in early to mid-adolescence, and BN and BED in late adolescence or young adulthood.

GENERAL APPROACH TO ASSESSMENT AND TREATMENT OF FEDS

A multidisciplinary (MDT) team approach is advocated, core professionals including a psychiatrist, family therapist, psychologist, paediatrician, nurse, dietician and other therapists. Services without a large MDT need to develop links with key professionals such as speech and language therapists and social workers. Paediatricians are vital for early recognition and entry into services, as well as for differential diagnosis. The integrated 'whole team' approach found in specialist services, associated

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with good leadership and a coherent treatment philosophy, appears to be more important for outcome than the specific skills of any one individual working in a different setting.[5]

The initial interview is an opportunity to establish rapport and engage the often reluctant young person and their family. Exploring motivation helps the clinician determine factors that may promote recovery. Assessment covers physical, psychological and social needs and should be developmentally informed. It will include both an individual and a family component because the young person may not disclose weight or shape concerns or risk issues in a family context. Medical and psychiatric risk must be assessed in an ongoing way as risks fluctuate over the course of treatment. Direct mealtimes observation is important for assessment of some presentations.

At the end of the assessment, a shared formulation of factors influencing the development or maintenance of the FD or ED will help tailor treatment for the individual and their family. Feedback should include factors in the various domains of assessment (physical, psychological and social), together with a diagnosis, comorbidities and risks. The young person is offered choice where possible and should be involved in decision-making where appropriate. Growth monitoring should be instituted for those who have not completed puberty.

ASSESSMENT TOOLS

These can be used to measure FD or ED pathology, assist with diagnosis and monitor progress but should not be used without a clinical interview that includes the child and family and uses the expertise of the clinician. Tool selection depends on the clinical setting, time available and access to training.

Semistructured interview

The Eating Disorders Examination (EDE), considered the 'gold standard', assesses ED symptoms over the preceding 28 days. It is validated for diagnosis in adults and adolescents down to age 9, although the children's (ChEDE) version is generally used for children and adolescents aged ≥7 years.[6] In children and young people, information from parents and carers is needed to corroborate the diagnosis. Adolescents and children tend to score lower than adults, and young people with AN and boys tend to have lower scores than those with BN. It is therefore important to compare scores to the age-appropriate normative data. Training is required.

The Development and Wellbeing Assessment (DAWBA) generates likely DSM diagnoses on the basis of both parent and self-report, corroborated by clinician review and can be completed online (http://www.DAWBA.info). The DAWBA is more likely that the EDE to agree with clinical diagnosis in young people and also generates information about possible comorbidities.

A comparable interview to the EDE is in development for the diagnostic assessment of ARFID, Rumination Disorder and pica (Bryant-Waugh, personal communication).

Self-report questionnaires

The self-report EDE-Q is adapted from the EDE, is quicker to complete and does not require training to administer. Differences between the EDE and EDE-Q can be mitigated by giving instructions. Some clinical features, like binge eating, are reported more accurately by questionnaire than interview. The reverse is true for restrictive eating behaviours, which tend to be minimised on self-report. A short (15 item) form of the EDE-Q has been developed. The EDE-Q has not been validated for children under the age of 12.

Other shorter self-report questionnaires appropriate for AN, BN and BED include the Children's Eating Attitudes Test, the Eating Attitudes Test (age 13+), The Kids Eating Disorders Survey and The Eating Disorders Inventory for Children. The Dutch Eating Behaviour Questionnaire is useful for assessment of emotional overeating and binge eating in overweight populations.

A number of structured measures for FDs exist that allow information from parent report and clinician observation to be systemically captured, the most reliable of which is the Behavioral Pediatrics Feeding Assessment Scale.[7]

INITIATING MEAL PLANS

Whatever the ED or FD, the primary aim is for regular meals and snacks to be eaten and spread out throughout the day. Without regular eating, the young person can slip into the habit of skipping meals, lose hunger signals or a bingeing episode might be triggered. Anxiety can escalate due to worry about when the next meal will be. Emotional support or distraction during and after mealtimes is invaluable.

The skills of parents, carers and staff in supporting feeding are fundamental. In outpatient settings, families are given help for meal support, within the therapy sessions or through psychoeducation or videos. The emphasis is on reinforcing parental knowledge of normal eating expectations. Where there are specific nutritional concerns, a meal plan can be developed in consultation with a dietician. It will take time for the young person to accept a range and quantity of food that is desirable but the clinician must keep encouraging change. Individual psychological interventions in which the emphasis is on the patient taking responsibility, such as cognitive behavioural therapy (CBT), include direct work around regular eating patterns and intake. Nutritional therapy alone is less effective and has high rates of dropout.

Meal and snack choices should be appropriate in type and quantity for normal healthy eating (20–35% energy intake from fat, 45–65% from carbohydrate and 10–35% from protein), and the importance of fluids emphasised. Higher energy choices allow for weight gain without volume. An underweight person will have lower-than-expected energy needs due to their hypometabolic state. Refeeding of a severely malnourished child needs to follow published guidelines and be undertaken by a team knowledgeable of the risks of both underfeeding and overfeeding. The Junior MARSIPAN report gives detailed guidance on refeeding,[8] and a recent randomised controlled trial (RCT) suggests a reasonably high initial calorie intake (≥1200 kcal) except in those at ultra high risk.[9] As metabolic rate increases, so will energy requirements.

ANOREXIA NERVOSA

Aetiology

The aetiology of AN is complex and multifactorial. Where parent blaming and feminist theories once dominated, current understanding emphasises the role of heritability, neuropsychological risk including cognitive inflexibility and weak central coherence, and the role of perfectionism and deficits in social cognition. Sociocultural factors, such as family and peer relationships, self-esteem and coping style, and the impact of media and other cultural influences, are potentially modifiable and more likely to account for secular trends in incidence. Conversely underlying neurobiological factors, including autism spectrum traits, appear to influence treatment response.[10] Interestingly, genetic factors appear to play a greater role in adolescent onset than childhood AN, suggesting that puberty triggers epigenetic phenomena, especially in girls.[11]

Diagnosis

Although diagnostic criteria are the same for children and adults (box 1), young people lack the capacity to express abstract concepts such as self-awareness or motivation. Diagnosis therefore takes into consideration behaviour as well as cognitions. Young people are typically brought to a health professional by concerned parents with a history of restricting energy dense, fatty or sugar-containing foods, accompanied by increasingly rigid eating patterns. Social withdrawal, increased restlessness and low mood develop as weight loss escalates. Specific energy-eliminating behaviours, such as excessive exercising, self-induced vomiting or, more rarely in young people than adults, laxative misuse, may be present.

It can take some time for parents and health professionals to recognise when these behaviours fall outside the normal range, and the young person may be very sick by the time they present. This is particularly the case with children who have a shorter duration of illness and more rapid weight loss,[12] with high levels of medical instability.[13] Males may also not be recognised as readily, as they may exercise excessively, and be focused on muscular build than low body weight.

Underweight is estimated from comparing body mass index (BMI) to age-matched norms. Below the second BMI centile, % BMI (BMI/% median BMI for age and gender) is the most useful way of quantifying underweight. However, medical instability can be present without low weight so the degree of weight loss and other parameters must also be considered when assessing severity. The Junior MARSIPAN guidelines[8] provide a risk assessment framework by which risk and medical stability can be assessed to guide management decisions.

Differential diagnosis

AN and obsessive compulsive disorder (OCD) share obsessions and compulsive behaviours. When these solely focus on eating and weight, the diagnosis is AN. Depression, anxiety and rumination disorder can cause significant weight loss but concerns about weight and shape and fear of weight gain will not be present. Those with rumination disorder will regurgitate food effortlessly after eating rather than it being a deliberate act.

Medical causes of weight loss are summarised in table 1, and it is imperative that these are ruled out, although managing acute malnutrition should not be delayed while investigations take place.

Comorbidities

Comorbid conditions are common. Fifty percent of young people will have an anxiety disorder such as social anxiety or OCD. Major depressive disorder is also common. A small but important minority will have comorbid autism spectrum disorder. Diagnosis of comorbidity must be made cautiously as effects of underweight can mimic these symptoms, which resolve with weight restoration.

Box 1 Diagnostic criteria for anorexia nervosa (based on ICD 10 and DSM-5)

- Intentional restriction or avoidance of food intake leading to significantly low body weight, or in young people, failure to make expected weight gain.
- Distorted view of weight and shape, fear of fatness or lack of insight into the seriousness of low body weight.
- Emergence of behaviours such as avoidance of foods perceived as fattening, excessive exercise, use of weight control medications or purging.
- Endocrine dysfunction that might cause loss of periods in females or sexual potency in males. In young people, puberty may be delayed or arrested.

Table 1 Medical differential diagnoses for anorexia nervosa

Endocrine	Hyperthyroidism, glucocorticoid insufficiency, diabetes
Gastrointestinal	Inflammatory bowel disease, coeliac disease, peptic ulcer disease
Neoplastic	Central nervous system tumours or other malignancies
Other	Chronic infections such as tuberculosis

Treatment

Outpatient-based care is as effective as inpatient and more cost effective[14] provided the child is medically stable and psychiatric risk can be managed. Medical unstable patients will require inpatient stabilisation in either a paediatric or psychiatric setting before discharge to outpatient care. Most specialist services offer a range of treatment options from outpatient, intensive treatments with meal support, and access to day programmes and inpatient treatment. Targets for weekly weight gain are generally 0.5–1 kg in the inpatient setting and 0.5 kg in outpatient setting.

Psychological treatments

The current evidence for adolescents <19 years who have had the illness for <3 years best supports family based therapy (FBT),[15] but there are relatively few studies comparing it with other treatment modalities. The Maudsley Model of FBT focuses on behavioural change, regards parents as experts of their family and empowers them to take charge until recovery begins. The therapy consists of three phases (table 2) conducted over a 6-month period, although this may need to be increased to 12 months for those with marked rigidity and obsessional anxiety. At the end of treatment, 50–75% will have returned to a healthy weight and relapse rates are generally low. Intensive multifamily therapy over 5 days has also shown good outcomes and may be useful where FBT is insufficient alone.

Adolescent focused therapy (AFT)[16] has been shown to be similarly effective compared with FBT but outcomes are better for FBT at 6 and 12 months.[17] It is therefore a valuable alternative when family dynamics are such that they interfere with treatment or if the family is unable to commit to FBT, perhaps due to ill health. In this therapy, the adolescent is taught to identify and cope with negative emotions and developmental challenges hypothesised to be driving the ED. In trials, rates of hospitalisation for medical instability were higher with AFT than FBT since AFT does not focus on managing eating behaviour specifically.

Enhanced cognitive behavioural therapy (CBT-E) has shown good outcomes in adult settings for both AN and BN, and

Table 2 Phases of family-based treatment

Phase of family based therapy	Treatment (weeks)
Parents in charge of weight restoration	1–10
Adolescent taking increasing responsibility for eating	11–16
Adolescent issues	17–20

substantial improvements in weight and eating disorder pathology maintained over a 60-week period have been seen in an adolescent cohort.[18] This therapy focuses on the cognitive processes maintaining the ED to drive changes in eating behaviour.

Parent groups are effective for addressing the emotional experiences of parents, relative lack of knowledge, can improve skills, confidence, understanding and adherence to the meal plan and may have an important role to play in early phases of illness[19] as well as being an important adjunct to care for those with established illness.

Group work for young people is often a feature of multimodal and intensive treatments, and tends to focus on self-esteem, body image or thinking style.

Medication

Antipsychotics are often used in the treatment of AN with aim of reducing anxiety, obsessional thinking and improving weight gain. Although case reports have shown promise, three small RCTs conducted in adolescents showed that risperidone and olanzapine provided no additional benefit during weight restoration.[20 21] In an open-label RCT, quetiapine led to psychological and physical improvements but the study was not adequately powered to draw firm conclusions.[22] There may be a subgroup of patients where antipsychotic medication is a short-term adjunct to help manage dysregulated behaviour and emotions. However, there is no research evidence to support this practice.

Similarly, there is no evidence of benefit for the use of selective serotonin reuptake inhibitors (SSRIs) for the treatment of the symptoms of AN or for preventing relapse. When considering an SSRI for a comorbid illness, it is important to consider whether starvation is driving the symptoms themselves. A family or personal history of mental illness or symptoms emerging prior to ED onset can be a helpful guide. SSRIs are less effective in those that are acutely ill and underweight. Caution must be taken prescribing any medication that could prolong the QTc interval, which may be lengthened due to underweight.

Despite the lack of evidence for effectiveness, psychotropic medications are widely prescribed.

Treatment-resistant adolescent AN

Following review and reformulation if needed, a number of strategies are used when first-line interventions are not effective. These include

- Enhancing parents' self-efficacy through intensive parent coaching alongside FBT[23] or through groups' teaching communication skills, motivational interviewing and meal support to parents and carers.[24]
- Providing therapy to parents alone using an FBT format (separated FBT) if levels of criticism and conflict are high in conjoint therapy.[25]
- Cognitive remediation therapy (CRT) is a specific approach developed on the basis of neuropsychological research into aetiology and maintenance of AN.[26] Sufferers of anorexia tend to be rigid in their thinking style and pay extreme attention to detail. Early studies using CRT show improvements in these domains, and when delivered in the group format, recipients reported feeling better able to change. It may therefore have a role in improving motivation or engagement in therapy.
- The role of inpatient psychiatric treatment for adolescent AN is undergoing reconsideration in light of evidence that it does not improve, and may even worsen, outcome,[14] and is no more effective than day patient treatment.[27] There is undoubtedly a role for longer admissions as well as brief crisis admissions in a small number of treatment-resistant cases. The decision to admit should be made carefully by those with in-depth knowledge of the individual patient and a long-term view of their treatment history and response.

Outcomes

Adolescents generally do better than adults with AN, with 50–75% receiving a family-based treatment approach reaching a healthy weight. At follow-up, 60–90% will have recovered or partially recovered and relapse rates are generally low. Emphasis on early, intensive, outpatient, family-based treatment approaches, with preservation of home and school life where possible, appears to have improved outcome overall. There are however a proportion of patients who relapse or who are treatment resistant, an increased risk of psychiatric disorders in adulthood with comorbidity estimated around 50% and a suggestion that childhood onset AN may have a poorer outcome than adolescent onset.

BULIMIA NERVOSA AND BINGE EATING DISORDER

Aetiology

As for AN, psychological and environmental factors interact with and influence the expression of genetic risk to cause eating pathology.[28] Bulimia nervosa (BN) has features in common with AN and with BED, such that some argue that a categorical distinction is unjustified. Nonetheless, some risk factors and features of BN differ from restrictive AN, including impulsivity and sensitivity to reward and punishment, while others overlap such as high heritability, and temperamental traits such as perfectionism.

Diagnosis

Binge eating (box 2) is a characteristic of both BN and BED, and can occur in AN too. Loss of control may be more indicative of the binge eating than the amount of food consumed, particularly in children and young people, who may not readily be able to access food but nonetheless feel the drive to binge. Binges are often preceded or followed by periods of food restriction. The presence or absence of compensatory behaviours (self-induced vomiting, medications (laxatives or diuretics), fasting or excessive exercise) to prevent weight gain determines the diagnosis. Despite compensatory mechanisms, individuals with BN are usually of normal weight or overweight. Purging with laxatives is less common and secretiveness more common in young people. About two-thirds of individuals with BED are overweight, but weight does not correlate with disease severity.

Box 2 Definition of binge eating

- Regular episodes of eating much more than most people would in a similar situation over a short period of time.
- An experience of 'loss of control' while eating.
- In a binge episode, the individual may
  - eat alone due to shame and disgust about quantities eaten
  - eat when not hungry
  - eat rapidly
  - eat until uncomfortably full
  - feel depressed or guilty afterwards.

Table 3 Common differential diagnoses for bulimia nervosa or binge eating disorder

Medical	Obesity, insulinoma, hypothalamic tumours, gastric outlet obstruction, hyperemesis gravidum, Prader-Willi syndrome, Kleine-Levin syndrome, Kluver-Bucy syndrome
Psychiatric	Anorexia nervosa, obsessive compulsive disorder, depression, body dysmorphic disorder

BN was first described in 1979 as 'an ominous variant of AN' because binge purge behaviours arise in a proportion of those with AN, and this subgroup has a poorer treatment response. This presentation may also be accompanied by feelings of shame at having failed to maintain a low weight despite a strong cognitive drive to do so. If not preceded by AN, both BN and BED can go undetected for several years before a diagnosis is made, which may reflect embarrassment and poor insight. Sufferers typically present 5 years after symptom onset unless their behaviours are extreme or have been disclosed to a parent/carer. Guilt and shame are common, cognitions that require cognitive maturation and a capacity for self-evaluation. This may explain why BN and BED tend to occur in later adolescence and adulthood and are relatively rare in childhood.

Depressive symptoms and body dissatisfaction are important risk factors, as well as childhood obesity. Males can also suffer from the disorder and are more likely to present with overexercise and steroid use.

Screening tools can be useful in identifying young people with BN and BED, and self-referral may encourage earlier help seeking. Dentists and paediatricians are also key in detecting BN, through dental erosion, abnormal electrolytes, oesophageal bleeding, and so on.

Comorbidities

Anxiety and depression are common. Self-harm, suicidal ideation, substance abuse and other impulsive or risk-taking behaviours are also seen but less so in younger adolescents. Mood lability and emotional dysregulation are common features.

Individuals with onset of bingeing prior to the age of 18 have a higher lifetime risk of BN, greater likelihood of ED treatment, substance abuse and higher rates of post-traumatic stress disorder.

Differential diagnosis

Disorders to consider in the differential diagnosis are given in table 3.

Treatment

The majority of patients with BN and BED can be treated as outpatients. Hospitalisation or day patient treatment should only be considered for management of medical risk, suicide risk or severe self-harm. The evidence base for the treatment of both disorders in adolescence is limited.

Bulimia nervosa

In BN, FBT, cognitive behavioural therapy adapted for adolescents (CBT-A) and CBT self-care have all shown efficacy (table 4). FBT approaches have marginally better short-term but equal longer-term outcomes. CBT therapy focuses on the relationship between current thoughts, feelings and behaviours (figure 1), and specifically on symptom reduction and the function of BN in the person's life.

Binge eating disorder

CBT-E is effective for adults with ED who are not underweight. It has shown promise for BED and BN in adolescence, but no long-term follow-up data are available.[29]

Interpersonal psychotherapy (IPT) and health education groups targeting girls at risk of obesity and ED showed decreases in BMI, loss of control eating, depression and anxiety.[30] In the IPT group, objective binge eating frequency was also reduced at 12-month follow-up. IPT is a psychotherapy that focuses on interpersonal issues, which are understood to be factors in the development and maintenance of psychological distress.

Table 4 Summary of treatments for bulimia nervosa in adolescents

Type of therapy	Brief definition	Evidence
Cognitive behavioural therapy adapted for adolescents (CBT-A)	CBT is a therapy focusing on the relationship between current thoughts, feelings and behaviours and when used in eating disorders, focuses on the function of the bulimia in the person's life. Used widely in adults, this therapy has been adapted for adolescents.	FBT may be more effective in promoting abstinence from binge eating and purging than CBT-A early on but there are no statistically significant differences between the two treatments by 12 months FU.[37]
CBT-guided self-care	A manual that focuses on the function of the bulimia in the persons' life and increases self-monitoring of thoughts, feelings and behaviours. The patient works through the manual with the therapist.	CBT-guided self-care showed a faster reduction in binging episodes and was more cost effective compared with family based therapy (FBT) but no differences were seen at 12 months.[38]
Enhanced cognitive behavioural therapy for eating disorders (CBT-E)	A specific form of CBT modified to be suitable for all eating disorders.	Good outcomes for adults. One study showed substantial improvements in eating disorder psychopathology at the end of treatment but no long term follow up data are available.[29]
Family-based therapy (FBT)	Focuses on behavioural change and empowers parents to take charge until recovery begins.	More effective than supportive therapy.[39] Similar effectiveness to CBT-A and CBT-guided self-care.[37 39] Choice between these may depend on availability of family and level of conflict, individual factors and preference and skill set of therapist.
Dialectical behavioural therapy (DBT) (Fischer):	For individuals presenting with suicidal and self-harming behaviour as well as eating difficulties. Teaches regulation of strong emotions by learning about what triggers them and developing coping strategies.	Can reduce self-harm and other impulsive behaviours as well as eating disordered behaviours.[40]
Integrated DBT and FBT (Murray)	A programme integrating FBT and DBT.	In a recent pilot study, improvements only seen in ED pathology and not emotional regulation.[41]

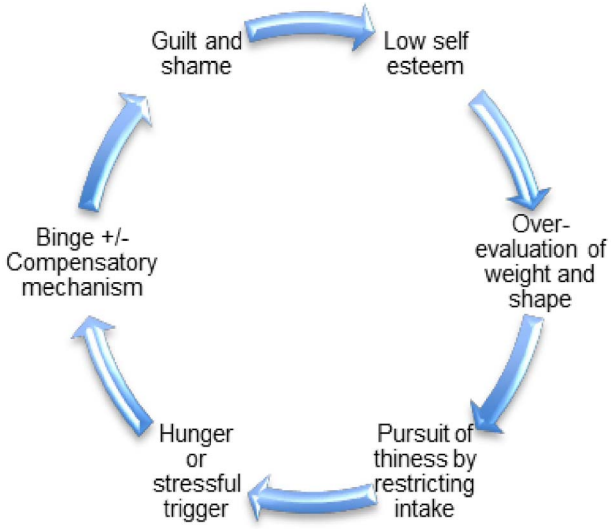

Figure 1 Cognitive behavioural model of bulimia nervosa and binge eating disorder.

Medication

Fluoxetine at a dose of 60 mg may be useful to reduce bingeing and purging episodes and overall illness severity.[31] Although the evidence is limited to one study in adolescent population, there is grade A evidence for its use in the adult population. CBT, however, is felt to be more effective with longer-lasting effects and is considered first line. Although some medications such as topirimate and sertraline appear to be useful for adults with BED, these have not been studied in adolescents.

Outcomes

BN is by nature episodic, acute or chronic. Around 40% recover after 6–12 months treatment with high relapse rate at follow-up. Remission rates at 5 years are estimated at around 50%. Rates of improvement and remission are better for BED, but the condition shows a similarly chronic course to BN. A small proportion of people with BN will develop AN but commonly revert back to BN, whereas individuals with BED tend not to develop another ED. Finally, both conditions are associated with obesity in later life,[32] with treatment rarely having an effect on overall weight. Data are not yet available to determine whether early intervention significantly improves prognosis, although this seems likely since duration of untreated illness is an important indicator of outcome.

AVOIDANT-RESTRICTIVE FOOD INTAKE DISORDER

Aetiology

ARFID describes a heterogenous group of presentations with likely multiple aetiologies. A large proportion have primary medical problems underlying the eating difficulty, most commonly neurological or gastroenterological disorders. Neurodevelopmental disorders, in particular autism spectrum disorder and attention deficit hyperactivity disorder, also underlie some presentations. Some subtypes of ARFID most closely resemble anxiety disorders (eg, choking phobia) and likely have similar aetiology. A proportion will have a 'failure to thrive' aetiology in the context of abuse, neglect or attachment disorder.

Diagnosis in children and adolescents

ARFID is a broad term that replaces FD and encompasses a variety of clinical presentations including a lack of interest in food, heightened sensitivity to textures of food and fear of the consequences of eating such as choking. A study looking at referrals to gastroenterology clinic found that 1.5% of 2231 referrals were likely to have ARFID with a further 2.4% having some features of the condition.[33]

In order to differentiate from developmentally normal variants in eating behaviour and style, a diagnosis is only made when the young person fails to meet their nutritional needs leading to significant weight loss (or a lack of weight gain or growth in children) or when nutritional deficiencies/dependence on nutritional supplements (orally or enterally) is present or when the problem markedly interferes with psychosocial functioning. An important part of assessment is the observation of a meal in which the child is presented with familiar and unfamiliar food and a variety of different food textures. Much can be established from this including the effectiveness of their swallow, levels of anxiety or interest in food, preference for certain textures and the interactions between parent and child. This highlights the importance of a comprehensive MDT approach.

Even so, it can be difficult to distinguish AN from ARFID particularly in younger patients, and careful assessment is needed to ascertain whether the specific cognitions of AN are maintaining the eating problem. ARFID sufferers often show the same complications secondary to being underweight and present at similar body weight as individuals with AN but the weight loss is not driven by weight or shape concerns. ARFID sufferers also tend to have a longer duration of illness prior to diagnosis and an earlier onset. Underlying medical conditions and dependence on nutritional supplements are more likely. Both conditions are associated with significant anxiety and distress around mealtimes. It is important to know that ARFID can develop into AN in a small number of cases or it may be that AN cognitions become more apparent. It is therefore important to keep reassessing the diagnosis, particularly if the young person is not progressing in treatment as expected.

If there is a concurrent medical or psychiatric illness, ARFID can only be diagnosed if the eating difficulty exceeds that normally associated with the condition. Medical causes of underweight to be considered in the differential diagnosis are summarised in table 1.

Comorbidities

Comorbidities include autism spectrum disorder, cognitive impairment and intellectual disability. More anxiety disorders but less depression are present compared with BN and AN.

Treatment

Very little research has yet been conducted into treatments for ARFID, although a number of interventions for FDs have been described in the literature. A pilot trial found support for an intensive, manual-based behavioural feeding intervention compared with waiting list for children with chronic food refusal and dependence on enteral feeding or oral nutritional formula supplementation.[34] In general, treatment is individualised on the basis of the main feeding or eating difficulty and the factors contributing to aetiology. For example, the child may be dependent on nasogastric feeding and have a lack of interest in eating. He/she may have experienced a delay in establishing normal feeding and pain associated with eating due to a severe food allergy prior to NG tube placement. During attempts to tube wean, the child's parents may have become anxious around mealtime, frustrated by their child's low appetite, leading to difficult mealtime interactions.

Once relevant medical issues have been addressed (such as reflux), interventions such as CBT focusing on anxiety management and exposure and response prevention may be used as well as family-based interventions.

Medications

When ARFID is comorbid with clear anxiety disorder, interventions for anxiety should be considered in the first instance unless the child or young person is very low weight, as above. Cyproheptadine, an antihistamine with appetite stimulation as a side effect, can be considered when psychological and nutritional interventions have not been effective.[35] Its safety and efficacy has been established in a paediatric population, and it has been used in children with dyspepsia, cystic fibrosis and cancer-related cachexia. Side effects include drowsiness and irritability, which can be minimised with cautious dose titration, and tolerance can be minimised by the use of intermittent dosage.

Of those who need paediatric hospitalisation, degree of weight loss predicts outcome, patients with ARFID having higher rates of remission at follow-up but requiring on average longer in hospital than patients with AN and requiring more in the way of nutritional support.[36]

SUMMARY

FEDs are relatively common, presenting to both paediatric and mental health services, and should be considered in the differential diagnosis of weight loss or change in eating behaviour. Effective psychological interventions are available while the role of psychotropic medication is limited. Further research is needed for this vulnerable subset of patients to evaluate treatments, understand trajectory and improve outcomes. Current objectives are increased recognition among paediatric and mental health professionals of the full range of FEDs, and the creation of specialist integrated MDT services in all localities aiming to reduce lengthy hospitalisations and improve outcome.

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