

Eating Disorders: Overview and Management in Women

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Introduction

Eating disorders (ED) are serious psychiatric conditions characterised by abnormal eating habits, with physical and psychological consequences. Anorexia nervosa's (AN) onset is usually in adolescence, while that of bulimia nervosa (BN) and binge eating disorder (BED) are in the late teens to early adulthood.¹ The severity can vary and symptoms can overlap and alternate between presentations over time. There is a clear female predominance, with only 10% of males being affected.² It is the third most common chronic medical condition in girls aged 15–18 years (after obesity and asthma)³ and the 10th leading cause of burden of disease and injury in young females.⁴ Of all the psychiatric illness, AN has the highest mortality rates,^{5,6} being three times higher than that of depression, schizophrenia, or alcohol-related illness. Half of the deaths in AN are due to suicide and violent methods are usually used.^{7,8}

A diagnosis of AN is emotionally and financially devastating for families with the average duration of the illness as long as 7 years,⁹ with a substantial risk of enduring disability, self-harm,¹⁰ and premature death.^{11,12} The mortality, morbidity, and service use and cost for inpatient care are among the highest of all psychiatric disorders.^{13–15} EDs, particularly AN, have a negative impact on fertility, and all are associated with negative consequences during pregnancy, birth, and infant outcomes.^{16,17}

Etiology is multifactorial, with studies showing a clear gene environment interaction. There is high heritability, 58–88% with several candidate genes, namely 5HT, DA, and opiate systems.^{18,19} Contributing factors include increase in significant life events and exposure to stress, increase anxiety and depression in pregnancy, and obstetric complications like prematurity and small for gestational age.^{20,21} The presence of obsessive compulsive traits and rigidity as well as excess attention to detail, high sensitivity to judgment of others, low self-esteem, and high striving contribute to development and maintenance of illness.

Eating disorders differ from other psychiatric illness not only in presentation but also in treatment, with as many psychological as physical symptoms and almost non-existent effective psychotropic medication to date. Moreover, less than half of the patients fully recover, one-third have a relapsing remitting course, and around one-fourth became chronic.²² Current treatments are based on the severity of illness as well as the associated risks, mostly to the physical health, although a significant number of the patients also suffer from psychiatric comorbidities like depression and anxiety. Evidence-based treatments are limited,^{23,24} nevertheless a lot of research is currently ongoing, which hopefully will improve care, outcomes, and prognosis in the future. Current treatments are only moderately effective, with better prognosis and outcomes when effective treatment is delivered in the first 3 years after onset of the illness.

Eating Disorders Classification

Eating disorders are divided in AN, BN, BED, and ED no otherwise specified (EDNOS). The later classification is given when a person does not fit full diagnostic criteria for the other types (see Table 1 for diagnostic criteria details).

Anorexia nervosa (AN)	Bulimia nervosa (BN)	Binge eating disorder (BED)
BMI <18 kg/m ²	Preoccupation and craving for Food	Recurring episodes of eating significant amount of food in a short period
Body image distortion	Episodes of bingeing	Marked by feelings of lack of control, guilt, embarrassment, or disgust
Food restriction with severe weight loss	Use of compensatory mechanisms to avoid weight gain	Binge eat alone to hide the behaviour
Pathological fear of becoming fat	At least once a week	At least once a week over 3 months

Epidemiology and Impact of Illness

EDs have a high cost to the person affected, their family and friends, and society. To the person with the burden of the illness with its symptoms, associated comorbidities, and complications. To the families through the distress of looking after a loved one with the illness, feeling frustrated and unable to effectively help and to the society given the costs of the illness, plus the costs of loss in productivity and shorter life.

In the USA, Hudson *et al.*²⁵ reported a lifetime prevalence estimate of AN, BN, and BED of 0.9%, 1.5%, and 3.5%, among women, and 0.3% 0.5%, and 2.0% among men, respectively. The incidence of AN increased over the past century, until the 1970s, while BN continued to rise. McBride *et al.*²⁶, employing data from the UK National Adult Psychiatric Morbidity Survey 2007, found a prevalence of 6.3% for AN and a recent study by Solmi *et al.*²⁷ of inner-city population in London reported 10% prevalence.

Studies over the years suggested that ethnic minorities might have a decreased risk of developing ED due to lower societal and cultural preferences for thin figures²⁸ with lower levels of bulimic symptoms among Black and Asian minorities²⁹ and higher levels of purging behaviours in Black minorities only,²⁹ and higher prevalence of binge eating in ethnic minorities' women.³⁰ A more recent study by Solmi *et al.*²⁷ showed similar results, showing Asian ethnicity to be associated with ED behaviours. A qualitative study, investigating ethnic differences in women's responses of mainstream beauty standards, found that Asian women were more likely than Black women to endorse them in a similar fashion to white women, as well as experiencing greater body dissatisfaction.³¹

A recent WHO survey¹ reported country-specific lifetime prevalence estimates consistently higher for BED (1.4%; 0.8–1.9%) than BN (0.8%; 0.4–1.0%). Median age of onset is in the late teens to early 20s for both disorders but slightly younger for BN. Persistence is slightly higher for BN (6.5 years) than BED (4.3 years). The lifetime prevalence of BED, in six European countries, was 1.9% for women and 0.3% for men.³² In the USA, higher lifetime prevalences were found among adults (women 3.5%; men 2.0%) and among 13–18-year olds (girls 2.3%; boys 0.8%).^{33,34} A longitudinal study of adolescents in USA reported an incidence rate for binge eating of 10.1 per 1000 person-years among females and 6.6 per 1000 person-years among males (both sexes ≥ 14 years).³⁵

Impact Cost of Illness

ED are estimated to affect 1.5 million European citizens, number very likely underestimated as it does not include patients with a diagnosis of EDNOS and BED.³⁶ The costs of the illness are around 0.8 billion Euro per annum (£0.6 billion per annum), not counting the loss of productivity, shorter length of life, and poorer health. Persons with ED have the highest proportion

of direct healthcare cost (72%).³⁷ The cost of treatment of ED in the UK is estimated to be around £80–100 million per year, including inpatient care, ambulatory (outpatient and primary) care, plus private healthcare. Perhaps 60% of these costs are for the age group of under 25 years.³⁸

Consequences of Eating Disorder on the Brain and Body

The brain requires a minimum of 500 Kcal a day for its basic functions. Starvation impairs brain and mind function, with multiple problems that can coexist and develop as a consequence or as comorbidities of the ED (see Table 2 for details).

Table 2. Brain and mind consequences of ED		
Brain function		
Cortical regulation	Executive function	Social cognition
↓ Neuroplasticity	↑ Rumination	↑ Sensitivity to criticism
↓ Less flexibility	Attention perseveration	Isolation
Impairment of new learning	↓ Emotional regulation	Fragmented and overdetailed thinking
	Excess control	Global connection impairment
	↑ Avoidance behaviours	
Mind function		
Starvation	Purging	
Low Mood, irritability or depression	Depression and anxiety with lifetime rates of 35–70% ^{39,40}	
Obsessions, rituals, compulsions, and obsessive compulsive disorder	Alcohol and substance misuse with rates of 20–30% in clinical samples ⁴¹	
Poor concentration and attention	Post-traumatic stress in 37% of cases ⁴²	
Excessive preoccupation, guilty, and fear of food	Cluster B personality disorder ^{43,44}	
Lack or reduced libido and disinterest in social and romantic relationships	Debt or have problems with the law for shoplifting and stealing food	
Cerebral oedema (rare)	Cerebral oedema	
Atrophy	Seizures	
	↑Risk of binge eating in the future (due to changes in brain processes)	

Family and careers are also affected. They frequently report high rates of anxiety, anger, frustration, and ending up treating the ill person with excess gentleness, neglecting their own needs, career, and life.

The signs and symptoms of starvation and purging on the body are described in Table 3.

Table 3. Signs and symptoms of starvation and purging on the body

	Starvation	Purging (namely vomiting)
Head	Parotid enlargement Eroded and teeth decay	Vitreous haemorrhage Eroded and tooth decay (acid) Cuts to mouth and tongue (fingernails) Parotid enlargement
Skin and hair	Hypothermia Alopecia Lanugo hair Dry skin and ↑ risk of developing sores Senile purpura Erythema ab igne Acrocyanosis	Cuts and abrasions in hands (from teeth) "Russell's sign" (callus on back of hand due to acid damage) Purpura
Heart and lungs	↓ Blood pressure and pulse ↓ Oxygen saturation Poor circulation Cardiac arrhythmias Heart failure Sudden death	Arrhythmias Sudden death
Gastrointestinal track	Gastric atrophy Gastric ulcers Gastroparesis Abdominal cramps Constipation Irritable bowel	Esophagitis and oesophagus ulcers Mallory-Weiss tears and haematemesis Boerhaave syndrome (ruptured oesophagus) Gastroparesis Abdominal cramps Gastric reflux with heartburn metabolic alkalosis Diarrhoea Constipation
Liver	Liver failure ↓ Albumin and protein production Raised cholesterol Impaired clotting Jaundice	Raised cholesterol

(Continued)

Kidneys	↑ Risk of pyelonephritis ↑ Risk of nephrolithiasis Renal failure	Renal failure
Bone marrow/ blood	Anaemia Leucopenia Thrombocytopenia	Anaemia
Electrolyte and water balance	Low sodium Low potassium Ankle oedema/generalised oedema	Low sodium Low potassium Ankle oedema/generalised oedema
Micronutrients	Low Iron Low folate Low vitamin B12 Low magnesium Low phosphate Low calcium Low vitamin D	Low Iron Low folate Low vitamin B12 Low magnesium Low phosphate Low calcium Low vitamin D
Pelvis	Pelvic floor weakness Double incontinence Uterine prolapse Frequency (often overnight)	
Reproductive system (see further details below)	Amenorrhoea Late or arrested puberty ↓ Libido Infertility	
Bones (see below)	Osteopenia/osteoporosis ↑ Risk of fractures	Osteopenia

Abuse of tablets, another purging behaviour frequent in ED, has their own particular set of consequences. These include the following:

- *Laxative abuse.* Diarrhoea, constipation, and dependence; fluid swings with big weight fluctuation and dehydration and not actual significant calorie loss.
- *Diuretics.* Nephrotoxicity, acute tubular necrosis, and renal failure.
- *Thyroid hormones.* Anxiety, mania, insomnia and palpitations, arrhythmias, and stroke.
- *Orlistat.* Diarrhea and incontinence of faeces.
- *Amphetamines.* Anxiety or even psychosis, irritability, euphoria, violent behaviour. Myocardial infarction, heart failure, arrhythmias, palpitations, stroke, seizures, coma, and dependence.

Bone Health

AN can cause osteopenia and osteoporosis. The latter may not be completely reversible, particularly if illness onset is before puberty and continues well into adult life. The greatest concern about osteoporosis and fractures are in girls who develop anorexia before they have their menarche, and in boys who are still growing in height and people with continuous illness for over 2–3 years. The degree of bone loss seems to be related to the severity and duration of the weight loss. Research shows that ED patients do not respond to usual treatments with hormones or other drugs, and the only effective means of improving bone health and reducing the risk of fracture seems to be achieving and maintaining a healthy weight.⁴⁵ Exercise and calcium supplements seem not to be useful, and the former can worsen the ED and therefore should be avoided.

ED and Reproductive Function

Lifetime ED can impact women's fertility, be associated with unexpected pregnancies, and impact negatively the antenatal and postnatal period. A study by Easter *et al.*⁴⁶ found that women with AN and women with AN plus BN more frequently saw a doctor for fertility problems, and women with AN plus BN were more likely to take longer than 6 months to conceive and having had fertility treatment to conceive. Unplanned pregnancies were more common in the AN population compared with the general population. All ED patients more frequently experienced negative feelings upon discovering their pregnancy. Most women with EDs have an improvement of symptoms during pregnancy due to worries about the consequences to the unborn baby, although changes in body shape frequently increase anxiety about weight gain. Studies showed that these women diet more, exercise more, and use laxatives in excess. On the other hand, women with subclinical or BEDs are at risk for an escalation of the disordered behaviours, putting both mother and fetus at risk for negative outcomes.^{17,47,48}

ED are associated with higher rates of miscarriage (two- to threefold increase in BN), intrauterine growth restriction, and small for gestational age babies in AN and BN, and one study suggested an increased risk of large-for-gestational-age in women with BED. Obstetric complications including premature labour, low birth weight, microcephaly, and problems with episiotomy repair.^{17,47,49,50}

There is high risk of relapse of the ED in the postnatal period, particularly in the first 6 months postpartum, as well as higher risk of developing postpartum depression and anxiety perinatally.^{17,47,51} A retrospective study of almost 500 women reported an 11.5% prevalence of some type of ED in the 3–7 months postpartum, with predominance in younger women.⁴⁷ In the Avon

Longitudinal Study of Parents and Children (ALSPAC), women with history of past depression and past/current ED showed high rates of anxiety and depression in all women across the perinatal period compared to controls, and this was more evident in women with ED symptoms in pregnancy.⁵¹ Women with EDs seem to stop breastfeeding earlier than other women; this was reported in a retrospective questionnaire study surveying 454 women at 3–7 months postpartum, which found that 11.5% were significantly less likely to be breastfeeding at 3 months postpartum.⁵²

Mortality

A meta-analysis of 35 published studies¹² found a standardised mortality ratio of 5.86 for AN, 1.93 for BN, and 1.92 for EDNOS. One in five individuals with AN who died had committed suicide.

The mortality rate for BED is related to obesity and its comorbidities and complications.⁵³ A more recent meta-analysis of 66 studies⁶ supports these findings of a consistently high prevalence of suicidality associated with ED, particularly AN. Moreover, contrary to previous findings, some recent studies have found that suicide risk appears also elevated in BN, although there is more variability in the literature. Self-injurious behaviours are frequent in EDs, higher among those with bingeing and purging behaviours, with suicide rates of 20–30% versus 7.4% in restrictive subtype. Risk factors associated with suicidality include chronicity of illness, comorbid diagnoses, history of abuse, and self-injurious behaviours. Furthermore, recent investigations on individuals who engage in rigorous physical exercise (considered a form of self-injurious behaviour among the ED population) are significant as they suggest that exercise may change the ability to endure pain and in turn affect individuals' acquired capability for suicide, using more lethal methods like hanging.

Investigations

Investigations are important in the assessment and treatment of ED as well as a full history from informants whenever possible. At assessment, they provide valuable information about severity and risks, and are essential in the differential diagnosis. During treatment, they aid in monitoring response and risk to physical health.

On assessment. Weight, height, body mass index (BMI), sitting and standing blood pressure and pulse; respiratory rate and temperature. Blood investigations including FBC, U&E, LFTs, bilirubin, TFTs, glucose, calcium, phosphate, magnesium, iron, ferritin, vitamin B12, folate, celiac screen and ESR. ECG, especially if severe electrolyte imbalance. DEXA (dual energy X-ray absorptiometry) scan, if lasting more than 2 years.

During treatment. Weight, height, BMI, sitting, and standing blood pressure and pulse; respiratory rate and temperature. Blood investigations weekly to fortnightly depending on risks and weight, to include at least FBC and U&E. If severe illness, include liver function.

After discharge (follow up by GP). Weight plus blood investigations depending on weight/symptoms; DEXA scan every 2 years according to need and age.

After assessment and based on the investigation results, it is useful to classify patients according to severity and risk, which in turn will inform treatment, including delivery setting and urgency of the intervention (Table 4).

Table 4. Classification of severity and risk

	Moderate risk	High risk
Examination	BMI < 14 kg/m ²	BMI < 12 kg/m ²
	Weight loss > 0.5 kg week	Weight loss > 1 kg week
	Systolic <90 mmHg Diastolic <70 mmHg	Systolic <80 Diastolic <60 mmHg
	Pulse rate >110 <50 min	Pulse rate >120 or <40 min
	Postural drop >10 mmHg	Postural drop >20 mmHg
	Squat test – difficulty getting up	Squat test – unable to get up without arms as levers
	Temperature <35°C	Temperature <34.5°C
	Skin breakdown >0.1 cm	Skin breakdown >0.2 cm
		QTc >450 ms, ST changes, arrhythmias Purpuric rash
Bone Marrow	WCC <4.0	WCC <2.0
	Neutrophil count <1.5	Neutrophil count <1.0
	Hemoglobin <11	Hemoglobin (HB) <9.0
		Acute Hb drop but MCV and MCH raised are not markers of acute risk
	Platelets <130	Platelets <110
Electrolytes	Potassium <3.5	Potassium <3.0
	Sodium <130	Sodium <130
	Magnesium 0.5-0.7	Magnesium <0.5
	Phosphate 0.5-0.8	Phosphate <0.5
	Urea >7	Urea >10

(Continued)

Table 4. (Continued)		
Liver	Bilirubin >20	Bilirubin >40
	Alkpase >110	Alkpase >200
	AST >40	AST >80
	ALT >45	ALT >90
	GGT >45	GGT >90
Nutrition	Albumin <35	Albumin <32
	Creatinine kinase >170	Creatinine kinase >250
	Glucose <3.5	Glucose <2.5

Treatments

There are several types of treatment interventions and many more being currently researched and developed, namely looking into brain-directed interventions.²² Treatments of ED should be ideally provided in specialist services, as available. The treatments for AN are distinct from that for BN and BED, with the intervention for the latter ones being similar, so we will describe them together in this brief description of management interventions.

The treatment of ED depends on the severity and associated risks to the physical and mental health of patients as described above.

Anorexia Nervosa Treatment

For the moderate and mild cases, outpatient treatment is recommended. Psychological interventions are currently the treatment of choice, both individual and group. There is no leading modality so far, but many modalities show good results, namely, enhanced cognitive behaviour therapy (CBT) for ED, cognitive analytical therapy (CAT), Maudsley Model of Treatment for Adults with AN (MANTRA), Specialist Supportive Clinical Management (SSCM), Cognitive Remediation Therapy (CRT), just to name a few.^{22,47,50,54,56–58,74,75} Family and carers' intervention, with communication skills workshops and motivational interviewing skills training, is also an essential part of the treatment,⁵⁹ along with occupational and vocational work and input from a dietician.

For the chronic, severe, and enduring cases, supportive care in outpatient clinics is recommended, with monitoring every 3–4 weeks and increase frequency at times of crisis and/or deterioration, aiming at preventing full relapse and need for inpatient admission.

For the severe and moderate-risk cases, EDs' day hospital treatment is recommended, with attendance to accident & emergency (A&E) when and if needed for punctual physical health problems. ED day hospital care provides individual and group psychological interventions, dietician input, meals, and occupational therapy interventions.

The treatment for very severe cases – with BMI below 13, with rapid weight loss (more than 1 kg week), and at high risk of re-feeding syndrome (see medical risk above - Table 4) – usually requires inpatient admission: either directly to a specialised EDs unit or, when urgent medical treatment is needed, with short admission to a medical unit or emergency medicine department/A&E department prior to transfer to EDs unit. This should be ideally on an informal basis or under compulsive admission, if needed. Nasogastric feeding should be the last resort.

New brain-directed treatments are in trial phases and showing promising results. We will not go into much detail about them here as it is out of the scope of this chapter, but just to name a few – cognitive attention bias training and positive mood and food exposure,^{60,61} repetitive transcranial magnetic stimulation (rTMS),⁶² deep brain stimulation (DBS),⁶³ and on the neuropharmacology side, for example, the use of d-cycloserine⁶⁴ and intranasal oxytocin.⁶⁵

Bulimia Nervosa and Binge Eating Disorder Treatment

For BN the treatment is usually provided on an outpatient basis, although when very severe purging with high risk to the physical health, brief inpatient treatment may be beneficial as well as correction of electrolyte imbalances in A&E or medical units.

Obese and very obese BED patients often need treatment for multiple medical comorbidities. They are frequently considered for bariatric surgery, after the ED has been effectively treated, due to the risk of being unsuccessful otherwise. Current UK guidelines advice that patients being considered for this procedure should be screened and, if suspicion of psychiatric illness or if known past psychiatric illness, should be assessed by psychiatric services.

The mainline treatment currently showing evidence for BN and BED is based on psychological interventions, individual or group. For the milder cases and with no associated comorbidities, online self-help CBT based programs such as overcoming bulimia online (OBO) have shown to be effective, with email and phone supervision from a clinical psychologist.⁶⁶ It is also useful to provide self-help books and materials, refer for dietician input, and occupational therapies.

New treatments are also being researched for these illnesses.

Re-feeding Syndrome and Electrolytes

Re-feeding syndrome is a risk in very-low-weight patients starting to eat again, placing them at high risk of quick deterioration and ultimately death. It is essential to regularly monitor electrolytes (namely phosphate, magnesium, and potassium) for early detection of high-risk abnormalities.

Potassium is often chronically low in purging, with no immediate sequelae. Acute changes are more dangerous and regular feeding with control of purging is usually sufficient to correct this. If replacement is needed, preference should be given to oral rehydrating with oral solution. Regular electrolyte review and examination of fluid and water status (measurement of urea and lying and standing blood pressure) are advised.

Refractory hypokalaemia can also be due to concurrent low magnesium or calcium, and these should be rectified. If vomiting persists, potassium may remain low and a proton pump inhibitor, to inhibit gastric acid secretion, may reduce metabolic alkalosis and help to conserve potassium.

Rebound hypophosphataemia can occur on initial re-feeding as it is sequestered by carbohydrate metabolism and it can be fatal. Initial re-feeding should include foods with high phosphorus content, for example, milk-based products (>2 pints/day). If necessary, about 4 days of oral phosphate supplementation may also be needed plus 10 days of thiamine. Particularly in AN, as frequently there is multivitamin and mineral deficiencies, we recommend additional prescription of multivitamin and mineral tablet.

Re-feeding oedema is common and harmless and resolves spontaneously in a few weeks but should be distinguished from oedema secondary to heart failure. Dehydration should be assessed, identified with oral replacement being preferable.

Screening and Management of Eating Disorder in the Perinatal Period

Women with ED often go undetected during pregnancy and are generally reluctant to disclose their symptoms. The National Institute for Health and Clinical Excellence (NICE) advises regular screening questions for midwives and obstetricians, during the perinatal period (e.g., Do you think you have an eating problem? Do you worry excessively about your weight?) in women with history of EDs, low BMI, lack of weight gain, expressing concerns about weight but not overweight, with gastrointestinal symptoms, hyperemesis gravidarum, and physical signs of starvation, or repeated vomiting and psychological problems.^{17,47}

The Eating Disorder Examination Questionnaire (EDE-Q) is a well-established self-report questionnaire which is easy to administer and can

identify people with an ED.⁵⁵ The questionnaire can be found in the Royal College of Psychiatrist website. For mild cases and people who refuse referral to an ED service, follow-up questions are suggested for each antenatal visit to assess the severity of illness, namely, "What is your current eating pattern? Are you restricting your dietary intake? Do you binge? Do you vomit or take laxatives after eating? How do you feel about your shape and weight? What is your weight? Are you gaining weight appropriately? What exercise are you doing? Are you exercising too much? What is your mood like? Do you feel low or anxious?"⁴⁸

Women with an active ED should be advised first of all to postpone pregnancy whenever possible, until they have received treatment and are largely recovered. They should be referred to a specialist service as soon as possible, especially if AN. They should be screened for the abuse of laxatives, appetite suppressants, or diuretics, which may not be safe in pregnancy.

Early education prior to conception or in early pregnancy about body changes, cravings, and hyperemesis gravidarum as well as expected weight gain and nutrition during pregnancy and impact on foetal growth is advised. Joint obstetric care is recommended, with midwives and obstetricians and ED and perinatal psychiatric services, when available. Monitoring for postnatal depression and relapse of ED is essential for early detection and treatment as well as support in breastfeeding, working together with the health visitor to monitor infant growth and weight gain. Involving the partner throughout the pregnancy and postpartum period improves outcomes.

Prescribing in Eating Disorders

Finally here is a brief note on prescribing. There is limited evidence for the use of psychotropic medication in the treatment of ED.⁶⁷⁻⁶⁹ Claudino *et al.*⁷⁰ did not find enough evidence in their Cochrane Review to recommend antidepressants in the treatment of AN. In the Cochrane Review⁷¹ by Bacaltchuk and Hay, the conclusion was that the use of a single antidepressant agent is clinically effective for the treatment of BN when compared to placebo, although there was no difference between classes, with an overall greater remission rate but a higher rate of dropouts compared with CBT. The meta-analysis by Stefano *et al.*⁷² showed that binge eating remission rates were higher in patients receiving antidepressants when compared with placebo. However, most studies were short trials (median duration of 8 weeks) and the only 16-week study did not show superiority of antidepressants over placebo.

Recently there have been small studies on the use of aripiprazole in all types of EDs, with evidence that it reduces distress around eating, reduces obsessional thoughts about food, weight, and body image, significantly lessening ED behaviours. Other comorbidities, like depression, generalised anxiety, and cognitive flexibility, improved as well.⁷³

Due to low BMI, in particular lower than 16, the response to psychotropic medication is poor when prescribed for other comorbidities. Therefore it is advised that the dose of these medications needs to be quickly increased to their maximum dose, according to tolerance and efficacy, and longer time should be allowed to ascertain efficacy or lack thereof. On the other hand, the doses of other medications, like analgesics and antibiotic, need to be adjusted and frequently reduced, depending on liver and renal functions.

Key Points

- Eating disorders are the psychiatric illnesses with highest mortality, 50% by suicide in AN.
- Eating disorders can affect fertility and unexpected pregnancies can occur. They can negatively impact pregnancy outcomes, and even though frequently there is remission during pregnancy, relapse is frequent in the postnatal period.
- Eating disorder should be regularly screened during perinatal period, particularly if there are concerns about weight gain and abnormal gastrointestinal symptoms.
- Eating disorders are difficult to treat and treatments are only moderately effective; therefore early intervention is essential for better prognosis and outcomes.
- Management depends on severity and risk factor and should be, whenever possible, administered by specialist ED services. Psychological treatments are still the treatments of choice at present.

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MULTIPLE CHOICE QUESTIONS

- Q1.** What percentage of patients fully recover from eating disorder:
- 10%
 - 4%
 - Less than 50%
 - More than 80%
- Q2.** Which one does not predict higher risk of suicide in eating disorders:
- Lower BMI
 - Purging behaviours
 - Deliberate self-harm
 - No excess exercise
- Q3.** Which one is false:
- Around 30% of patients have a relapsing remitting course
 - Women with ED are at higher risk of postnatal depression
 - Women with ED stop breastfeeding earlier than other women
 - The postnatal period is protective of relapse of ED
- Q4.** Which one is true:
- Patients with anorexia nervosa frequently present with Russell sign
 - Patients with Bulimia Nervosa can develop kidney failure
 - Patient with binge eating disorder are usually normal weight
 - Patients that abuse laxatives lose more weight
- Q5.** What is the treatment of choice for eating disorders:
- Psychological interventions
 - Fluoxetine
 - Nasogastric feeding
 - None of the above

Answers:

Q1. c; Q2. d; Q3. d; Q4. b; Q5. a
