SECTION 15: TREATMENT OF EATING DISORDERS
15.1 Introduction

Please review the Trust document “Guidelines for the assessment and treatment of eating disorders” in the CAMHS Operational Policy.

When screening for eating disorders one or two simple questions should be considered for use with specific target groups

1. Do you think you have an eating problem?
2. Do you worry excessively about your weight?

Early detection may be helped by five screening questions using The SCOFF questionnaire.

A score of two or more positive answers should raise clinical suspicion and lead to an in depth diagnostic evaluation.

1. Do you ever make yourself Sick because you feel uncomfortably full?
2. Do you worry you have lost Control over how much you eat?
3. Have you recently lost more than One stone in a three month period?
4. Do you believe yourself to be Fat when others say you are too thin?
5. Would you say that Food dominates your life?

It is important to take into account that clients with eating disorders can develop Acute Kidney Injury through a variety of mechanisms associated with each condition. Clinicians should be vigilant in the monitoring of physical health especially serum creatinine and levels of hydration.3

15.2 Anorexia nervosa

The following would represent a reasonable initial screen for Anorexia Nervosa in primary care if there are no other indications or diagnostic concerns:

Full Blood Count, ESR, Urea and Electrolytes, Creatinine, Liver Function Tests, Random Blood Glucose, Urinalysis, ECG (should be considered in all cases and essential if symptoms/signs of compromised cardiac function, bradycardia, electrolyte abnormality and/or BMI less than 15 kg/m2 or equivalent on centile chart).

Further tests may be required in more severe cases or to assess complications: Calcium, Magnesium, Phosphate, Serum Proteins, Creatine Kinase. Tests that may be needed in the differential diagnosis of amenorrhoea and weight loss include Thyroid Function Tests, Follicle Stimulating Hormone, Luteinising Hormone, Prolactin and Chest X-Ray.

A DXA scan may be considered for identification of osteopenia/osteoporosis, which may occur after six to twelve months of amenorrhoea. Although this is not necessarily a primary care level investigation, it has been suggested that it may be helpful in encouraging motivation for change in those not yet ready to accept referral by demonstrating the real physical consequences of anorexia nervosa.
Section 15. Treatment of eating disorders

There is a very limited evidence base for the pharmacological treatment of anorexia nervosa. A range of drugs may be used in the treatment of co-morbid conditions but caution should be exercised in their use given the physical vulnerability of many people with anorexia nervosa.

15.2.1 Pharmacological interventions

- Medication **should not** be used as the sole or primary treatment for anorexia nervosa.

- Caution should be exercised in the use of medication for co-morbid conditions such as depressive or obsessive compulsive features, as they may resolve with weight gain alone.

- When medication is used to treat people with anorexia nervosa, the side effects of drug treatment (in particular, cardiac side effects) should be carefully considered and discussed with the patient because of the compromised cardiovascular function of many people with anorexia nervosa.

- Prescribers should be aware of the risk of drugs that prolong the QTc interval. These include antipsychotics, tricyclic antidepressants, macrolide antibiotics, and some antihistamines. The prescribing of drugs with side effects that may compromise cardiac functioning should be avoided in patients with anorexia nervosa at risk of cardiac complications.

- If a prescription for medication that may compromise cardiac functioning is essential, ECG monitoring should be undertaken.

- Patients with a diagnosis of anorexia nervosa should have an alert placed in their prescribing record concerning the risk of side effects.

15.2.2 Physical management

- Regular physical monitoring, and in some cases treatment with a multi-vitamin/multi-mineral supplement in oral form is during both inpatient and outpatient weight restoration.

- Oestrogen administration should not be used to treat bone density problems in children and adolescents as this may lead to premature fusion of the epiphyses.

15.3 Bulimia nervosa

In **Bulimia Nervosa** and related conditions, characteristic physical signs include parotid enlargement, Russell’s sign (callus formation on the dorsum of the hand) and dental enamel erosion, all of which are usually manifestations of purging. In practice these are not seen in the majority of patients presenting in primary care with bulimic disorders, although electrolyte abnormalities are reasonably common so urea, and electrolytes should be routinely obtained.

Fluid and electrolyte disturbances occur in bulimia nervosa and relate to the severity of symptoms and the general nutritional status. Common abnormalities include dehydration, hypokalaemia, hypochloraeemia, and alkalosis. Dehydration can cause volume depletion and consequently low blood pressure with a rapid pulse. Patients can complain of
dizziness because of orthostatic hypotension and weakness. In extreme cases, renal function can be compromised. Secondary hypoaldosteronism can lead to rebound, fluid retention and peripheral oedema when laxatives and diuretics are withdrawn. Low potassium causes weakness in all muscle and most worryingly cardiac arrhythmias, which may lead to death in severe cases. Renal function can also be affected. Metabolic alkalosis may augment potassium depletion. Diuretic abuse, particularly thiazide and loop diuretics, can produce marked potassium and sodium depletion. Low sodium and magnesium levels occur less commonly but both have potentially serious consequences. If severe the former may cause central nervous system disturbances, whilst the latter results in muscle weakness, cardiac arrhythmias and mood changes. Low magnesium is also associated with other abnormalities such as hypocalcaemia and hypokalaemia. Clinicians should consider the presence of low magnesium levels in the face of refractory hypokalaemia. In general, these abnormalities settle with cessation of purging behaviours. If needed, oral rather than IV supplementation is advised. Advice from a physician and/or paediatrician may be necessary if there is severe metabolic disturbance (Connan, Lightman & Treasure, 2000). Very rarely patients may require hospital admission to manage severe purging behaviour.

15.3.1 Pharmacological interventions

- As an alternative/additional first step to using an self-help programme, adults with bulimia nervosa may be offered a trial of an antidepressant drug.

- Patients should be informed that antidepressant drugs can reduce the frequency of binge eating and purging, but the long-term effects are unknown. Any beneficial effects will be rapidly apparent.

- Selective serotonin reuptake inhibitors (specifically fluoxetine) are the drugs of first choice for the treatment of bulimia nervosa in terms of acceptability, tolerability and reduction of symptoms. For people with bulimia nervosa, the effective dose of fluoxetine is higher than for depression (60 mg daily).

- Other than antidepressants, no other drugs are recommended for the treatment of bulimia nervosa.

15.3.2 Physical management

- When electrolyte disturbance is detected, it is usually sufficient to focus on eliminating the behaviour responsible. In the small proportion of cases where supplementation is required to restore electrolyte balance, oral rather than intravenous administration is recommended unless there are problems with gastrointestinal absorption.

15.3 Atypical disorders including binge eating disorder

As an alternative or additional first step to using an evidence-based self-help programme, consideration should be given to offering a trial of an SSRI antidepressant drug to patients with binge eating disorder.

Patients with binge eating disorders should be informed that SSRIs can reduce binge eating, but the long-term effects are unknown. Antidepressant drug treatment may be sufficient treatment for a limited subset of patients.
15.4 Re-feeding syndrome

Re-feeding problems encompass life-threatening acute micronutrient deficiencies, fluid and electrolyte imbalances, and disturbances of organ function and metabolic regulation that may result from over-rapid or unbalanced nutrition support. They can occur in any severely malnourished individual but are particularly common in those who have had very little or no food intake, even including overweight patients who have eaten nothing for protracted periods. Re-Feeding Syndrome is characterised by a range of life-threatening clinical and biochemical abnormalities including cardiac failure, pulmonary oedema, dysrhythmias, acute circulatory fluid overload or circulatory fluid depletion, hypophosphatemia, hypokalaemia, hypomagnesaemia and occasionally hypocalcaemia and/or hyperglycaemia.

The problems arise because starvation causes adaptive reductions in cellular activity and organ function accompanied by micronutrient, mineral and electrolyte deficiencies. Abnormalities in malnourished individuals may therefore include:

- Deficiencies of vitamins and trace elements
- Whole body depletion of intracellular potassium, magnesium and phosphate;
- Increased intracellular and whole body sodium and water
- Low insulin levels and a partial switch from carbohydrate metabolism to ketone metabolism to provide energy
- Impaired cardiac and renal reserve with decreased ability to excrete an excess salt and water load
- Abnormalities of liver function

Giving nutrients and fluid to malnourished patients will reverse these changes but in doing so leads to an increase in demands for electrolytes and micronutrients, and a simultaneous shift of sodium and water out of cells. Over-rapid or unbalanced nutrition support can therefore precipitate acute micronutrient deficiencies and dangerous changes in fluid and electrolyte balance.

Criteria for determining people at high risk of developing re-feeding problems

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<th>Patient has one or more of the following:</th>
<th>Or patient has two or more of the following:</th>
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<td>1. BMI less than 16 kg/m²</td>
<td>1. BMI less than 18.5 kg/m²</td>
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<td>2. Unintentional weight loss greater than 15%</td>
<td>2. Unintentional weight loss greater than 10% within the last 3–6 months</td>
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<td>3. Within the last 3–6 months</td>
<td>3. Little or no nutritional intake for more than 5 days</td>
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<td>4. Little or no nutritional intake for more than 10 days</td>
<td>4. A history of alcohol abuse or drugs including insulin, chemotherapy, antacids or diuretics</td>
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<td>5. Low levels of potassium, phosphate or magnesium prior to feeding</td>
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Focussing on patients with eating disorders, people at most risk are those with a BMI less than 12 kg/m², those who vomit, abuse laxatives and binge, and those with concurrent physical conditions. Patients at high risk of re-feeding syndrome should commence feeding at very low levels of energy and protein but with generous provision of thiamine and other B group vitamins, along with a balanced multi-vitamin and trace element supplement (since they are likely to have multiple deficits that cannot be met by low level oral, enteral or parenteral intake). Levels can then be increased over the next few days as careful monitoring reveals no problems.

Most patients at high re-feeding risk also need generous supplementation of potassium, magnesium and phosphate from the onset of feeding unless blood levels are already high (this may be the case in patients who have renal impairment). It is important to appreciate those patients with normal pre-feeding levels of potassium, magnesium and phosphate can still be at high risk, and that many of those with high plasma levels will still have whole body depletion and may therefore need supplementation as re-feeding progresses and renal function improves.

Treatment of re-feeding syndrome should be undertaken in an acute medical setting and not in a psychiatric ward. A suggested monitoring schedule with parameters can be found in ‘Clinical Laboratory Investigation and Psychiatry: A Practical Handbook, 2008’. Informa Healthcare USA, Inc.

Appendix 1 provides a guide to the medical risk assessment for eating disorders.

References

3. Guidance for Mental Health Professionals on the management of Acute Kidney Injury
A GUIDE TO THE MEDICAL RISK ASSESSMENT FOR EATING DISORDERS
by Professor Janet Treasure (2009)

adapted for use with patients with eating disorders:
outpatients in primary and secondary care, medical inpatients, general psychiatric inpatients and
eating disorder inpatients.

People with eating disorders, in particular those with anorexia nervosa, are at high risk in
terms of their own health and safety. They have the highest mortality of any psychiatric
illness. Both their physical state and suicidal behaviors contribute to this risk. Risk to
others is less of a concern.

The factors involved in the assessment of risk in people with eating disorders include:
• medical risk
• psychological risk
• psychosocial risk
• insight/capacity and motivation.

A proxy measure for insight/motivation is the response to treatment. If medical risk is
high and there is no response to outpatient treatment, it is necessary to measure
capacity and consider the use of mental health law.

This Guide aims to help in the understanding of:
1. the medical risk - how to assess it, evaluate it and where to refer.
2. the use of the Mental Health Act in treatment.

MEDICAL RISK

The medical risk arises from a combination of the restrictive behaviours (food and in some
cases fluid) and the compensatory behaviours.

Features in history that indicate medical risk are:
• excess exercise with low weight
• blood in vomit
• inadequate fluid intake in combination with poor eating
• rapid weight loss
• factors which disrupt ritualised eating habits (journey/holiday/exam).

Body mass index (weight/height²) is a proxy measure of medical risk in anorexia nervosa
(see Maudsley Body-Mass Index Table). Metabolic changes are most problematic if weight
control measures such as vomiting and laxative abuse are used. Neither BMI nor blood
tests alone are adequate markers of risk. Screening for risk with an examination of muscle
strength, blood pressure, pulse rate, peripheral circulation and core temperature is
essential.