CME CREDIT ***EDUCATIONAL OBJECTIVE:*** Readers will anticipate and manage the medical consequences of eating disorders

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Recognizing, managing medical consequences of eating disorders in primary care

ABSTRACT

Eating disorders can lead to serious health problems, and as in many other disorders, primary care physicians are on the front line. Problems that can arise from intentional malnutrition and from purging affect multiple organ systems. Treatment challenges include maximizing weight gain while avoiding the refeeding syndrome.

KEY POINTS

The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5), released in 2013, has updated the criteria for some eating disorders and has added some new disorders.

Starvation can cause cardiac, cerebral, gastrointestinal, and endocrine problems.

Purging can lead to problems with oral health, electrolyte imbalances, and even renal failure.

Refeeding poses the risk of refeeding syndrome, with fluid overload and electrolyte imbalances. Many patients undergoing refeeding are best managed in the hospital.

EATING DISORDERS are debilitating biopsychosocial illnesses associated with serious medical illness and a high risk of death.[1]

Primary care physicians are often the first to see young women who have these problems, diagnose them, and start their evaluation and treatment.[2–4] Many patients require acute medical interventions as well as long-term care for chronic medical issues. Therefore, primary care physicians play essential front-line and long-term roles in the multidisciplinary treatment team.

DEFINITIONS OF EATING DISORDERS HAVE CHANGED

Several problems existed in the category of eating disorders in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-4) and in the DSM-4 Text Revision (DSM-4-TR). These problems have been addressed in the fifth edition (DSM-5), released in 2013.[5]

One problem in the earlier editions was that many patients referred for treatment of eating disorders—more than 50% in one study[6]—did not meet the criteria for anorexia nervosa or bulimia nervosa and thus had to be categorized as having "eating disorder not otherwise specified." Further, the earlier editions did not recognize that young children and adolescent males can be affected.[7]

Eating disorders are now recognized as an equal-opportunity disease, with all ethnic and socioeconomic groups affected. Children can run into medical trouble with even a small amount of weight loss or falling off the growth curve. Moreover, children and adolescents do

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TABLE 1

Commonly used drugs that prolong the QTc interval

Antibiotics and antifungals
Fluoroquinolones
Erythromycin, azithromycin
Trimethoprim-sulfamethoxazole
Fluconazole, ketoconazole

Antipsychotics
Haloperidol
Risperidone
Chlorpromazine

Tricyclic antidepressants
Imipramine
Desipramine
Amitriptyline
Nortriptyline

Antiemetics
Dolasetron
Ondansetron

Antihistamines
Diphenhydramine

Antiarrhythmics
Class IA (quinidine, procainamide, disopyramide)
Class IC (flecainide, encainide)

A full list of drugs that prolong the QTc interval is maintained at http://www.torsades.org.[18]

'Eating disorder not otherwise specified' was a wastebasket category

not "experience" their bodies in the same way adults do; they may lack the vocabulary for eating-disorder thoughts.

For these reasons, the definitions of eating disorders have changed in the DSM-5.[5]

Anorexia nervosa. Older editions of the DSM listed amenorrhea as a criterion. This has been eliminated in DSM-5, since amenorrhea does not necessarily predict medical risk or treatment outcome; also, it is not applicable to males or premenorrheal girls and postmenopausal women.[8] In addition, the requirement of low weight is now defined in the context of "age, sex, developmental trajectory, and physical health," rather than the old threshold of 85% of expected weight.[9]

What remains unchanged is that anorexia nervosa is still characterized by self-starvation in order to maintain an abnormally low body weight, along with an intense fear of being fat and a disturbed self-image.

Bulimia nervosa. In both the old and the new editions of the DSM, bulimia nervosa is characterized by episodes of binge eating followed by inappropriate compensatory behaviors to avoid weight gain, such as vomiting, laxative abuse, diuretic abuse, and overexercise. In DSM-5, bulimia nervosa no longer has subtypes and requires only one binge per week with compensatory behavior, for at least 3 months. This change was based on the finding that there is no clear difference in psychopathology or treatment outcome between patients with one and two binge-purge episodes a week.[10]

"Eating disorder not otherwise specified" was a wastebasket category, lumping all those who did not meet the criteria for anorexia nervosa or bulimia nervosa or who did not neatly fit into a specific category.[10] In DSM-5, subcategories were designed to help distinguish different treatment needs and outcomes between various subtypes.

Binge-eating disorder, one of the new subcategories, is characterized by binge eating without inappropriate compensatory behaviors.[9] Patients with binge-eating disorder are often obese, have greater functional impairment, and are more likely to develop components of metabolic syndrome than obese patients without eating disorders.[11]

Avoidant/restrictive food intake disorder is another new DSM-5 diagnosis, characterized by failure to meet nutritional needs for reasons other than weight control. Reasons include disinterest in eating, dislike of sensory characteristics of food, or avoidance of consequences of eating. This disorder replaces the category "feeding disorder of infancy or early childhood," since the condition can also occur in adolescents and adults.[12]

Other new diagnoses are:

- Atypical anorexia nervosa (if the patient is not underweight)
- Purging disorder
- Subthreshold bulimia nervosa (if the patient has < 1 episode per week or has had them for < 3 months)
- Subthreshold binge eating disorder (< 1 time a week or < 3 months)
- Night eating syndrome
- Pica and rumination disorder.

Regardless of the diagnostic label, the medical evaluation and treatment of anyone

with an eating disorder should be tailored to the specific behaviors of the eating disorder. Medical complications can be subdivided into those from starvation, from purging, and from refeeding.

MEDICAL COMPLICATIONS OF STARVATION

Cardiovascular effects of starvation

Malnutrition and starvation have multiple adverse effects on the heart.

Electrophysiologic effects. Sinus bradycardia (< 60 bpm) and hypotension are common cardiac manifestations of starvation.[13] Bradycardia has been attributed to an adaptive increase in parasympathetic vagal tone.[14] QTc prolongation is also seen in patients with malnutrition.[15]

Together, these electrocardiographic abnormalities predispose the patient to ventricular arrhythmia and sudden cardiac death.[16] The risk of ventricular arrhythmia is particularly relevant when treating psychiatric symptoms, since antipsychotics and tricyclic antidepressants are among several drug classes that can cause further QTc prolongation (TABLE 1).[17,18]

In patients with QTc prolongation, bradycardia, or both, the standard of care involves acute hospitalization for refeeding using continuous telemetric monitoring until normal rhythm is restored and the heart rate is above 40 at night and 50 by day.[4,19]

Structural changes. Starvation also causes structural changes in the heart. Loss of lean body mass can reduce cardiac muscle mass, compromise cardiac output, and lead to mitral valve prolapse.[20] These changes are fully reversible with restored nutrition and regaining of heart mass.[21,22]

Effects of starvation on the brain

Starvation can affect brain structure and cognitive function. Undernourished patients have reduced volumes of white and gray matter, a change that can occur within months. Cortical volumes may increase with weight gain, but a reduction in gray matter volume may not be completely reversible.[23]

Furthermore, starvation impairs cognitive functions that are needed to stop eating-disorder behaviors; namely, decision-making, emotional control, regulation of appetite, and reward pathways. Therefore, undernourished patients may not have sufficient insight into the disease to be able to make the best choices for recovery. This finding lends support for using the Maudsley method in adolescents, in which parents take control of their child's eating until the child can maintain a healthy weight.[24]

Gastrointestinal consequences of starvation

Patients with malnutrition have prolonged gastric emptying and colonic transit time with solid foods.[25] They often complain of early satiety, abdominal pain, bloating, and constipation, all symptoms that complicate the refeeding process. A prokinetic such as metoclopramide (Reglan), given 1 hour before meals and at bedtime, may provide some relief from gastrointestinal symptoms.[26]

Patients may also experience transient lactose or fructose intolerance after prolonged starvation. Taking a lactase supplement (eg, Lactaid 1–10 tabs) before consuming dairy products and dextrose (contained in candies such as Smarties) before eating fruit or fructose-containing foods can sometimes partially relieve symptoms. In general, gastrointestinal function returns over time as nutritional status improves.

Patients with severe or prolonged starvation can develop steatosis accompanied by elevated levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT). In reports of starvation-induced steatosis, liver enzyme levels rapidly normalize with nutritional rehabilitation.[27]

Endocrine consequences of starvation

Amenorrhea. Dysregulation of the hypothalamic-pituitary-gonadal axis is a major endocrine complication of nutritional insufficiency. Weight loss disrupts the normal pulsatile secretion of gonadotropin-releasing hormone, reduces secretion of luteinizing hormone and follicle-stimulating hormone, and decreases estrogen levels.[28] Leptin deficiency likely plays a role in suppressing gonadotropin secretion with subsequent development of amenorrhea. With weight gain, levels of leptin and gonadotropins normalize and menstruation eventually returns.[29,30]

Hypothyroidism. Starvation can also lead to dysregulation of the hypothalamic-pituitary-

Bradycardia plus QTc prolongation with electrolyte abnormalities predisposes to ventricular arrhythmia and sudden cardiac death

thyroid axis. Typically, the concentration of triiodothyronine (T3) is reduced, the ratio of thyroxine (T4) to T3 is elevated, and thyroid-stimulating hormone (TSH) is close to or within the normal range, creating a euthyroid sick syndrome. In eating disorders, this thyroid disturbance is a result of starvation and resolves with weight restoration. Therefore, thyroid hormone replacement therapy is not medically indicated.[28]

Osteoporosis. Amenorrhea resulting from low estrogen levels in undernourished patients can raise the risk of osteoporosis and fractures, particularly in patients with a low body mass index. Osteopenia results from a negative balance between bone deposition and resorption.

Lack of bone deposition can be especially problematic when disordered eating occurs during peak bone mass development, ie, ages 11 to 14 for girls, and ages 15 to 17 for boys.[31,32] Even a 5% to 10% decrease in bone deposition can result in significant risk of osteopenia.[33] However, after age 30, bone resorption is a greater contributor.[34]

Does hormone therapy correct bone loss? Given the association between estrogen deficiency and bone loss, estrogen supplementation was expected to be an effective treatment for bone loss in patients with eating disorders.[35] Also, the restoration of menses through hormone replacement may give underweight patients a false sense of achieving a "healthy" weight.[36]

Undernourished patients may lack the insight to make good choices for recovery

Golden et al[37] prospectively studied 50 adolescents and found no significant difference in bone mineral density at 1 year of follow-up between patients treated with estrogen and those who received only standard nutritional therapy. However, increased bone mineral density was achieved in adolescents with anorexia nervosa treated with transdermally administered estrogen dosed to mimic physiologic pubertal levels.[38]

Klibanski et al[39] found that hormone therapy resulted in a 4% gain in bone density in an extremely low-weight subset of women with anorexia nervosa (< 70% of ideal body weight), whereas similar patients in the control group lost 20%. However, in all groups, only weight gain correlated with bone gain in women who were within 70% of their ideal body weight.

Divasta et al[40] evaluated 60 girls and women ages 13 to 27 with anorexia nervosa, randomized to receive either placebo or dehydroepiandrosterone combined with an estrogen-progestin oral contraceptive, and followed for 18 months. As in the study by Klibanski et al,[39] bone loss was prevented in the treatment group, but significant bone gain occurred only in the context of weight gain.

The bottom line is that only weight gain has resulted in significant increases in bone density in patients with anorexia nervosa, and hormone therapy without weight gain has not been shown to increase bone density effectively in this population. Although calcium and vitamin D in oral therapeutic doses through foods or through supplementation are required for bone gain, the combination is not enough to augment bone density in the absence of weight gain.[37] Although not curative, weight gain is currently the best option for treating bone loss, and no single pharmacologic treatment is effective.

COMPLICATIONS OF PURGING

Oral complications of purging

Patients who purge by vomiting are at risk of complications from exposure of the esophagus, pharynx, and mouth to acidic gastric contents.

Dental problems. Over time, contact with gastric acid wears down enamel on the lingual and occlusal surfaces of teeth, resulting in dental caries and periodontal disease. Until they can give up purging, patients should be instructed to rinse with mouthwash or water immediately after vomiting to reduce the acidity in the mouth.[41,42] We recommend that patients not brush their teeth after vomiting, because brushing can deliver acid to otherwise unreachable surfaces and thus worsen tooth erosion. For patients who are determined to brush after vomiting, a bicarbonate toothpaste might mitigate harm.[42]

Sialadenosis (hypertrophy of the salivary glands) is another consequence of repeated vomiting, with elevated salivary amylase. Both the size of the glands and the salivary amylase level generally normalize on their own after vomiting is stopped, but parotitis can take up to a year to resolve. Similar to smoker's cough, parotitis may acutely worsen when the patient

abruptly stops vomiting and may worsen before it improves.

To reduce discomfort, patients can use hot compresses or sugarless hard candies.[44] However, the latter should not be substituted as a chronic habit in a patient with disordered eating. Patients need to be reassured that the swelling is not permanent, since they often interpret it as having fat cheeks (the "chipmunk sign").

Hypokalemia, metabolic alkalosis, renal dysfunction

Chronic vomiting can cause electrolyte and acid-base imbalances, the most worrisome of which is hypokalemia. With repeated vomiting, loss of potassium and gastric acid causes metabolic alkalosis with hypokalemia, hypochloremia, and hypomagnesemia. Loss of water and the resultant volume contraction activates the renin-angiotensin-aldosterone system, and elevated aldosterone further decreases serum potassium.

In patients with eating disorders, who often have other factors contributing to electrolyte imbalance, vomiting-induced hypokalemia heightens the risk of cardiac arrhythmias.[43]

Hypokalemia can also cause rhabdomyolysis and kidney damage.[41,43] Prolonged hypokalemia and reduced kidney perfusion in the setting of volume depletion causes acute kidney injury and impaired concentrating ability of the renal tubules. Hypovolemia can cause prerenal azotemia and increases the risk for nephrolithiasis and nephrocalcinosis.[44,45]

When a patient stops vomiting, elevated aldosterone from prior hypovolemia results in water retention and can manifest in significant edema associated with hypochloremic alkalosis. This condition, known as pseudo-Bartter syndrome, usually resolves without treatment. In the meantime, salt restriction and leg elevation can help reduce edema.[26]

Laxative abuse: A mode of purging

Many patients with eating disorders abuse laxatives to lose weight or to prevent weight gain. Believing that laxatives will prevent calorie absorption, patients commonly take them to compensate for caloric intake (eg, during a binge episode). The immediate weight loss, albeit artificial, is highly reinforcing for an eating-disorder patient. In some cases, patients with eating disorders also abuse laxatives to self-treat the constipation that results from chronic starvation.[46]

Over time, tolerance to laxatives develops, and patients use increasingly larger doses. This can lead to activation of the renin-angiotensin-aldosterone system.[47] Patients interpret the resultant edema as true weight gain and again take laxatives to get rid of it. If laxatives are stopped abruptly, the patient may need inpatient and outpatient support for the resultant fluid shifts.

Gastrointestinal complications of laxative abuse include reflex hypofunction of the bowel, malabsorption, steatorrhea, and gastrointestinal bleeding.[47] Reflex hypofunction during laxative withdrawal is a consequence of the bowel becoming tolerant of laxatives.[48] Cathartic colon syndrome is a rare complication characterized by loss of the normal haustral markings and slowed or absent peristalsis in segments of the colon.[49]

Systemically, the major risk of laxative abuse relates to electrolyte and acid-base imbalance. Loss of potassium and water in the stool can cause hypokalemia and metabolic alkalosis.[48] The disturbances caused by laxative abuse are similar to those caused by vomiting and diuretic use and have the same treatment.

Amenorrhea is a major endocrine complication of nutritional insufficiency

The most important component of treating laxative abuse is giving patients realistic expectations to help them tolerate temporary discomfort and to help manage the edema and fluid shifts that can happen acutely with shifting of fluid into the intracellular space. In extreme cases, this may need to be managed in the hospital. To help relieve the initial anxiety, doctors should emphasize that any bloating the patient experiences is not true weight gain and will go away within a few days to weeks. In addition, explaining that laxatives reduce nutrient absorption only minimally may lessen the temptation to resume taking them.[48]

Diuretic abuse: Another form of purging

Diuretic abuse is yet another mode of purging, with its own set of medical complications. Like laxatives, diuretics are not effective weight-loss agents, and the weight reduction they cause is only temporary.

TABLE 2

Guidelines for refeeding undernourished patients[45]

Baseline assessment
Complete metabolic panel, including serum electrolytes (sodium, potassium, chloride, bicarbonate, calcium, phosphorus, magnesium) glucose, aspartate aminotransferase and alanine aminotransferase (liver function), blood urea nitrogen and creatinine (renal function)
Thyroid-stimulating hormone
Complete blood cell count
Erythrocyte sedimentation rate or C-reactive protein level
Urinalysis

Continuous ECG monitoring

Supplement during refeeding
Phosphate (sodium and potassium phosphate 500 mg [2 Neutraphos packets] twice daily for 5 days) unless levels are elevated at baseline
Multivitamin, including trace elements and vitamin B complex

Begin refeeding
Begin at 1,500–2,000 kcal/day
Increase by 200–300 kcal/day to achieve weight gain of 0.2 kg/day
Begin fluids at 1,500–2,000 mL/day, favoring caloric drinks over free water

Monitor daily until stable, then as clinically indicated
Vital signs
Input and output
Blood chemistry (if electrolytes abnormalities require correction)
Urinalysis

Detect refeeding syndrome early
Altered mental status
Hepatosplenomegaly
Atypical abdominal pain
Peripheral edema
Tachycardia
QTc prolongation
Severe hypokalemia (< 3 mmol/L) or hypophosphatemia (< 0.8 mmol/L)

As with vomiting, there is a compensatory activation of the renin-angiotensin-aldosterone system, and therefore subsequent fluid intake will lead to water retention, which encourages further diuretic use.[41] Diuretics can also contribute to hypokalemia, hypomagnesemia, hypochloremia, and metabolic alkalosis.

Ipecac abuse can lead to heart failure

Ipecac syrup has long been used to induce vomiting, but this practice has become much less common since ipecac has become harder to obtain in the United States.[50] The emetine base contained in ipecac binds irreversibly to cardiac and skeletal muscle. With continued use, irreversible cardiomyopathy develops and can lead to heart failure. Treatment should include supportive care and immediate cessation of ipecac use.

Diabetic patients may skip insulin to lose weight

Patients with diabetes, especially those with type 1 that begins in childhood, are at greater risk of eating disorders over time.[51] They may withhold insulin to lose weight, a practice referred to in the nonmedical literature as "diabulimia," and they seem particularly more likely to develop bulimia nervosa than those without diabetes.[52]

The medical prognosis is poor for patients with diabetes who develop eating disorders and do not receive intensive treatment.[51] In addition, if a diabetic patient on an insulin pump becomes depressed in addition to having an eating disorder, careful monitoring for suicidal thoughts and a rapid follow-up with mental health services are in order.

REFEEDING SYNDROME

When refeeding is started, a high glucose load stimulates insulin secretion, resulting in cellular uptake of phosphorus along with potassium, magnesium, and glucose. In addition, total body phosphorus is depleted by the increased demand for adenosine triphosphate and 2,3-diphosphoglycerate for cellular metabolism.

When liver enzyme levels increase, the astute clinician will closely monitor the patient for evidence of refeeding syndrome. In a child, adolescent, or young adult, the standard of care is inpatient monitoring for acute stabilization.[4,19]

Hypophosphatemia is the hallmark of refeeding syndrome, although hypomagnesemia, hypokalemia, and hypoglycemia can also occur.[53] In addition, sodium and water retention can lead to fluid overload, with shifting of fluid into the intracellular space, resulting in dependent edema.

Cardiovascular complications are the most worrisome manifestations of refeeding syndrome. Electrolyte shifts and increased fluid

volume can cause arrhythmias and heart failure. Furthermore, severely undernourished patients may have reduced myocardial mass as well as electrocardiographic abnormalities associated with starvation, which further increase their vulnerability to electrolyte shifts and fluid retention during refeeding.[15]

Other manifestations of refeeding syndrome include delirium, seizures, rhabdomyolysis, and respiratory failure. In the most extreme cases, refeeding syndrome causes sudden death.[53]

Fortunately, refeeding syndrome is easily preventable and treatable when recognized early. Electrolytes and cardiovascular and renal function must be carefully monitored, especially during the first week of nutritional restoration.[53] In patients with extremely low body mass (< 70% of ideal body weight) or with precipitous weight loss, close monitoring of the complete metabolic panel including electrolytes, AST, ALT, calcium, magnesium, and phosphorus may be required to detect changes that can affect cardiac status. Specific suggestions for refeeding are discussed below and in TABLE 2.[45]

TABLE 3

Criteria for hospitalization of an eating disorder patient[4,54]

Medical

< 75% ideal body weight, significant or continued weight loss
Temperature < 96°F (36°C)
Heart rate < 50 bpm daytime; < 40 bpm nighttime
Systolic pressure < 90 mm Hg
Orthostatic changes in pulse (> 20 bpm) or diastolic blood pressure (> 10 mm Hg)
Cardiac arrhythmia, including prolonged QTc interval
Serum potassium < 3.2 mmol/L
Serum chloride < 88 mmol/L

Behavioral

Refusal to eat
Need for physical activity restriction or bed rest
Failure to respond to outpatient treatment
Suicide risk

ACUTE CARE OF PATIENTS WITH EATING DISORDERS

Refeeding in the inpatient setting

The decision to hospitalize an eating-disorder patient is based on the current or potential risk of serious medical complications and the likelihood of success at home. Medical criteria for hospital admission are outlined in TABLE 3.[4,54]

In refeeding undernourished patients, the challenge is to maximize weight gain while preventing refeeding syndrome. Undernourished patients are generally hypometabolic at baseline but become hypermetabolic once refeeding begins.

How many calories should refeeding start with? The traditional principle of "start low and go slow" has been recently challenged.[55] Starting at 1,200 kcal/day or less in the typical patient can result in failure to gain weight or even in weight loss in the first week of refeeding.[56] The goal is to achieve a weight gain of 0.2 kg/day while the patient is in the hospital. Thus, we start higher, and to date we have seen no cases of life-threatening refeeding syndrome. In all patients who need hospitalization or who are beginning the refeeding process as outpatients, caloric intake should be started at 1,500 to 2,000 kcal/day.[45,57] However, for exceptionally low-weight patients, intake may be started lower.

In Australia, patients are started at 1,900 kcal/day.[56] All patients in one program there receive nasogastric feeding initially in an intensive care unit and then are moved to a regular nursing floor where they graduate to full oral feeding as they improve cardiovascularly and behaviorally. In the United States, some programs use nasogastric feeding at night for caloric restoration; our program and others use nasogastric feeding as a behavioral modification strategy for patients who refuse food or supplements by mouth.

Over time, tolerance to laxatives develops, and patients use increasingly larger doses

Phosphorus supplementation. Many centers give phosphorus supplements preventively. In our center, we give potassium phosphate (Neutra-Phos) 500 mg orally twice daily for 5 days, and we have seen no life-threatening cases of refeeding syndrome with that regimen. Other centers give phosphorus supplements in a dose of 250 mg orally twice a day for 5 days, while still others only supplement phosphorus reactively once a deficit has been identified. The latter method requires daily blood draws for monitoring and is reactive rather than proactive. Further studies can help clarify the optimal dosing and timing of phosphorus supplementation.

Managing fluid balance. Fluid-loading these patients may tip them over the edge into refeeding syndrome. Except in cases of shock, patients with eating disorders should not be given intravenous fluids, as it is safer to rehydrate and feed them orally. Electrolyte imbalances can be corrected orally with no need for intravenous supplementation. To avoid fluid overload, fluids can be started at 1,500 mL to 2,000 mL per day, with strict monitoring of intake and output. Fluids are liberalized if ALT and AST levels remain normal and to gradually correct orthostatic hypotension; caloric fluids are ideal to help address energy needs and improve bradycardia.

Laboratory monitoring. On admission, a urinalysis, complete blood cell count, complete metabolic panel, TSH, erythrocyte sedimentation rate, serum magnesium, and phosphorus should be obtained.[26] In addition, continuous electrocardiographic recording should begin on admission.[45] Inpatient use of a telemetry bed helps identify extreme tachycardia with arrhythmia, as well as profound bradycardia.[45,56]

Some protocols call for daily laboratory monitoring, although that degree of testing is less cost-effective. If initial results are normal, clinical judgment can be used on when to repeat laboratory evaluation. For instance, patients with edema require repeat complete metabolic panels to assess for elevated ALT and AST, electrolyte imbalances, and other abnormalities.

Signs of refeeding syndrome include tachycardia, hepatosplenomegaly, peripheral edema, altered mental status, and electrolyte disturbances, specifically, acute or severe hypophosphatemia or hypokalemia.[26,45] If refeeding syndrome is suspected, the rate of caloric intake should be reduced or not advanced, fluid intake should be urgently reassessed for volume overload, and supportive care with close monitoring should be provided.

KNOWLEDGE SAVES LIVES

Eating disorders can lead to potentially life-threatening medical complications that require attentive care by the primary care clinician and subspecialist. Without thoughtful consideration, it is easy for even a caring medical team to unintentionally enable patients with these illnesses or to cause active harm in the case of underrecognized pathology.[58]

Acute medical stabilization on an inpatient unit trained to recognize pathology and treat sequelae can be lifesaving. Arming patients and families with medical knowledge, as provided in the Academy for Eating Disorders' brochure, "Critical Points for Early Recognition and Medical Risk Management in the Care of Individuals with Eating Disorders"[59] can help save patients' lives.

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