Refeeding Syndrome

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Practice Gap

Refeeding syndrome can have potentially devastating metabolic consequences. It is important for the clinician to identify at-risk populations and to evaluate, recognize, and effectively manage this condition.

Objectives

After completing this article, readers should be able to:

1. Define refeeding syndrome.
2. Analyze patient scenarios for refeeding syndrome risk factors.
3. Evaluate the patient at risk for refeeding syndrome.
4. Interpret refeeding syndrome sequelae.
5. Manage the patient with refeeding syndrome.

CASE EXAMPLES

• **Case #1**: A 3-month-old infant is directly admitted to the hospital by his pediatrician for failure to thrive.

• **Case #2**: A 16-year-old girl with anorexia nervosa fails outpatient treatment and is referred for emergency department evaluation. Five months ago, the patient’s body mass index (BMI) was 18 and Z-score was -1.05, indicating mild malnutrition. Her current BMI is 14 and Z-score is -3.95, indicating severe malnutrition. (1)

• **Case #3**: A 9-year-old boy with multiple medical problems who is tracheostomy- and gastrostomy tube-dependent is referred to the hospital because of a 9-lb weight loss in the last 2 months.

INTRODUCTION

Refeeding syndrome was first described in the 1940s. (2) However, there is little consensus on the evaluation and management of this condition, particularly in children. One of the primary reasons for this lack of agreement is the inherent difficulty in studying patients with refeeding syndrome. In the 1940s, the Minnesota Starvation Experiment prospectively examined the effects of prolonged starvation in adults via a randomized, controlled trial, but this remains one of the few experiments of its kind examining this condition. (3) The reason for the dearth of research is likely due to the highly morbid complications of refeeding syndrome and starvation. Scant

AUTHOR DISCLOSURE Drs Pulcini and Srinath and Ms Zettle have disclosed no financial relationships relevant to this article. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.
evidence underlies the treatment recommendations for refeeding syndrome in pediatrics. Thus, current knowledge of this condition relies on cross-sectional and retrospective studies, and pediatric clinicians have limited tools to effectively evaluate and manage potential or recognized refeeding syndrome.

DEFINITION

Refeeding syndrome has been consistently described as the “electrolyte depletion, fluid retention, and altered glucose homeostasis that occurs in malnourished patients on commencing oral, enteral, or parenteral nutrition.” The difficulty in clearly defining and diagnosing refeeding syndrome lies in the discrepancy between shifts in homeostatic mechanisms and clinical symptoms. Some suggest making the diagnosis based on the onset of clinical symptoms, but many fluid and electrolyte changes occur in the absence of symptoms, and early awareness and appropriate therapy can decrease the chance of clinical deterioration. Thus, refeeding syndrome is defined as the metabolic and clinical changes that occur when a malnourished patient is aggressively nutritionally rehabilitated.

EPIDEMIOLOGY

Partly due to the lack of a precise definition of refeeding syndrome and its numerous potential clinical manifestations, it is difficult to estimate a true incidence of the condition. Hypophosphatemia is the most commonly reported and easily measured complication of refeeding syndrome. It is also the most widely reported surrogate marker for refeeding syndrome. An increased incidence of hypophosphatemia has been noted among those with eating disorders, most prominently among those with less than 68% of ideal body weight or BMIs less than 15.1. Other studies of the same patient population have identified hypophosphatemia in 27.5% of patients in the first week of nutritional rehabilitation. Adult studies have shown a 61 times greater chance of hypophosphatemia in malnourished patients. In addition, patients who had severe hypophosphatemia had an all-cause mortality of 18.2% compared with 4.6% among those with no hypophosphatemia ($P < .001$). In adults who require ICU level care, incidences of hypophosphatemia as high as 34% have been reported as well as increased rates of mechanical ventilation and length of stay.

Refeeding syndrome has been identified in cancer patients receiving nutrition support, with an incidence as high as 25%. The incidence of refeeding syndrome among patients admitted to the ICU for eating disorders is as high as 10%, with electrolyte disturbances cited as the most common manifestation. Byrnes and Stangenes identified multiple case reports illustrating the wide array of sequelae of refeeding syndrome, ranging from Wernicke-Korsakoff syndrome (acute/subacute) and acute edema/cutaneous distension syndrome to acute respiratory failure.

Perhaps the most important finding comes from the National Confidential Enquiry into Patient Outcome and Death of the United Kingdom, which reviewed records of 877 adult patients. Researchers found that only 50% of those later identified as at risk for refeeding syndrome were correctly identified. Other studies have also highlighted the underreporting and underrecognition of refeeding syndrome.

RISK FACTORS

Recognizing patients at risk for refeeding syndrome is perhaps the most important step for a clinician because it may prevent clinical sequelae. The general principles of reduced energy intake, poor absorption, and/or increased metabolic demands give rise to a variety of presentations and illnesses. Some specific patient risk factors involving these principles are listed in Table 1.

PATHOGENESIS

An understanding of the mechanism of starvation (Fig 1) and refeeding state (Fig 2) is helpful to recognize the biochemical

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<tr>
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<tbody>
<tr>
<td>CATEGORIES</td>
<td>RISK FACTORS</td>
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<tr>
<td>--------------------------------</td>
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</tr>
<tr>
<td>Psychiatric</td>
<td>Anorexia nervosa, depression, chronic alcohol and drug use</td>
</tr>
<tr>
<td>Chronic Malnutrition</td>
<td>Prolonged fasting (25 days), failure to thrive, children with complex health needs, oncologic patients, kwashiorkor/ marasmus</td>
</tr>
<tr>
<td>Renal/Endocrine</td>
<td>Diabetic hyperosmolar states, chronic diuretic use</td>
</tr>
<tr>
<td>Gastrointestinal Losses</td>
<td>Inflammatory bowel disease, chronic pancreatitis, short bowel syndrome, significant vomiting/diarrhea</td>
</tr>
<tr>
<td>Other</td>
<td>Child abuse and neglect/food insecurity/homelessness, acute weight loss of greater than 10% in 1-2 months, chronic infectious disease (AIDS, tuberculosis), cystic fibrosis, congenital heart disease, prolonged nil per os status</td>
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and clinical manifestations of refeeding syndrome. The most well-described manifestation of refeeding syndrome is hypophosphatemia, but hypokalemia, hypomagnesemia, and shifts in thiamine and trace elements can also occur (see the nutritional replacement portion of Fig 2). Fluid and glucose shifts are also important to monitor if refeeding syndrome is suspected.

Malnutrition/Starvation (Figs 1 and 2)

During periods of starvation, the liver transitions to gluconeogenesis, when fat and subsequently proteins are broken down. Salt and other electrolyte (notably phosphate, magnesium, potassium) shifts occur to accommodate fluid shifts and maintain homeostasis, although there is still total body depletion of these essential metabolites (which can explain why laboratory test results may be within normal ranges in the initial starvation phase).

Nutritional Replacement (Fig 2)

Refeeding results in hyperglycemia (due to lack of adequate insulin supply), leading to osmolar shifts, dehydration, and other common symptoms of elevated blood glucose. Carbohydrates become the primary source of energy. The liver transitions to anabolism, halts gluconeogenesis, and begins to produce glycogen and protein.

Refeeding Syndrome (Fig 2)

The process of refeeding potentiates an intracellular shift of electrolytes to accommodate cellular processes. This poses a serious problem because the starved individual already has experienced whole body depletion of these electrolytes. The serum electrolytes are further decreased as those electrolytes cross into the cells. Hypokalemia and hypomagnesemia are caused by potassium and magnesium shifting rapidly into cells as glucose and amino acids are taken up. The switch in energy source to carbohydrates and a large increase in adenosine triphosphate production cause hypophosphatemia from glucose phosphorylation. Thiamine deficiency occurs for several reasons: 1) low reservoir due to nutritional depletion before refeeding syndrome; 2) shift from fats to carbohydrate use for energy production via glycolysis, which increases thiamine requirements; and 3) an increased role as cofactor for the new anabolic state created during refeeding (to synthesize glycogen, fat, and protein). (20) Hypernatremia can also occur to maintain positive ion homeostasis, which can lead to a hypervolemic state and possible edema.
RECOGNIZING THE CLINICAL MANIFESTATIONS OF REFEEDING SYNDROME

The clinical manifestations of refeeding syndrome vary widely and are outlined in Table 2, listed by the corresponding underlying biochemical abnormality.

MONITORING AND MANAGEMENT OF REFEEDING SYNDROME

The level of evidence for the following refeeding syndrome monitoring and management recommendations is limited to cohort studies and expert opinion, and clinical scenarios and settings vary widely in each of the studies. Therefore, clinicians should adjust the recommendations according to individual patients and practice settings. To frame the following recommendations, most electrolyte disturbances occur in the first 2 to 3 days of initiating refeeding but can occur up to 7 to 10 days later. (9)

Refeeding

The general agreement among experts is that initial refeeding must be slow. Recommendations vary widely, but starting at 50% of estimated caloric needs is prudent according to existing evidence (recommendations vary from 20%-75%). (11) For the highest-risk patients, the general recommendation is to start at 25%. Dietary advancement should occur over 3 to 7 days, with caloric increases of 10% to 25% per day until the recommended caloric goals are achieved. (25)

Monitoring

The first step in proper monitoring and management of patients with refeeding syndrome is recognition of those at risk and prevention of clinical deterioration. Monitoring can occur in an inpatient, outpatient, or a combination of the 2 settings, according to the patient’s clinical and social needs. If a patient is identified as at risk through careful history and physical examination (including detailed nutritional history), monitoring should commence upon presentation and can include:

- Continuous cardiorespiratory monitor (if available) for concerning cases, full vital sign collection every 4 hours (adjust as needed).
- Daily detailed physical examinations, with focus on the neurologic and cardiac evaluation (perform more often if concerned).
- Strict intake and output monitoring with calorie counts.
- Daily weights (goal weight gain is undetermined in children but is 1 kg/wk for adults).
- Baseline and at least daily metabolic profile, with phosphorus, magnesium, potassium, sodium, glucose, and renal function measured. Monitor more frequently

<table>
<thead>
<tr>
<th>MECHANISM</th>
<th>CARDIAC</th>
<th>PULMONARY</th>
<th>MUSCULOSKELETAL</th>
<th>HEMATOLOGIC</th>
<th>GASTROINTESTINAL</th>
<th>NEUROLOGIC</th>
<th>OTHER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypophosphatemia*</td>
<td>Sudden death, arrhythmia, heart failure, hypotension, shock</td>
<td>Dyspnea, respiratory failure, impaired diaphragm function</td>
<td>Weakness, myalgia, rhabdomyolysis</td>
<td>Hemolysis, thrombocytopenia, leukocyte dysfunction</td>
<td>Confusion, delirium, paresthesias, paralysis, seizures, hallucinations, tetany, seizures, coma</td>
<td>Metabolic acidosis, insulin resistance, acute tubular necrosis</td>
<td></td>
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<tr>
<td>Hypokalemia</td>
<td>Anhythmia</td>
<td>Respiratory failure</td>
<td>Weakness, rhabdomyolysis, muscle necrosis</td>
<td>Nausea, vomiting, constipation</td>
<td>Paralysis</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>Hypomagnesemia</td>
<td>Anhythmia</td>
<td>Weakness</td>
<td>Nausea, vomiting, diarrhea</td>
<td>Tremor, tetany, seizures, altered mental status, coma</td>
<td>Refractory hypokalemia and hypocalcemia, death</td>
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<td></td>
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<tr>
<td>Vitamins/Thiamine Deficiency</td>
<td>Encephalopathy</td>
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<tr>
<td>Hyperglycemia</td>
<td>Hypotension</td>
<td>Respiratory failure</td>
<td>Weakness, rhabdomyolysis, muscle necrosis</td>
<td>Nausea, vomiting, constipation</td>
<td>Paralysis</td>
<td>Infection, death</td>
<td></td>
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<tr>
<td>Fluid Overload</td>
<td>Heart failure</td>
<td></td>
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<tr>
<td>Trace Element Deficiency</td>
<td>Anhythmia, heart failure</td>
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*Most commonly reported mechanism of refeeding syndrome.
if electrolyte replacement is needed and/or if there are concerning trends. If parenteral nutrition is being used, obtain initial and weekly hepatic enzymes (alanine aminotransferase, aspartate aminotransferase, total and direct bilirubin, alkaline phosphatase, albumin, total protein), cholesterol, coagulation profile, and triglycerides.

- Prealbumin, albumin, and zinc measurements can be considered, especially in those who have the highest risk profile for refeeding syndrome.
- Urinary electrolyte monitoring can be considered.

A multidisciplinary team of physicians, nurses, pharmacists, and dietitians is often needed to provide adequate care of children with potential and diagnosed refeeding syndrome. (20)(26)

Vitamin and Mineral Replacement

There is wide debate over whether thiamine should be administered before feeding. In general, available evidence suggests that clinicians can consider administering 100 to 300 mg/day oral thiamine (50-100 mg intravenously) before feeding because it is tolerated well with no daily upper limit. (20) Thiamine at this dose should be administered for a total of 3 days. Thiamine replacement should be increased for Wernicke encephalopathy and for confirmed thiamine deficiency, although these scenarios are rare. (20) The dose should be adjusted according to weight, and we suggest following the general recommendations of a practitioner’s available pharmacist/dietitian.

A multivitamin with or without iron (because the utility of initial iron therapy is yet unclear) should also be administered either orally or intravenously. The multivitamin should be administered as soon as a patient is recognized as being at risk for refeeding syndrome.

Fluid

Fluid overload is a common complication of refeeding syndrome and, therefore, fluids should be closely monitored with daily examinations and laboratory studies as outlined previously. However, data on how much fluid to provide in pediatric patients who are at risk for refeeding syndrome are lacking. To avoid fluid overload during necessary rehydration, we suggest administration of maintenance rates to achieve basic needs without causing fluid overload. Clinicians should pay attention to sodium intake, which should be restricted further if edema develops. (9)

Correcting Electrolyte Disturbances

Debate surrounds whether electrolyte disturbances detected at presentation should be addressed before refeeding, and there is no good evidence to suggest correction is better before or during feeding. Evidence from the National Institute for Health and Care Excellence group suggests that correcting electrolyte imbalances while starting feeding is the best method to achieve expedited homeostasis with little risk. (26) Table 3 outlines electrolyte replacement.

<table>
<thead>
<tr>
<th>ELECTROLYTE DISTURBANCE</th>
<th>SEVERITY</th>
<th>MAINTENANCE (PO)</th>
<th>TREATMENT</th>
<th>MAXIMUM DOSE</th>
<th>ADMINISTRATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypophosphatemia</td>
<td>Mild: 2.3-2.7 mg/dL (0.74-0.87 mmol/L) Moderate: 1.5-2.2 mg/dL (0.48-0.71 mmol/L) Severe: &lt;1.5 mg/dL (0.48 mmol/L)</td>
<td>0.3-0.6 mmol/kg per day</td>
<td>0.3-0.6 mmol/kg per day PO* 0.08-0.24 mmol/kg IV over 6-12 hours*</td>
<td>Single dose: 15 mmol/kg (IV) Daily: 1.5 mmol/kg (IV)</td>
<td>Over 6-12 hours, measure phosphate 2-4 hours after infusion completion</td>
</tr>
<tr>
<td>Hypomagnesemia</td>
<td>Mild/Moderate: 1.0-1.8 mEq/L (0.50-0.90 mmol/L) Severe: &lt;1.0 mEq/L (0.50 mmol/L)</td>
<td>0.2 mmol/kg per day</td>
<td>25-50 mg/kg per PO dose (0.2-0.4 mEq/kg per dose)*</td>
<td>Single dose: 2,000 mg (16 mEq) (oral)</td>
<td>Over 4 hours</td>
</tr>
<tr>
<td>Hypokalemia</td>
<td>Mild/Moderate: 2.5-3.4 mEq/L (2.5-3.4 mmol/L) Severe: &lt;2.5 mEq/L (2.5 mmol/L)</td>
<td>1-2 mmol/kg per day</td>
<td>0.3-0.5 mEq/kg per dose** (IV)</td>
<td>Single dose: 30 mEq/dose (IV)</td>
<td>Over at least 1 hour, measure potassium 2 hours after infusion completion</td>
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*50% less for impaired renal function.
**Urine output must be 0.5 mL/kg per hour or greater, continuous monitoring recommended while replacing potassium.

IV = intravenous; PO = oral.
Hypoglycemia and hyperglycemia have not been shown to cause substantial morbidity or mortality in this population. Accordingly, these conditions should be addressed as dictated by local practice patterns.

**ENTERAL VERSUS PARENTERAL FEEDING**

The advantages of enteral feeding are well documented, and this also holds true for malnourished patients. (26) If enteral feeding can be initiated within the caloric and nutritional recommendations stated previously (whether by mouth, nasogastric tube, or other postpyloric feeding method), it is recommended over parenteral feeding. The choice of administration route depends on the patient. A variety of enteral formulas is available to achieve the necessary caloric and nutritional needs, and this choice should be tailored according to the patient’s needs and formula availability.

Overall, enteral feedings should begin as soon as possible. Due to the higher infection risk and other complications associated with parenteral nutrition, (27) it should only be initiated if a patient is unable to tolerate the number of calories recommended by an enteral route. This may especially apply to patients with obstructive physiology or malabsorption and/or illness, which would preclude enteral feeding. Parenteral nutrition should be discontinued as soon as possible. The only definitive recommendations are based on a low-level quality of evidence from the adult literature and suggest that when patients can tolerate 60% of needed calories by an enteral route, parenteral nutrition may be discontinued, with no tapering necessary. (25)(29) Some pediatric practitioners may prefer to achieve 100% of needed calories before discontinuing parenteral nutrition in children, based on the poor quality of adult evidence suggesting otherwise. Evidence on the composition of parenteral nutrition is also conflicting, but general recommendations for malnourished patients that have previously been identified as safe include the following composition in relation to total calories: (24)(28)

- 20% protein
- 65% carbohydrates (dextrose)
- 15% fat

The clinician and care team must be diligent in calculating caloric, fluid, and nutritional needs as well as confirming that the composition of parenteral nutrition is appropriate.

**CASES REVISITED**

**Case #1:** A 3-month-old infant (birthweight 3.3 kg) is directly admitted to the hospital by his pediatrician because of failure to thrive. The family missed their 2-month appointment and has only had a single encounter with a pediatrician at postnatal day 5, when the infant had regained his birthweight. At that time, the patient’s weight was in the 38th percentile. He is now in the 3rd percentile for weight. By eliciting a further history (including detailed nutritional history) and performing a complete physical examination, clinicians discover that the infant received inadequate calories and that there are no suspected underlying medical conditions causing failure to thrive. Clinicians note decreased subcutaneous tissue on the buttocks and face. Initial metabolic profile that includes phosphorus of 4.8 mg/dL (1.55 mmol/L) (normal 4–6.5 mg/dL [1.20–2.10 mmol/L]) and magnesium of 2.1 mEq/L (1.05 mmol/L) (normal 1.6–2.4 mEq/L [0.80–1.20 mmol/L]) do not identify any concerning electrolyte abnormalities. (29)

- **Initial Management:** Order metabolic profile with magnesium and phosphorus
- **Inpatient Monitoring:** Initiate cardiorespiratory monitoring, obtain vital signs every 4 hours, conduct daily physical examinations, maintain strict intake and output with calorie counts, obtain daily weights (goal of weight gain is 20–30 g/day for infants), and obtain at least daily metabolic profile with phosphorus and magnesium while hospitalized.
- **Inpatient Feeding:** Administer 100 mg thiamine by mouth, and then initiate formula feeding at 50% estimated needs (in this case, approximately 50–60 kcal/kg per day for infants), slowly increasing by 25% each day. Administer multivitamin daily. The infant gained an average of 55 g/day over the course of the first 4 days of hospitalization. There were no concerning vital sign changes or electrolyte abnormalities. The infant was discharged to a safe, reliable environment. Follow-up weights obtained daily by the primary care physician for 3 days after discharge indicated appropriate weight gain. At his 6-month health supervision visit, the infant’s weight was at the 16th percentile.

**Case #2:** A 16-year-old girl with anorexia nervosa fails outpatient treatment and is referred for emergency department evaluation. Five months ago, the patient’s BMI was 18 and Z-score was -1.05, indicating mild malnutrition. Her current BMI is 14 and Z-score is -3.95, indicating severe malnutrition. Her weight-for-height Z-score has declined by 3, also indicating severe malnutrition. (1) On physical examination, she has pallor, temporal wasting, thinning hair, and a global decrease in subcutaneous fat tissue. She tearfully admits that she relapsed and had been “cheeking” her food to prevent ingestion. Initial metabolic profile documents her potassium at 2.5 mEq/L (2.5 mmol/L). There are no symptoms or signs of hypokalemia.

- **Initial Management:** Same as detailed in Case #1.
- **Inpatient Monitoring:** Same as detailed in Case #1.
- **Inpatient Feeding:** Administer 200 mg thiamine, then 0.3 mEq/kg potassium over 1 hour, with measurement 2 hours after administration. Insert nasogastric tube if necessary, initiate feeding at 50% estimated caloric
needs, and slowly increase by 25% each day. Administer multivitamin daily.

The adolescent was engaged in inpatient treatment for 2 weeks. She had borderline hypophosphatemia and hypomagnesemia, which was treated via slowing the increase in feedings. She did not require any additional electrolyte supplementation. After 2 weeks, she had gained 6 lb. With the assistance of mental health clinicians, she was discharged to a half-day inpatient treatment facility and her weight continued to improve.

Case #3: A 9-year-old boy with multiple medical problems who is tracheostomy- and gastrostomy tube-dependent is referred to the hospital because of a 9-lb weight loss in the last 2 months. His body weight before the loss was 71 lb. His parents are unsure how much nutrition he has been receiving. Further questioning reveals that the boy had relocated to new housing and had not been receiving consistent home health nursing for the last 2 months. The parents are unclear as to how much they are supposed to feed him via gastrostomy tube and he is now nil per os. The parents also report nonspecific intolerance of feedings for at least 1 month, leading them to administer water or rehydration fluids instead of formula. On physical examination, the patient’s respirations are somewhat labored, and the parents admit that he seems “out of it” when the examiner notes worsening mental status from the previous examination. Findings on history and physical examination raise no concerns for physical abuse.

Initial metabolic profile is notable only for hypoglycemia (34 mg/dL [1.89 mmol/L]) and mild hyponatremia (136 mEq/L [136 mmol/L]). Dextrose is administered through peripheral intravenous access in the emergency department according to institutional recommendations and his glucose increases to 126 mg/dL (6.99 mmol/L). His neurologic findings improve in the emergency department, with his parents noting that “he seems more himself.”

- **Initial Management:** Order metabolic profile with magnesium and phosphorus. Also order hepatic enzymes (alanine aminotransferase, aspartate aminotransferase, total and direct bilirubin, alkaline phosphatase, albumin, total protein), prealbumin, cholesterol, triglycerides, coagulation studies, electrocardiography, and complete blood cell count with differential count because of the high risk for refeeding syndrome and potential need for parenteral nutrition.
- **Inpatient Monitoring:** Initiate cardiorespiratory monitoring; obtain vital signs with neurologic examination every 2 hours until stable; complete daily full physical examinations; measure strict intake and output with calorie counts; obtain daily weights; and obtain daily metabolic profile with phosphorus and magnesium for 7 days (then 3 times in following week) and weekly hepatic enzymes, albumin, prealbumin, cholesterol, triglycerides, and coagulation studies.

- **Inpatient Feeding:** Administer 100 mg thiamine, place peripherally inserted central catheter line, start parenteral nutrition at 25% of estimated needs, and advance by 10% each day (very high risk so starting and moving slower). Start enteral feedings at 25% of estimated needs, increasing by 10% each day. Continue parenteral feedings until 60% of calories can be obtained by enteral route, then discontinue. Administer multivitamin daily.

On day 3 of hospitalization, the patient develops severe hypophosphatemia (1.3 mg/dL [0.42 mmol/L]), which is replaced with intravenous phosphate 0.2 mmol/kg. His physical examination findings do not change during this period. He is discharged on hospital day 8 with scheduled laboratory follow-up on full enteral feeding through the now functional gastrostomy tube. His formula is changed and is well tolerated.

His primary care physician has performed monthly weight checks, home nursing was confirmed in the hospital, and he continues to maintain good nutritional status.

**Summary**

- Refeeding syndrome, or the potential for refeeding syndrome, is often underrecognized in children.
- A host of underlying conditions and pathologies put children at risk for refeeding syndrome.
- It is of the utmost importance for clinicians to recognize at-risk patients to allow institution of proper monitoring and management.
- Despite the lack of quality evidence for treating patients with refeeding syndrome, the condition is difficult to study. Nonetheless, retrospective primary data along with an interdisciplinary team can aid clinicians in achieving optimal outcomes.

To view PowerPoint slides that accompany this article, visit http://pedsinreview.aappublications.org and click on the Supplemental tab for this article.

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This journal-based CME activity is available through Dec. 31, 2018, however, credit will be recorded in the year in which the learner completes the quiz.

1. A 6-month-old boy is brought to you for poor feeding. He is at the 75th percentile for height and at less than the 3rd percentile for weight. He has been living with his 18-year-old mother, who is his sole caregiver. She relates that he does not seem very interested in eating. On physical examination, the infant is thin and lethargic. You admit the infant to the hospital for intensive nutritional management. Which of the following laboratory findings is most likely to be seen in this infant as a complication of refeeding?
   A. Hyperkalemia.
   B. Hypermagnesemia.
   C. Hypernatremia.
   D. Hypocalcemia.
   E. Hypophosphatemia.

2. You are admitting a 13-year-old girl to the hospital with an 18-lb weight loss in the past month. You discuss a plan for hydration and fluid management with your ward team. You request daily laboratory monitoring. Over the first few days of hospitalization, you observe increasing glucose values. Which of the following is the most likely cause of this finding?
   A. Carbohydrate-based diet.
   B. Decreased insulin supply.
   C. Fluid restriction.
   D. Increased appetite.
   E. Overhydration.

3. A 2-year-old boy with severe malnutrition is admitted to the hospital for initial refeeding. You discuss vitamin supplementation with the pharmacist who works on the inpatient unit. While this patient will require multivitamin supplementation, deficiency of which of the following vitamins is most important to correct?
   A. Beta-carotene.
   B. Cobalamin.
   C. Folic acid.
   D. Phylloquinone.
   E. Thiamine.

4. You admit a 15-year-old girl to the hospital with a history of a 25-lb weight loss over the past 2 months for intense nutritional management. She has hair loss, fatigue, and constipation. Today in rounds with the ward team, she appears very swollen. You discuss with the team her care plan. Which of the following is the next best step in management of this patient?
   A. Aggressive diuresis.
   B. Compression stockings.
   C. Evaluation for syndrome of inappropriate secretion of antidiuretic hormone.
   D. Fluid management due to her high risk of volume overload.
   E. Limitation of dietary salt intake.

5. You are evaluating a 5-year-old boy who was admitted with severe malnutrition due to abuse and neglect. He was started on parenteral nutrition and is slowly gaining weight. He is starting to eat and is now taking about 70% of his calories by mouth. You discuss discontinuing parenteral nutrition with the boy’s new foster parents. His foster parents are adamant that he needs to continue receiving parenteral nutrition until he is at his expected weight. Which of the following is the most appropriate way to address the concerns of the new foster parents?
   A. Continue parenteral nutrition until he is at his expected weight.
   B. Explain that parenteral nutrition carries a concern for potential infection.
   C. Instruct them to limit his oral diet to carbohydrates.
   D. Prescribe probiotics to improve his overall nutrition.
   E. Suggest they seek out dietary supplements that are not present in the parenteral nutrition.
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