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Refeeding the Malnourished Patient: Lessons Learned



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Refeeding Syndrome (RS) was first recognized in the 1940s in starved prisoners of war who suffered complications after being refeed. Today, the problem has become more widely appreciated due to current advances in medical care and nutritional support. However, despite the increased recognition, no standard definition or treatment approach has been established by randomized clinical trials. Symptoms of RS can vary from a mild fall in serum electrolytes to critical electrolyte disarray and even death in the most severe cases. The goals of this article are to help clinicians better understand the mechanism of RS, recognize patients at risk, and identify the clinical circumstances that may require special attention.

CASES

Which of the following cases are refeeding? (Answers at the end)

For all cases, normal (UVA) reference ranges for electrolytes are as follows:

Phosphorus (Phos): 2.3 – 4.5 mg/dL (0.74-1.45 mmol/L)

Magnesium (Mg): 1.6 – 2.6 mg/dL (0.66-1.07 mmol/L)

Potassium (K⁺): 3.4 – 4.8 mEq/L (3.4 – 4.8mmol/L)

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Case #1

65 year old male admitted to the ICU with COPD exacerbation. Patient was well nourished prior to admission (just returned from a Caribbean cruise with his family). Now intubated and sedated. Enteral feeding initiated at a low rate within 24-48 hours of admission. Phosphorus level on hospital days 2 and 3, respectively: 1.7 mg/dL (0.55mmol/L) and 1.9 mg/dL (0.61mmol/L). Magnesium and potassium levels were within normal limits.

Case #2

65 year old female admitted with fever, UTI, and dehydration. History of hypertension and stroke four

(continued on page 58)

(continued from page 56)

months ago when she was discharged to a skilled facility on a pureed diet with thickened liquids. Her weight on discharge to the facility was 65 kg; at the time of this admission she was 56 kg. She failed a swallow evaluation and enteral feeding was initiated via nasogastric tube. Next morning labs revealed:

Phos: 1.8 mg/dL (0.58 mmol/L)

Mg: 1.4 mg/dL (0.58 mmol/L)

K+: 3.1 mEq/L (3.1 mmol/L)

Case #3

45 year old female admitted from the ER with diabetic ketoacidosis. Eating well until 2 days ago when she became ill from a virus and stopped taking food and medications. Current weight 62 kg; usual weight: 65 kg. Receiving IV fluids at 125mL/hr, an insulin drip with potassium replacement. On admission her potassium level was 5.8 mEq/dL (5.8 mmol/L), phosphorus level was 4.6 mg/dL (1.49 mmol/L), and magnesium level was 2.5 mg/dL (1.03 mmol/L). Phosphorus level now: 1.4 mg/dL (0.45 mmol/L).

INTRODUCTION

Refeeding syndrome (RS) is the metabolic response to nutrient provision in a malnourished patient. The driving force behind RS is the physiologic shift from a starved, catabolic state to a fed, anabolic state. Under normal conditions, the body's preferred fuel is carbohydrate. Carbohydrate is stored as glycogen in the liver for readily available energy. During starvation, glycogen stores are depleted, and the body responds by utilizing protein and lipid as the primary fuel source. This shift in fuel source results in decreased insulin levels and increased glucagon levels. Prolonged starvation will lead to decreased lean body mass as muscle is burned for energy. This results in decreased skeletal, cardiac, and respiratory muscle mass, as well as overall strength.

Prolonged periods without nutrition also result in total body loss of electrolytes (including phosphorus, magnesium, potassium), as well as vitamins and minerals. Serum electrolyte levels may not reflect total body stores as only about 1% of phosphorus and magnesium stores are reflected in the serum level.^{1,2} Serum electrolyte levels may remain normal despite overall depletion; this can be attributed to adaptation, intracellular contraction, decreased renal excretion, and/or dehydration.³⁻⁵

Table 1. Clinical Complications Associated with Refeeding Syndrome^{3,9,12,20}

- Cardiovascular Abnormalities
 - Arrhythmia, CHF, cardiomyopathy, cardiac arrest
- Respiratory Problems
 - Respiratory failure, diaphragmatic muscle weakness, failure to wean from mechanical ventilation
- Musculoskeletal
 - Rhabdomyolysis, muscle pain and cramps, weakness
- Neurologic
 - Confusion/delirium, Wernicke's encephalopathy, ataxia, tetany
- Hematologic
 - Anemia, thrombocytopenia, decreased oxygen delivery to tissues
- Coma
- Death

Insulin, in response to carbohydrate provision, is the primary stimulus for the cascade of events associated with RS. Insulin not only drives glucose into the cells, but also vitamins and electrolytes required for utilization of the substrate. This intracellular shift of electrolytes (and resulting decreased serum levels) account for many of the clinical complications associated with RS.

Signs and Symptoms

Symptoms of RS will vary from mild drops in serum electrolytes to severe electrolyte disorders with complications, or even death. Most symptoms will first occur between 1–3 days after refeeding is initiated,⁶ although in some cases up to 5 days.⁷ The duration of symptoms will vary based on the degree of malnutrition, feeding advancement and other factors. There is no standard definition for what defines RS or how many symptoms must be present to constitute RS. The majority of symptoms associated with RS are due to electrolyte dysregulation with cardiac, respiratory, neurologic and other systems affected (see Table 1). Cardiac arrhythmia is the most common cause of death from RS.³

Table 2. Non-Refeeding Causes of Hypophosphatemia^{7-9,12}

- Malabsorption
- Alcoholism
- Glucose administration
- Diabetic ketoacidosis
- Metabolic or respiratory alkalosis
- Initiating mechanical ventilation / correction of respiratory acidosis
- Volume repletion
- Vitamin D deficiency
- Renal Issues: rhabdomyolysis, hemodialysis, initiation of continuous renal replacement therapy, conditions resulting in renal tubular phosphate loss
- Infections: sepsis, gram-negative bacteremia
- Hyperparathyroidism
- Hyperaldosteronism
- Medications, including: diuretics, phosphate binders, insulin, beta-antagonists, epinephrine, antacids, sucralfate, glucagon, bicarbonate, corticosteroids, cisplatin, theophylline

Hypophosphatemia

Hypophosphatemia is the classic sign associated with RS. In fact, some authors have suggested that the term “Refeeding Hypophosphatemia” may be more appropriate for cases where hypophosphatemia is observed, and no other electrolyte disorders or symptoms of RS are present.⁷ In a review of 27 cases of RS, hypophosphatemia was documented in 96% of the cases.⁷

Phosphorus is required for a number of systems including the respiratory, neuromuscular, cardiac, endocrine, and hematologic systems.^{2,5,8} Phosphate is a component of adenosine triphosphate (ATP) and therefore is critical to providing energy to the cells. Phosphate is important in respiratory and cardiac muscle function, white blood cell function, nerve conduction, and oxygen delivery. Phosphorus is required for the pathway which allows for the release of oxygen from hemoglobin.⁹ Respiratory alkalosis or metabolic alkalosis can cause phosphorus redistribution, resulting in decreased serum phosphorus concentration.⁸ Hypophosphatemia has been shown to result in longer length of stay, longer ICU and ventilator days, and a higher mortality rate.^{10,11}

Hypomagnesemia and Hypokalemia

Other serum electrolyte abnormalities are associated

with RS, primarily magnesium and potassium. Magnesium is required for more than 300 enzyme pathways.¹ Among its many functions, it is important in the synthesis of proteins and is required for normal muscle, cardiac and nerve function. Hypomagnesemia is defined as serum Mg < 1.8 mg/dL (0.74 mmol/L), although symptoms most often occur with Mg < 1.2 mg/dL (0.5 mmol/L).¹ Hypomagnesemia can lead to muscle weakness, ventricular arrhythmia, neuromuscular problems, metabolic acidosis and anorexia.

Hypokalemia (serum potassium < 3.5 mEq/L [3.5mmol/L]) can lead to weakness, paralysis, and confusion. Severe hypokalemia can lead to life threatening arrhythmias, cardiac arrest or sudden death. Because of the severity of potential complications, hypokalemia is rarely left unattended by the medical team and is usually replaced promptly.

A full list of complications associated with hypophosphatemia, hypomagnesemia, and hypokalemia is available.¹²

Other Complications

Complications other than electrolyte disarray may also occur. Increased carbohydrate provision may decrease water and sodium excretion, resulting in fluid overload. This is most common in severely malnourished patients, such as those with anorexia nervosa. Hyperglycemia can

be seen as carbohydrate is provided to a body adapted to fat metabolism. Micronutrient deficiencies are likely if the patient has been without adequate nutrition for a prolonged period.

Thiamine should also be of primary concern in the patient at risk for RS. Depleted thiamine stores can lead to neurological compromise and other complications (Wernicke's encephalopathy). Thiamine supplementation should be provided to patients with a history of alcohol abuse, as well as patients who are markedly malnourished for any reason. In addition, until better data is available, thiamine is provided in our institution both before and for the first few days of feeding in these patients. Theoretically, if thiamine is given without concurrent nutrient delivery, there may not be "recruitment" (i.e. demand for thiamine), and it is unclear whether the thiamine would be utilized. Several recent reviews of thiamine and Wernicke's encephalopathy are available.¹³⁻¹⁵

Incidence

The true incidence of RS is difficult to determine, as there is not a standard definition for RS. The incidence reported in the literature varies greatly and is often based solely on the appearance of hypophosphatemia. Reported rates in specific populations include:

- 34% of all ICU patients¹⁰
- 10% in anorexic patients admitted to the ICU¹⁶
- 15% of hospitalized patients¹⁷
- 9.5% of patients hospitalized for malnutrition from gastrointestinal fistulae¹⁸
- 48% of severely malnourished patients being refeed¹⁹

This broad range in reported incidence is likely due to the wide variety of patient populations reported upon; varying degrees of malnutrition among the populations, different criteria used to diagnose malnutrition, different definitions of RS, and varied refeeding protocols among institutions.

Confounding the identification of RS is the fact that electrolyte disorders have many causes in the hospitalized setting. Therefore, it is important to remember that not all low electrolyte levels are a result of RS. Metabolic or respiratory acidosis, sepsis, volume repletion, changing renal function, initiation or stopping of insulin drips, or other factors may affect phosphorus levels. Many medications may lower serum

phosphorus as well. Patients, such as those with COPD, may experience hypophosphatemia when mechanical ventilation is initiated. This is due to the intracellular shift of phosphorus that occurs when pH normalizes as respiratory acidosis corrects.⁸ Table 2 lists some of the causes of hypophosphatemia in the hospitalized patient. The myriad of factors altering potassium and magnesium are outlined in an earlier article.¹²

Patients at Risk^{9,12,20}

Any patient who has been without adequate nutrition for a prolonged period of time may be at risk for RS. Critically ill patients may experience hypophosphatemia upon refeeding after a relatively short period of time (48 hours) without nutrition.⁹ Table 3 identifies some conditions that put patients at risk for RS. Interestingly, and likely to become more prevalent, is RS seen after severe weight loss from gastric bypass surgery.^{21,22} A recent study by Manning cites a low incidence of RS in alcoholics; however, it is important to note that these patients were not identified as malnourished, presented voluntarily, and were provided with an oral diet as desired.²³ Patients with chronic alcohol abuse should be presumed to have a component of malnutrition and be provided with thiamine (opinion of authors).

The National Institute for Health and Clinical Excellence (NICE) in England and Wales published guidelines in 2006 for identifying patients at high risk for RS.²⁴ While such screening tools may be helpful, it is often difficult to determine which patients will show signs and symptoms of RS. Zeki, et al. retrospectively reviewed the records of 321 hospitalized patients.¹⁷ The authors evaluated the risk for RS based on the NICE guidelines, and looked at serum phosphate levels before and after feeding initiation. Ninety-two patients (29%) were identified at risk of RS; of these, 23 patients (25%) developed refeeding hypophosphatemia (RH) compared with 26 patients (11%) who were not identified at risk, but still developed refeeding hypophosphatemia ($p=0.003$). This study demonstrates that not all patients identified at risk will show symptoms, and some patients not identified at risk will experience signs of refeeding. Other authors have also found that patients identified at risk do not always go on to develop RS.²⁵

RS can occur when consistent nutrients are provided regardless of source—oral, enteral, parenteral nutrition or IV dextrose. While in the past, overzealous PN was associated with RS, other reports have shown that RS

(continued on page 62)

(continued from page 60)

can occur when any source of nutrition is provided.^{7,17} In the Zeki article discussed above, the authors found that at-risk patients in the enteral group were more likely to develop hypophosphatemia than at-risk patients in the PN group.¹⁷ The authors postulate that lower levels of phosphorus seen in enteral feeding compared to PN may play a role, as well as increased stimulation of insulin secretion with enteral compared to PN due to first pass metabolism may be responsible.

Treatment

There is no one regimen that has been proven to prevent RS. Recently, one group undertook one of the first randomized, controlled trials to assess outcomes associated with a treatment regimen for RS in critically ill patients.²⁶ Patients who experienced hypophosphatemia upon feeding (<2 mg/dL [0.65mmol/L] within 72 hours of feeding) were randomized to either the control group vs. calorie restricted group. Patients in the control group (n=170) continued on nutrition support per standard protocol. Patients in the calorie restricted group (n=169) received 20 kcal/hour for 2 days, and then advanced to goal in a stepwise manner over several days. The results of this study were mixed. In the short term, the calorie restricted group had higher phosphorus levels on days 1 and 2, and less hyperglycemia on days 1–4. There was no difference in the primary study outcome of days alive after discharge from the ICU. This study provides some additional information; however, nutrition support was initiated at standard levels and the study group had calories decreased if hypophosphatemia occurred. This is different than the more standard practice of beginning nutrition support conservatively in patients at risk for RS and replace electrolytes as the need arises (see Table 4).

The NICE guidelines recommend initiating calories at 10 kcal/kg in patients at high risk for RS.²⁴ In the most severe cases, such as patients with anorexia nervosa, even lower levels may be recommended.^{16,24} Hofer, et al. evaluated 86 cases of severely malnourished anorexia nervosa patients (in 74.4% of cases, patients were less than 70% IBW).²⁷ The authors evaluated the use of a refeeding regimen which included initiation of feeding at 10 kcal/kg/day, fluid and sodium restriction, and electrolyte supplementation and monitoring (for full protocol see article). They found a low incidence of complications with this protocol, and no deaths were reported.

For patients deemed to be at mild to moderate risk of RS (or those patients where risk is unclear and clinicians just want to err on the safe side), such low calorie levels may not be necessary. Prior to the NICE guidelines, a calorie level of 15-20 kcals/kg was generally recommended for most patients at risk for RS.¹² and is currently used at our institution unless a patient is deemed to be at severe risk.

While repleting the malnourished patient is essential, repletion can only occur so quickly and a “rush” to refeed may lead to complications. On the other hand, once the refeeding calorie level has been established, there is no need to also initiate nutrition support below the designated refeeding calorie goal. For example: if the refeeding level is determined to be 1000 kcals/day, a continuous tube feeding rate for a 1 cal/mL product would be ~45mL/hr. We base our flow rates on 20-22 hours per day, as this is what is typically received.

When initiating nutrition support, all calorie sources should be taken into account such as: D5%, D10%, or calories coming from glucose or lipids in medication administration. These calories alone can cause RS in a malnourished patient. If it is not possible to stop any of these additional calorie sources, the nutrition support regimen should be adjusted to take these calories into account. Protein calories should always be included as part of the total calories.

Calories should be increased slowly as the refeeding risk subsides. An advancement of 200–300 calories every 1–3 days is generally recommended.^{28,29} It is important to make sure this advancement takes place so that patients are not left on hypocaloric feeding levels for a prolonged period of time. This is especially important if a patient is discharged home during the advancement period. It is also important to monitor whether patients are actually receiving and utilizing (see special circumstances discussed later), the nutrition prescribed during this period before further calorie advancement. Feeding interruptions, NPO status, hyperglycemia, malabsorption, or other issues may thwart efforts at nutrition support and leave patients still at risk for RS and further malnutrition.

Electrolyte Monitoring and Replacement

Electrolytes should be checked prior to initiation of nutrition support and low levels replaced. However, there is no need to withhold nutrition support until electrolyte levels are normal.^{20,24} Some guidelines

Table 3. Patients at Risk for Refeeding Syndrome^{3,9,12}

- ◆ Malabsorptive syndromes (including inflammatory bowel disease, short bowel syndrome, untreated celiac disease)
- ◆ Post bariatric surgery / morbidly obese with excessive weight loss
- ◆ Anorexia nervosa
- ◆ Chronic alcoholism or drug abuse
- ◆ Prolonged NPO status/ hospitalization/ post op periods
- ◆ Cancer
- ◆ Patients admitted from skilled nursing facilities
- ◆ Hunger strikers
- ◆ Hyperemesis gravidarum
- ◆ Chronic disease or infection (including HIV)
- ◆ Chronic vomiting / gastroparesis
- ◆ Dysphagia/esophageal dysmotility
- ◆ Psychiatric disorders, depression
- ◆ Hyperglycemia causing poor utilization of nutrients
- ◆ Resolving diabetic ketoacidosis (DKA)
- ◆ Patients deprived of nutrients for a prolonged period of time for any reason

recommend proactive supplementation of electrolytes, vitamins, and minerals.^{24,27} Thiamine should be provided to severely malnourished patients and those with a history of alcohol abuse or chronic vomiting prior to the initiation of nutrition support.

Serum electrolytes should be checked after 8 – 12 hours of nutrition support initially, then daily during the refeeding period (first 48-72 hours). The frequency and duration of electrolyte monitoring will vary depending on the degree of malnutrition and whether electrolyte disorders occur, as well as their severity.

Mild to moderate drops in electrolytes can be replaced orally/enterally in patients with a functioning GI tract. Severely low levels should be replaced intravenously. IV replacement may also be necessary

for patients without a functional GI tract, those who do not seem to be responding to enteral replacement, or in other situations where oral replacement is not possible or is contraindicated. Specific guidelines for phosphorus and magnesium replacement are available.¹²

Oral magnesium replacement may be poorly absorbed and can have a cathartic effect, causing or exacerbating diarrhea. As an example, some forms of magnesium (such as Magnesium Citrate and Magnesium Sulfate) are available over the counter as laxatives. Consider slower, more gradual dosing of oral magnesium (smaller doses over the day, or given at night on an empty stomach before bed), or forms of magnesium which provide more free magnesium per dose to the GI tract (such as magnesium oxide).

Intravenous magnesium is often given as a bolus over 60 minutes. This exceeds the renal threshold for magnesium and the kidneys will dump 50% or more of this dose.³⁰ Experience at our institution indicates a slower IV infusion of magnesium (over 10-12 hours) will be better utilized and retained. In this era of shortages and increasing healthcare costs, it is important to ensure the therapy being provided is being utilized.

Hypomagnesemia can exacerbate hypokalemia and make it more difficult to replace potassium. Potassium levels may not normalize until the corresponding hypomagnesemia is corrected. According to one report, 42% of patients with a low potassium level will also have a low magnesium level.³¹ Concurrent hypomagnesemia may also worsen the symptoms associated with hypokalemia. In addition, a recent review reports that patients with a history of alcohol abuse are often deficient in magnesium and discusses the role of magnesium in the treatment of Wernicke’s encephalopathy.¹⁴

SPECIAL CONSIDERATIONS

Renal Failure

Patients with renal failure are at high risk for malnutrition.^{32,33} However, due to the underlying disease state, these patients may have elevated serum electrolyte levels when feeding is first initiated. Serum levels may drop more gradually or over a longer period of time due to the “protective” effect of the renal failure. Therefore, in such patients, there may be a delayed response to refeeding. Electrolyte levels may need to be monitored over a longer period of time, and replacement may be

Table 4. Suggested Guidelines to Prevent Complications from Refeeding³⁶

1. Anticipate those patients at risk (see Table 3).
2. Initiate nutrition support, including total calories and fluids, slowly (~10 kcal/kg for severe cases; 15-20 kcal/kg for others).
 - ◆ Consider all sources of calories and fluids in your calculations.
3. Check baseline electrolytes (especially phosphorus, potassium and magnesium) before initiating nutrition support and replace any low levels promptly.
4. Monitor electrolytes every 8, 12 or 24 hours (depending on severity of refeeding risk).
 - ◆ Replace electrolytes as needed.
 - If enteral replacement, consider scheduled dosing if levels continue to be low, such as 1-2 packets neutra- or Kphos q 6, 8 or 12 hours until phosphorus consistently remains > 2.0mg/dl (0.65mmol/L).
 - ◆ Decrease blood draws as soon as electrolytes stabilize.
5. Routinely administer vitamins & minerals to malnourished patients, especially thiamine, for 3 – 5 days.
6. Unless hemodynamically unstable, keep sodium-containing IV fluids to ~ 1 liter/day initially in severely malnourished patients such as those with anorexia nervosa who may have a component of cardiomyopathy.
7. Increase calories cautiously in a stepwise manner.
 - ◆ By 200-300 calories every 2-3 days.
 - ◆ Continue to monitor electrolytes as calories are increased.
8. Outline a plan for nutrition advancement (especially if patient is to be discharged before goal calories reached) to prevent the patient from remaining on a refeeding calorie level longer than necessary.

needed after several days of nutrition support rather than in the first few days. Electrolyte replacement must be done carefully in patients with renal failure.

Hepatic Failure

Patients with severe end-stage liver disease will have depleted glycogen stores and may be unable to maintain serum glucose levels within a safe range.³⁴ This also may occur in patients with anorexia nervosa or severe malnutrition from any cause. In patients unable to maintain their serum glucose level in a safe range, dextrose infusion (D10%) may be required. In some, this infusion may provide calories that exceed the refeeding calorie goal. However, maintaining serum glucose levels above 80mg/dL (4.44mmol/L) trumps

any concern for RS. Electrolytes should be monitored closely and replaced as needed. Adequate thiamine, as well as other vitamins and minerals, should also be given during the first few days.

Hyperglycemia or Diabetic Ketoacidosis

If a patient presents with hyperglycemia, or becomes hyperglycemic after nutrition support is initiated, the refeeding process may be delayed as there is inadequate insulin to drive glucose and electrolytes into the cells. Hyperglycemia is essentially a continuation of the starved state (“starvation in the midst of plenty”). When insulin therapy is initiated, the refeeding response is accelerated. Clinicians should anticipate this response, and monitor for signs of RS. Note that if insulin therapy

is delayed and the patient is hyperglycemic for the first days of feeding, the signs of RS may be seen later, once insulin therapy is initiated and glycemic control is achieved.

Treatment for diabetic ketoacidosis is a form of refeeding.^{12,35,36} Exogenous insulin provided to treat diabetic ketoacidosis will power glucose and electrolytes into the cells causing electrolyte levels to drop and supplementation will be needed. Of note, exaggeration of hypocalcemia can be seen with aggressive phosphorus repletion (8.5mmol/hr, or 6 g inorganic phosphate); therefore caution is required.³⁷

GI Tract issues

Patients with gastrointestinal disease or malabsorptive disorders also may face unique challenges related to RS. Several scenarios can be seen.

First, it is not unusual for patients undergoing work-up on a GI service to frequently be made NPO for any number of reasons (procedures, GI bleed, access issues and symptoms). If the patient is to receive oral or enteral nutrition support, delivery may be inconsistent at best, and the total amount of nutrition provided may vary greatly from what is ordered. The amount of nutrition actually received needs to be determined in order to evaluate whether the patient has received enough nutrition for RS to occur. For example, a patient identified at refeeding risk may have an order to receive nutrition support on day #1, but be NPO off and on the next several days (while refeeding electrolytes are being monitored). If consistent nutrition actually starts several days later, RS may occur at that time. Ongoing monitoring should be coordinated with this timing. Also, clinicians may need to consider continuing thiamine supplementation, as the demand for thiamine occurs when patients are fed, not while they are NPO. It is unknown if patients will just excrete supplemental thiamine in the non-fed state, so until better data is available, it may be prudent to continue dosing until feeding is consistent for 3–5 days (opinion of the authors).

In patients with possible malabsorption, RS will not occur if the nutrition delivered is not absorbed (unless an IV source of nutrition is provided). For example, if a malnourished patient with suspected malabsorption shows signs of RS when enteral nutrition is provided, at least some absorption is occurring. This is certainly a ‘gross’ test at best, but it does provide some clues to the level of absorption. Patients with malabsorption may receive enteral nutrition for a period of time, but at some point require the initiation of parenteral nutrition due to failure to thrive, change in status, etc. Patients should be monitored for RS at this time also (even though they have been “fed” for some time).

BACK TO CASES

Case #1

Unlikely. Patient well-nourished prior to admission. However, mechanical ventilation and correction of respiratory acidosis in a patient with COPD can lead to significant hypophosphatemia (8).

Case #2

Most likely RS. Multiple electrolyte disorders in a patient with significant recent weight loss.

Case #3

Possibly. Resolution of DKA will mimic RS due to the potent effects of insulin driving electrolytes intracellular. Although this patient may be refeeding after experiencing excess loss of potassium, magnesium and phosphorus in the urine due to the catabolic effects of DKA, the exogenous insulin provided will accelerate serum drops as glucose and electrolytes move intracellularly.

SUMMARY

RS is a concern for any patient who has been without consistent or adequate nutrition for a prolonged period of time. Serious complications can be avoided with appropriate identification of patients at risk, slow initiation of feeding, and careful monitoring. An understanding of the causes and mechanisms of RS can aid the clinician in better caring for patients, as well as recognizing when special circumstances arise or additional care and monitoring may be needed. ■

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