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Much Ado About Refeeding


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Refeeding syndrome is a series of metabolic events precipitated by the provision of nutrients, primarily carbohydrate, to a patient in a nutritionally compromised state. Refeeding syndrome is associated with hypophosphatemia, hypokalemia, and hypomagnesemia; fluid retention and micronutrient deficiencies, including thiamin, are also of concern. If severe, refeeding syndrome may result in respiratory, cardiac, and neuromuscular dysfunction, especially in the stressed, elderly, or severely malnourished patient. This article will discuss the pathophysiology behind refeeding syndrome, identify patients at greatest risk, and provide practical tips for prevention and treatment.

CASE STUDY

MH is a 73-year-old nursing home resident. She has a history of cerebrovascular accident approximately four months prior to admission resulting in dysphagia; she has been on a pureed diet with thickened liquids since that time. Her medical history includes hyper - tension, atrial fibrillation and asthma. MH is admitted to the hospital with fever, aspiration pneumonia, and dehydration. She is made NPO; IV fluids are started (D_5, ½NS). A modified barium swallow demonstrates aspiration with all consistencies. Nasogastric feedings are initiated according to standard protocol. A weight obtained the following day is 56 kg; her weight at the time of her stroke four months earlier was 65 kg. The following day, MH developed worsening respiratory distress and was transferred to the ICU for further care. The results of her blood chemistries were: phos - phorous—1.1, magnesium—1.3, and potassium—2.9.

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INTRODUCTION

Historically, problems such as cardiac dysfunction, edema, and neurological changes were observed when malnourished refugees were suddenly provided with adequate food. Such problems have also been observed when relief efforts provide food to victims of famine. A 1940's study examined the effects of starvation and refeeding in a group of previously healthy volunteers (1). After a period of severe food restriction, some subjects in the study experienced cardiac compromise when normal intake was resumed. The advent of nutrition support has brought renewed attention to what has become known as refeeding syndrome, especially in light of two deaths attributed to this syndrome (2). An increased awareness and understanding of refeeding syndrome may help prevent further such incidents as clinicians learn to recognize patients at risk and avoid over-aggressive nutrition support regimens.

INCIDENCE OF REFEEDING SYNDROME

The incidence of refeeding syndrome is unknown. In studies of patients on TPN, the incidence of hypophosphatemia ranged from 30%–38% when phosphorous was provided in the solution, to 100% when PN without phosphorous was administered (3). In cancer patients it has been reported as high as 25% (4). It is more common in the elderly, although it may be masked by co-morbid conditions (5). There are no randomized, controlled trials looking at the incidence of refeeding syndrome itself. Mild symptoms often go unnoticed or may be attributed to other causes. Conversely, low serum levels of phosphorous, potassium and magnesium thought to be due to refeeding syndrome, may be due to other factors including decreased dietary intake, ineffective gastrointestinal absorption, increased renal and non-renal losses and transcellular shifts (Table 1).

PATHOPHYSIOLOGY

Under normal conditions, glucose is the body's preferred fuel. Glucose is primarily derived from carbohydrates in the diet. The body depends on a consistent intake of carbohydrate for continued glucose provision. Glucose is available in the blood stream for fuel for two to three hours after ingestion and can be stored as glycogen, approximately 300 grams of glucose are stored as glycogen in the liver and muscles. In the absence of oral intake, glycogen can provide up to 24 hours of energy. A minimum of 100–150 grams of glucose per day is needed to provide adequate glucose to the brain and to prevent protein breakdown for glucose production (6). When adequate glucose is available, protein is used for non-fuel functions, such as building muscle and the synthesis of enzymes, hormones, antibodies, and transport proteins. Carbohydrate, protein or fat intake in excess of needs is stored as fat. Fat is the primary storage fuel in the human body.

During a short period of fasting (up to 24 hours), obligatory glucose is obtained through glycogenolysis. When glycogen stores are depleted, amino acids are mobilized from muscle and fatty acids are mobilized from adipose tissue; both are degraded further to provide glucose and glycerol respectively for gluconeogenesis. Glucose is also produced via gluconeogenesis from lactate and pyruvate. The initial period of starvation is marked by increased protein catabolism. As nutrient deprivation is prolonged, the rate of proteolysis decreases as the body further adapts to the decreased nutrient intake, basal metabolic rate slows by as much as 20%–25% (7). Peripheral tissues and most organs can adapt to using fatty acids as a fuel source. The brain, as a primary end user of glucose, switches over to ketones as a partial fuel source and decreases its reliance on glucose as a substrate. These adaptive mechanisms, in the non-stressed state, decrease the need for glucose, thereby sparing muscle mass.

Prolonged starvation leads to loss of lean body mass, adipose tissue and fluid. Eventually, starvation also effects the visceral protein mass and the function of vital organs. Respiratory function may decline due to respiratory muscle wasting which may limit carbon dioxide excretion (8), cardiac mass and output are also decreased as well. Of note, in the absence of additional stressors, such as illness or injury, serum protein levels are maintained at relatively normal levels during pure protein-calorie malnutrition. A severely malnourished patient may have a normal albumin level despite prolonged starvation. This is due, in part, to decreased breakdown of serum proteins and to extravasation of

Table 1
Potential Cause of Hypokalemia, Hypophosphatemia, and Hypomagnesemia (8,21,30,31,32,33,34)

Hypophosphatemia

General	*Metabolic*	*Medications*
Malabsorption	Hypomagnesemia	Diuretics
Glucose administration	Hypokalemia	Beta-agonists
Vitamin D deficiency	Hypocalcemia	Epinephrine
Volume repletion	Systemic alkalosis	Insulin
Rhabdomyolysis	Hyperparathyroidism	Antacids
Hemodialysis	Hyperaldosteronism	Carafate
Initiation of CVVHD		Glucagon
Diabetic ketoacidosis		Bicarbonate
Alcoholism		Corticosteroids
Sepsis		Cisplatin
Gram-negative bacteremia		Theophylline
Refeeding Syndrome		

Hypokalemia

General	*Metabolic*	*Medications*
Vomiting / Diarrhea	Hypomagnesemia	Albuterol
Insulin delivery	Metabolic alkalosis	Diuretics
Refeeding Syndrome		Insulin
		Carbenicillin
		Terbutaline
		Ticarcillin

Hypomagnesemia

General	*Metabolic*	*Medications*
Diarrhea	Diabetes Mellitus	Amphotericin B
Malabsorption		Cisplatin
Alcoholism		Cyclosporin
Refeeding Syndrome		Ticarcillin
		Carbenicillin
		Digoxin
		Alcohol
		Gentamycin
		Insulin
		Diuretics

fluid into the interstitial space that may make serum levels appear falsely elevated. In fact, it is not uncommon for a severely malnourished patient to initially lose weight after nutrition support is started due to diuresis of this additional extracellular fluid.

Decreased nutrient intake and poor nutritional status may lead to depleted vitamin and mineral status, including depletion of phosphorous, magnesium and potassium. Magnesium depletion may be further exacerbated if there are gastrointestinal losses or excess urine losses

Table 2
Complications Associated with Refeeding Syndrome (1,6,8)

Hypophosphatemia	*Hypokalemia*	*Hypomagnesemia*
Respiratory	**Cardiac Dysfunction**	**Cardiac Abnormalities**
• Failure	• Arrhythmia	• Arrhythmias
• Ventilator dependency	• Cardiac arrest	• Tachycardia
Cardiac	• EKG changes	**Neurologic**
• Arrhythmia	• Digoxin toxicity	• Altered Mental Status
• Congestive heart failure	**Neurologic**	• Weakness
• Cardiomyopathy	• Weakness	• Tetany
• Decreased cardiac contractility	• Paralysis	• Paresthesia
• Hypotension	• Rhabdomyolysis	• Seizures
Skeletal	• Lethargy/confusion	• Ataxia
• Rhabdomyolysis	**Metabolic**	• Vertigo
• Weakness	• Metabolic alkalosis	• Tremors
Neurologic	**Gastrointestinal**	**Gastrointestinal Problems**
• Altered mental status	• Paralytic ileus	• Abdominal pain
• Paralysis	• Constipation	• Diarrhea
• Seizures		• Constipation
Endocrine		• Anorexia
• Insulin resistance		**Electrolyte**
• Osteomalacia		• Hypokalemia
Hematologic		• Hypocalcemia
• Leukocyte dysfunction		**Hematologic**
• Altered RBC morphology		• Anemia
• Thrombocytopenia		
• Decreased platelet function		
• Hemolytic anemia		
• Decreased oxygen release from oxyhemoglobin		

in the setting of hyperglycemia. This decrease in total body minerals, especially those required by glycolysis (phosphorus, potassium, magnesium) may be masked as renal excretion soon adjusts and normal serum electrolyte levels are often maintained. Other adaptive mechanisms such as increased bone mobilization of phosphorous also occur, especially if acidosis is present. Unlike glucose metabolism, fatty acid oxidation does not require phosphorous intermediates, hence phosphorous requirements are decreased during starvation.

There are a number of consequences that occur when nutrition, namely carbohydrate, is provided to a starved patient. The driving factor for the refeeding sequelae is insulin secretion. Insulin promotes glucose uptake, along with phosphorous and other electrolytes into the cells. This intracellular shift, along with an already depleted electrolyte pool, can lead to dangerously low levels of serum phosphorous, potassium and magnesium (but not necessarily all three). Serious respiratory, cardiac and neurological complications may occur (Table 2); in the most severe cases, these complications may be fatal. Refeeding syndrome is often considered a complication of PN, however, it can also occur after the initiation of enteral nutrition, dextrose containing IV fluids, oral intake, or any source of calories. Although the pathophysiology of refeeding syndrome is known, it is unclear why some starved patients are asymptomatic with the exception of mild decreases in serum electrolyte levels, while others exhibit severe consequences, even death (2). The elderly, chronically ill, and chronically starved patient

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may be at highest risk for complications from refeeding syndrome, possibly due to decreased cardiac, respiratory and muscle reserve.

In addition to intracellular trapping and decreased total body stores, other factors contribute to the decreased serum electrolyte levels and clinical symptoms observed with refeeding syndrome. The hypophosphatemia seen after a patient is refed is exacerbated by an increased need for phosphorylated intermediates (such as ATP). One of the potential consequences of hypophosphatemia may be decreased oxygen delivery to the cells (ischemia) as hypophosphatemia leads to decreased levels of 2,3 diphosphoglyceride (2,3 DPG) consequently impairing oxygen release from hemoglobin (9,10). Increased demand for potassium during repletion may further aggravate hypokalemia associated with refeeding syndrome. Adequate magnesium is essential for normalizing phosphorus and potassium levels and is also required for over 300 metabolic pathways, including those involving ATP production. Therefore, hypophosphatemia and hypokalemia associated with refeeding may be exacerbated by an underlying hypomagnesemia.

Insulin secretion associated with sudden carbohydrate infusion also leads to sodium and fluid retention. This is thought to be due to insulin's effect on the renal tubules leading to antinatriuresis. This antidiuretic effect causes an increase in extracellular fluid volume. Therefore, excessive sodium containing fluid infusion can lead to edema or, in severe cases, the combination of increased extracellular fluid volume and the cardiomyopathy often present in severely malnourished individuals can lead to pulmonary edema, respiratory compromise and cardiac failure.

THIAMIN

While not a hallmark of refeeding syndrome, thiamin deficiency can still be of significant importance to the malnourished patient at risk for refeeding. Thiamin, (B_1) is a water-soluble vitamin with a biological half-life of approximately 9½ to 18½ days (11). In patients with suboptimal intake, it is estimated that deficiency can occur in less than 28 days (12).

Thiamin is the precursor for the active metabolite thiamin pyrophosphate (TPP), essential for optimal glucose utilization and metabolism. TPP is a co-factor for three critical enzymes, in particular, pyruvate dehydrogenase. In thiamin deficiency, the conversion of pyruvate to acetyl coenzyme-A (CoA) is blocked and the accumulating pyruvate is subsequently converted to lactate. This results in an overproduction of lactate and lactic acidosis follows. Inadequate levels of thiamin can also precipitate Wernicke's Encephalopathy (WE) in malnourished patients. Glucose load, whether delivered enterally or parenterally, will increase the metabolic demand for thiamin.

There is a lack of evidenced-based literature concerning an appropriate repletion dose of thiamin. Literature in support of thiamin repletion for alcoholics at risk for (or diagnosed with) WE endorse varying dosages and routes. Doses cited range from 50 mg intramuscularly daily (13), 100 mg IV BID (14), to a 24-hour infusion of 1 gm (15). The 2004 guidelines of the British Association of Pharmacology recommend a minimum of 500 mg IV TID for a minimum of two days, followed by 500 mg IV daily for five days for patients with suspected or diagnosed WE (16). Of note, the active form of thiamin requires adequate levels of magnesium (11).

Thiamin depletion may be exacerbated by dialysis, diuretics, diarrhea, alcohol, folate deficiency, or malabsorption (17,18,19). Although there is a dearth of literature concerning thiamin deficiency and refeeding syndrome, any malnourished patient at risk for refeeding syndrome should receive thiamin supplementation until better evidence is available. It is also noteworthy to recall the reports of lactic acidosis in patients receiving thiamin deficient TPN during a nationwide shortage of IV multivitamins in 1989 (14).

"ACCELERATED REFEEDING"

Metabolic events, very similar to refeeding syndrome, occur during the treatment of diabetic ketoacidosis (DKA) or hyperosmolar, hyperglycemic, nonketotic syndrome (HHNS) (9,20). Patients with diabetes mellitus (DM) are at higher risk for symptoms and complications of refeeding for multiple reasons. Uncontrolled DM is, by definition, a catabolic state mimicking starvation (albeit in the "midst of plenty" with hyperglycemia running rampant). Patients with uncontrolled DM may

Table 3
Patients at Risk for Refeeding Syndrome (32,35,36,37)

- Anorexia nervosa
- Chronic alcoholism
- Oncology patients
- Post-operative patients
- Residents admitted from skilled nursing facilities
- Depression in the elderly
- Uncontrolled diabetes mellitus (diabetic ketoacidosis)
- Chronic malnutrition:
  - Marasmus
  - Kwashiorkor
  - Prolonged hypocaloric feeding
  - Morbid obesity with profound weight loss
  - Prolonged fasting (including patients with non-nutritional IV fluids)
  - High-stress patient not fed for >7 days
  - Hunger strikers
  - Victims of famine

experience anorexia, poor nutritional intake and vomiting—resulting in depleted nutritional status. In addition, patients are especially at risk for depleted potassium stores due to loss of lean body mass, the potassium-losing effect of aldosterone, osmotic diuresis, and depletion of glycogen stores. These changes may be more dramatic in the setting of HHNS as this condition occurs over a longer period of time. In certain settings, such as dehydration and metabolic acidosis, potassium levels may actually be elevated, thus clinicians may not expect the impending hypokalemia of refeeding that can occur once the patient is treated with insulin and IV fluid. It should also be noted that in patients with refractory hypokalemia, hypomagnesemia might be the cause (21).

In the treatment of hyperglycemia, it is *exogenous* insulin that drives the electrolyte shifts discussed earlier. The profound and efficient consequence of insulin on lowering potassium levels has been well documented (22). This appears to be due to insulin's effect on Na-K pump activity and correction of metabolic acidosis. It is often necessary to replace potassium during the treatment of DKA; however, studies have also shown that up to 90% of patients treated for uncontrolled DM are also hypophosphatemic and hypomagnesemic after 12 hours of treatment (9). Magnesium depletion in the setting of DM is caused primarily by renal wasting due to osmotic diuresis, induced by glucosuria. Metabolic acidosis, reduction in bone magnesium levels and cellular depletion from insulin deficiency also contribute to magnesium wasting (21,23).

PATIENTS AT RISK

The first step in preventing refeeding syndrome is to identify patients at risk (Table 3). A thorough nutritional assessment is needed including an assessment of recent weight change over time, recent nutritional intake and other factors, such as social issues or alcohol history. Serum protein levels often remain within normal limits in the marasmic patient. Low albumin levels likely indicate the degree of stress or illness rather than nutritional depletion. Finally, there is never a hurry to replete a patient. The metabolic machinery can only work so fast, therefore, if nutritional history is unclear, start with a conservative level of nutrition.

TREATMENT

At the sight of a severely malnourished patient, the clinician should avoid the temptation to initiate excessive amounts of nutrition support ("hyperalimentation"); rather, begin nutrition repletion slowly. While it is appropriate to start nutrition intervention as soon as feasible, it is dangerous to provide excessive calories or fluids to a patient whose body has adapted to starvation. Guidelines found in the literature for the initiation of calories for the patient at risk of refeeding are shown in Table 4. The practice at our institution uses the calories per kilogram method and initiates total calories at 20 kcal/kg in patients at risk for refeeding (possibly 15 kcal/kg in the most severely malnourished patients). A mixed fuel source of protein, fat and car-

Table 4
Various Methods for Initiating Nutrition Support in Malnourished Patients

- 20 kcals/kg (1)
- No more than 20% above BEE (7)
- 50% of total needs based on HBE $\times$ 1.3 – 1.5 (24)
- 1000 kcals/day (1)

Table 5
Suggested Guidelines for Phosphorus Replacement in Adults (38,39,40)

Intravenous

Serum PO_4 Level (mg/dl)	*Recent and Uncomplicated (mmol/kg of phosphate)*	*Prolonged or Complicated (mmol/kg of phosphate)*
1.6–2.1	0.8–0.16	0.16–0.24
1.1–1.5	0.16–0.24	0.24–0.30
<1.1	0.24–0.30	0.50

Oral Repletion
- Serum phosphate >1 mg/dl):
  - 1–2 g (32–64 mmol) of phosphorous/day
  - Divided in 3–4 doses

bohydrate should be provided. Glucose infusion should not exceed 150–200 grams per day or 2 mg/kg/minute initially (7). Protein can generally be initiated at full goal (up to 1.5 g/kg/day) (7). For estimating nutritional needs, the patient's actual weight (or adjusted weight if obese) should be used rather than an ideal weight, which may over (or under-) estimate needs (6). When calculating calorie needs for patients at risk for refeeding syndrome, consider any additional sources of calories such as Propofol (Diprivan®) or dextrose in IV fluids, medications, or dialysate solutions.

Table 6
Suggestions for Magnesium Replacement (41)*

Condition	*Magnesium Dose*
Daily Requirement	0.4 mEq/kg/day
Mild to Moderate hypomagnesemia (Serum level: 1.2–1.7 mg/dl)	Initial: 1 mEq/kg over 24 hours Follow: 0.5 mEq/kg over 24 hours × 5 days
Severe hypomagnesemia (Serum level: <1.2 mg/dl)	Initial: 2 g over 2 minutes Follow: 5 g over 6 hours Follow: 5 g over 12 hours × 5 days

*Note: Enteral magnesium is poorly absorbed and may aggravate stool (and hence magnesium) losses.

FLUID AND SODIUM

Fluid and sodium also need to be initiated slowly in the chronically starved patient. Fluid intake may need to be restricted; several authors recommend restricting initial net fluid intake to 800 mL/day in severely malnourished patients (6,24). Excessive sodium intake should also be avoided. Care should be taken as to which sodium concentration is used based on the goal of treatment—volume repletion versus hydration (NS vs ¼ – ½ NS). Rapid weight gain or weight gain of more than one to two pounds per week is likely fluid retention.

ELECTROLYTES

Electrolyte levels, especially potassium, magnesium, and phosphorous, should be monitored at baseline and replaced as needed. Electrolyte levels should be checked every six hours, twelve hours, or daily for approximately three days after initiation of nutrition (although some authors recommend up to one week) (6). If severe electrolyte abnormalities occur, further monitoring may be necessary. Prompt correction of any electrolyte deficiencies can prevent related complications. See Tables 5 and 6 for suggested guidelines to replace phosphorous and magnesium respectively. Repletion guidelines for potassium are available elsewhere (25).

In patients who demonstrate significant refeeding, especially those with gastrointestinal complaints, intravenous (IV) repletion may be the most effective route, at least initially. This is particularly true for patients who have diarrhea, as enteral electrolyte replacement may exacerbate this complication. In this type of patient, we often use a "refeeding cocktail," a custom IV fluid that provides consistent replacement of potassium, magnesium and/or phosphorus (dosage based on individual need) in a volume of fluid appropriate for the particular patient versus an IV piggy back given over one to two hours. In addition to

providing more effective replacement, it also saves nursing time from repeated IV piggy back (IVPB) dosing. With respect to magnesium, fifty percent (50%) of magnesium given as an IVPB will be eliminated by the kidney as rapid infusions exceed the renal threshold and rapid excretion follows. A greater percentage of magnesium will be retained when a continuous IV solution is provided (26–29) (and will allow for much smaller doses—personal experience of the editor).

Once electrolyte levels have stabilized, nutrition can be advanced to goal repletion levels in a stepwise manner. Based on our clinical experience, advancing by 200–300 calories every three to four days is generally well tolerated. However, the exact advancement will depend on individual patient response to the initial regimen and whether hyperglycemic episodes "delay" the refeeding response. If severe refeeding symptoms occur, advancement should be slower and electrolyte levels repleted before further advancement takes place. If no significant refeeding occurred with initiation of nutrition, advancement to goal can likely be accelerated. Continued monitoring and follow up is important to ensure that goal repletion levels are met. Without such monitoring and advancement, a patient might be left on a refeeding level of nutrition longer than necessary, delaying improvement of the overall nutritional status.

CLINICAL CONSIDERATIONS

Patients Discharged on Nutrition Support

Whenever possible, patients should be monitored for symptoms of refeeding in the hospital setting and electrolytes replaced prior to discharge. However, in this day and age, this is not always possible. In certain cases, it may be prudent to purposely advance nutrition support to induce refeeding in the safe setting of the hospital rather than at home; the process may even be started in some patients by adding D_5 to IV fluids. Efforts should be made to ensure that patients receive a significant amount of nutrition during the refeeding monitoring period. Take for instance, the patient who had tube feedings ordered for three days prior to discharge, but is NPO for procedures for most of that time. In such a situation, refeeding could likely occur after discharge when full nutrition delivery is actually achieved. There is no such thing as a "stat lab" in home care. If it is necessary to monitor a patient for refeeding in the home setting, specific plans should be made. Determine who will monitor the patient, the labs, and how the nutrition support should be advanced. It is not uncommon for a patient to be discharged on refeeding levels and still be on that regimen weeks later because no advancement plan was communicated to the home care providers.

Other Factors Associated with Electrolyte Shifts

Disease state and other individual factors also play a role in the clinical manifestation of refeeding syndrome. Degree of stress, glucose control, gastrointestinal and renal function may affect the timing and severity of refeeding syndrome. For example, critically ill patients have increased needs for protein as well as other nutrients and electrolytes. These increased needs, combined with the challenges of feeding the critically ill patient, can quickly lead to nutritional deficiencies or further diminish already depleted nutritional stores. Critical illness may also impair the body's normal adaptation to fasting, leading to continued high rates of catabolism. In one study, 34% of critically ill patients experienced hypophosphatemia when feedings were initiated after a period of as little as 48 hours without nutrition (30).

Hyperglycemia

As previously discussed, the primary trigger for refeeding syndrome is insulin secretion. Therefore, initiation of nutrition support without adequate glucose control may not elicit the full refeeding sequelae. The catabolic state will not be reversed while blood glucose levels remain out of control. This situation is counter-productive to improving nutritional status and may delay refeeding syndrome and complicate monitoring parameters. In such cases, refeeding may occur once exogenous insulin is provided and glucose levels are brought under control rather than during the first few days of feeding.

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Table 7
Summary Guidelines to Prevent Complications of the Refeeding Syndrome

1. Anticipate patients at risk for refeeding syndrome.
2. Check baseline electrolytes before initiating nutrition support and replace any low levels promptly—however, do not withhold nutrition support until serum levels are corrected, rather replete electrolytes concurrently with the nutrition support provided.
3. Initiate nutrition support, including total calories and fluids, slowly—this does not mean that the enteral or parenteral nutrition has to *progress* slowly to meet the "refeeding level" that has been predetermined.
   Example: If a refeeding level of 20 kcal/kg is appropriate (which equates to a continuous tube feeding rate of 45 mL/hour of a 1 kcal/ml product), there is no need to ***also*** start EN slower than this, as the amount of refeeding calories the patient is to receive in 24 hours has already been accounted for.
4. Consider additional sources of calories, such as dextrose in IV fluids, glucose or lipid calories from medications, etc. and include these in total calories.
5. Unless hemodynamically unstable, keep sodium-containing fluids to ~1 liter/day initially.
6. Monitor electrolytes daily for at least 3 days and replace any low levels as needed. Be wary of the malnourished patient in renal failure with elevated serum electrolytes secondary to decreased clearance, as they may be a "late refeeder."
7. Be prepared for accelerated refeeding and the need for aggressive electrolyte replacement in the hyperglycemic patient while glucose control is pursued.
8. Routinely administer vitamins to malnourished patients, especially thiamin; consider a "loading dose" prior to initiation of nutrition support.
9. Increase calories cautiously in a stepwise manner. Continue to monitor electrolytes as calories are increased.
10. Outline a plan for nutrition advancement (especially if patient is to be discharged) to prevent the patient from remaining on refeeding levels longer than necessary, thereby delaying improvements in nutritional status over time.

Used with permission from the University of Virginia Health System Nutrition Support Traineeship Syllabus (42)

Indirect "Gross" Gastrointestinal Absorption Test

In patients with questionable GI absorption, if refeeding syndrome is observed after initiation of enteral nutrition, at least partial nutrient absorption can be presumed (assuming no other medication or IV source of nutrients has been given). Conversely, if there is questionable absorption and enteral feeding does not induce refeeding, it cannot be assumed the patient will not refeed if parenteral nutrition is started. As mentioned earlier, the optimal route of electrolyte replacement should be carefully considered in the patient with gastrointestinal issues as enteral replacement may exacerbate diarrhea or other problems.

Renal Failure

Renal function may also affect both the presentation and treatment of refeeding syndrome. Electrolyte levels may initially be normal to high, but decrease over time, reaching a low level several days after initiation of nutrition ("late refeeding") (20). Renal function must, of course, also be considered when correcting serum electrolytes. Conservative electrolyte replacement and consultation with the renal team may be necessary for patients with renal impairment.

SUMMARY

In many cases, nutrition support is a lifesaving modality. However, if nutrition is not delivered properly, refeeding the malnourished patient can lead to serious complications or even death. Table 7 summarizes recommendations to avoid complications associated with refeeding syndrome due to overzealous nutrition support. Prospective, randomized controlled trials are needed to better identify patients prone to complications from refeeding syndrome. This, in turn, will help determine the best approach to refeeding the malnourished patient. ■

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