Refeeding Syndrome

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KEYWORDS

• Refeeding syndrome • Pediatric • Hypophosphatemia
• Nutrition support • Malnutrition

Refeeding syndrome (RFS) is a term that describes the metabolic and clinical changes that occur on aggressive nutritional rehabilitation of a malnourished patient. It is a well-described yet often underrecognized entity. Its recognition was heightened in the World War II era when prisoners who had undergone starvation developed cardiac failure and peripheral edema on nutritional replenishment.[1] In Leningrad and The Netherlands, cases of cardiac insufficiency and edema were reported after refeeding survivors of the war who were starved because of scant food supplies.[2] In 1944, Keys and colleagues deliberately starved and refed previously healthy men and observed cardiac decompensation in some patients who were orally fed.[2,3] In the 1960s, the advent of parenteral nutrition (PN) allowed for a more aggressive means of nutritional rehabilitation. Reports of hypophosphatemic hyperalimentation syndrome soon followed in the 1970s. In 1980, Silvis and colleagues[4] noted paresthesias, seizures, or coma in conjunction with hypophosphatemia in patients receiving PN. In the 1980s, Weinsier and Krumdieck[5] wrote a critical paper that described cardiopulmonary complications resulting in the death of two chronically undernourished patients who received PN.

Hypophosphatemia is the hallmark of RFS. Other electrolyte abnormalities are associated with RFS, however, such as hypokalemia and hypomagnesemia. Shifts in glucose, sodium, and fluid balance are also seen in RFS. Consequently, cardiovascular, pulmonary, neuromuscular, hematologic, and gastrointestinal complications occur. This syndrome can emerge with aggressive oral nutrition, enteral nutrition, or PN and can be fatal if not recognized and treated in a timely manner.

PATHOPHYSIOLOGY OF STARVATION

During starvation, the body tries to compensate for the lack of energy by means of changes in metabolism and hormone regulation. The body goes into a state of

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catabolism. A shift from carbohydrate metabolism to fat and protein catabolism occurs, which provides glucose and ketones for energy. This shift to protein catabolism results in a loss of lean body mass, which affects major organs, such as the heart, lungs, intestines, liver, and kidneys. Atrophy of the myocardium results in poor contractility and diminished cardiac output. Liver wasting results in decreased protein synthesis and further alteration in metabolism. Gastrointestinal atrophy causes malabsorption and dysmotility, further exacerbating the malnourished state, and increases the risk for infection. The kidneys also lose their ability to concentrate urine, resulting in diuresis.[6–8]

Cellular mass is also lost, contributing to functional loss of vital organs. Intracellular loss of electrolytes, including potassium, magnesium, and phosphate, occurs as a consequence of this change in metabolism. Insulin secretion decreases and the basal metabolic rate slows down to 20% to 25% to conserve energy.[8] Consequently, the body becomes bradycardic, hypothermic, and hypotensive. Growth and thyroid hormones also decrease.[6] These changes occur in an effort to conserve protein and organ function, which aids in survival. It is important to understand the pathophysiology of starvation and the metabolic shifts that occur, especially when one is about to feed a patient in this state.

PATHOPHYSIOLOGY OF REFEEDING SYNDROME

Once nutrition is reintroduced to a patient who has been starved for an extended period, anabolism begins instantaneously. The body shifts back to carbohydrate metabolism from protein and fat catabolism, and glucose becomes the primary source of energy once again. The increased glucose load, with a corresponding increase in the release of insulin, leads to cellular uptake of glucose, potassium, magnesium, and phosphate. This shift of electrolytes back into the cell causes hypokalemia, hypomagnesemia, and hypophosphatemia. Insulin also exhibits a natriuretic effect on the kidneys. Hence, sodium is retained, causing fluid retention and expansion of the extracellular fluid volume.[6–8]

Anabolism's high requirements may unveil or cause further deficiencies, which may lead to life-threatening circumstances. Rapid correction of undernutrition may cause fluid shifts and intravascular volume overload, which may precipitate congestive heart failure in an undernourished patient with myocardial atrophy.

DETERMINING PATIENTS AT RISK FOR REFEEDING SYNDROME

A critical step in preventing RFS is identifying patients who are at risk before initiating nutrition. These include patients with anorexia nervosa, chronic malnutrition, marasmus, or kwashiorkor; patients underfed or fasting for at least 10 to 14 days; prolonged fasting; prolonged intravenous hydration; and morbid obesity with massive weight loss (**Box 1**). There are several studies and case reports that describe refeeding hypophosphatemia and its consequences in patients with these conditions. Reports continue to emerge in the current literature documenting refeeding complications, notably in patients who have anorexia nervosa. Solomon and Kirby[9] commented that anorexia nervosa serves as a sobering model for the possible calamity inherent in refeeding severely malnourished hospitalized patients. Patients who weigh less than 80% of their ideal body weight or have recent weight loss of 5% to 10% in the past 1 to 2 months are also at risk. A study by Dunn and colleagues[10] showed that one of the most frequent identifiers for a pediatric patient at risk for RFS was a calculated body weight less than 80% of ideal body weight. Additional case reports have documented refeeding hypophosphatemia in children with less than 80% of ideal

Box 1
Patients at risk for refeeding syndrome

- Anorexia nervosa
- Less than 80% of ideal body weight
- Patients underfed or not fed for at least 10 to 14 days (including patients receiving prolonged intravenous fluids without adequate calories or protein)
- Acute weight loss of greater than 10% in the past 1 to 2 months (including obese patients with extensive weight loss in a short period)
- Kwashiorkor
- Marasmus
- Chronic conditions causing malnutrition (uncontrolled diabetes mellitus, cancer, congenital heart disease, and chronic liver disease)
- Malabsorptive syndromes (including inflammatory bowel disease, cystic fibrosis, chronic pancreatitis, and short bowel syndrome)
- Cerebral palsy and other conditions causing dysphagia
- Children of neglect
- Postoperative patients, including after bariatric surgery

Data from Refs.[4,6,7,12]

body weight. Worley and colleagues[11] describe refeeding hypophosphatemia in children with cerebral palsy and those who are abused and neglected. One recent study had cancer and congenital heart disease as the leading medical diagnoses in those found to have RFS.[10]

In the adult population, patients at risk include those admitted from nursing facilities; those with a history of excessive alcohol intake; and those with chronic diseases causing undernutrition, such as chronic obstructive pulmonary disease and cancer.[8]

INCIDENCE OF REFEEDING SYNDROME

In the past 40 years, the incidence of RFS in adults remains significant. Up to 50% of hospitalized patients are documented to be malnourished.[3] In a study of patients receiving total parenteral nutrition (TPN), the incidence of hypophosphatemia in patients receiving phosphorus ranged from 30% to 38%, and for patients receiving TPN without phosphorus, the incidence was 100%.[8] RFS is seen in up to 25% of adult patients who have cancer.[7,8] The true incidence of RFS in pediatrics in unknown, perhaps as a result of underrecognition or lack of reporting.

CLINICAL MANIFESTATIONS OF REFEEDING SYNDROME

Clinical manifestations of RFS are a direct result of the electrolyte and hormonal changes that occur as the basal metabolic rate rapidly increases. The patient may manifest signs and symptoms of hypophosphatemia, hypokalemia, hypomagnesemia, hyperglycemia, fluid overload, or thiamine deficiency (**Table 1**).

Hypophosphatemia

The one feature that characterizes RFS is hypophosphatemia. During the fasting state, catabolism results in depletion of intracellular phosphate. Rapid introduction of

Table 1
Clinical signs and symptoms of refeeding syndrome

Hypophosphatemia	Hypokalemia	Hypomagnesemia	Vitamin/Thiamine Deficiency	Sodium Retention	Hyperglycemia
Cardiac	Cardiac	Cardiac	Encephalopathy	Fluid overload	Cardiac
Hypotension	Arrhythmias	Arrhythmias	Lactic acidosis	Pulmonary edema	Hypotension
Decreased stroke volume	Respiratory	Neurologic	Death	Cardiac compromise	Respiratory
Respiratory	Failure	Weakness			Hypercapnea
Impaired diaphragm contractility	Neurologic	Tremor			Failure
Dyspnea	Weakness	Tetany			Other
Respiratory failure	Paralysis	Seizures			Ketoacidosis
Neurologic	Gastrointestinal	Altered mental status			Coma
Paresthesia	Nausea	Coma			Dehydration
Weakness	Vomiting	Gastrointestinal			Impaired immune function
Confusion	Constipation	Nausea			
Disorientation	Muscular	Vomiting			
Lethargy	Rhabdomyolysis	Diarrhea			
Areflexic paralysis	Muscle necrosis	Other			
Seizures	Other	Refractory hypokalemia and hypocalcemia			
Coma	Death	Death			
Hematologic					
Leukocyte dysfunction					
Hemolysis					
Thrombocytopenia					
Other					
Death					

Data from Kraft MD, Btaiche IF, Sacks GS. Review of RFS. Nutr Clin Pract 2005;20:625–33.

carbohydrate into the body inhibits fat metabolism and favors glucose metabolism, which causes an insulin surge, promoting cellular uptake and use of glucose and phosphate. The combination of depletion of total body phosphorus stores during catabolic starvation and increased cellular influx of phosphorus during anabolic refeeding leads to severe extracellular (serum) hypophosphatemia.[9] Additionally, when glucose once again becomes the predominant fuel source, a high demand occurs for the production of phosphorylated intermediates of glycolysis (ie, red blood cell ATP, 2-3-diphosphoglycerate [DPG]).[13] Low serum phosphorus levels are directly related to the depletion of ATP and DPG.[9] ATP and DPG are crucial to all processes in the body that depend on energy. Hypophosphatemia can impair neuromuscular function, and the patient may exhibit weakness, impaired muscular contractility, paresthesia, cramps, and seizures. Respiratory muscles can be affected, causing poor ventilatory function and respiratory compromise. Severe hypophosphatemia is also associated with rhabdomyolysis, hemolysis, thrombocytopenia, and leukocyte dysfunction. Psychologic changes include confusion, altered mental status, and coma.[11,13,14] As reported in several cases, even small decreases in serum phosphorus during this delicate state can result in large-scale dysfunction and affect virtually all systems in the body.[15] Refeeding hypophosphatemia can occur within 24 to 72 hours of introducing nutrition.[10] Several papers note the nadir of phosphorus to occur within the first week of refeeding.[12,13]

Hypokalemia

Potassium, which is the major intracellular ion, is also depleted during the catabolic state of starvation. With refeeding, increased insulin secretion leads to cellular uptake of potassium, resulting in hypokalemia. The electrochemical membrane potential is imbalanced, causing arrhythmias and cardiac arrest. Neuromuscular dysfunction, such as weakness, paresthesia, rhabdomyolysis, and respiratory failure, can occur.[10,13]

Hypomagnesemia

The physiology of hypomagnesemia is similar to that of phosphorus and potassium. Magnesium affects membrane potentials, and its imbalance presents in similar ways as hypokalemia. It is also important for the structural integrity of DNA, RNA, and ribosomes and serves as a cofactor for enzymes involved in ATP production and oxidative phosphorylation. Thus, the demand for magnesium increases as the metabolic rate increases. Hypomagnesemia manifests neurologically with weakness, tremors, tetany, seizures, and altered mental status; with cardiac arrhythmias; and with gastrointestinal symptoms, such as nausea, vomiting, and diarrhea. Low magnesium levels may also induce hypokalemia because of impaired Na^+/K^+-ATPase activity. Magnesium is required for parathyroid function, and low levels may induce hypocalcemia.[6,13,15]

Sodium Retention

Sodium retention is also seen in RFS. The infusion of carbohydrates leads to increased insulin secretion. Insulin causes decreased renal excretion of sodium and water. Patients may then develop fluid overload, pulmonary edema, and congestive cardiac failure. Low serum albumin may also contribute to edema during refeeding as a result of low oncotic pressure.[7,10,13,15]

Vitamin Deficiency: Thiamine

Vitamin deficiencies also occur because of inadequate intake. Deficiency in thiamine (vitamin B_1) has important consequences during refeeding. It is an important cofactor for enzymes needed in carbohydrate metabolism, and it is rapidly consumed in glycolysis during refeeding. Deficiency can occur in less than 28 days, because its half-life is 9.5 to 18.5 days.[8] Low levels of thiamine impair glucose metabolism and result in lactic acidosis. Further, thiamine deficiency may result in Wernicke's encephalopathy or Korsakoff's syndrome. Wernicke's encephalopathy is manifested by ataxia, confusion, hypothermia, ocular abnormalities, and coma. Korsakoff's syndrome is associated with amnesia and confabulation.[13–15] Of note, adequate magnesium levels are required for the active form of thiamine.[8]

Hyperglycemia

Administration of glucose in excess amounts leads to hyperglycemia; a high glucose load stops gluconeogenesis and leads to decreased use of amino acids. Thus, the body's ability to metabolize glucose is decreased. Further, a starved patient who is refed is experiencing a stress response that increases circulation of glucocorticoids, exacerbating hyperglycemia.[7] Elevated serum glucose may lead to osmotic diuresis, dehydration, hypotension, metabolic acidosis, and ketoacidosis. Another sequela is lipogenesis, attributable to insulin stimulation, which results in fatty liver, increased carbon dioxide production, hypercapnia, and respiratory failure.[15] Complications of hyperglycemia also include impaired immune function and increased risk for infection. Prolonged hyperglycemia may result in hyperosmolar, hyperglycemic, nonketotic coma.[2,7]

MANAGEMENT OF REFEEDING SYNDROME

There are several recommendations in initiating nutritional support. Regardless of the strategy, a gradual introduction of feeds is recommended. Proposed ranges for starting feeds include 25% to 75% of resting energy expenditure.[2,3,6–8,10,13–15] In adults, reports recommend starting at 20 kcal/kg/d or 1000 kcal/d.[2,8] In pediatric and adult patients, calorie intake is increased 10% to 25% per day or over 4 to 7 days until the calorie goal is met.[10] Advancement of nutrition is based on biochemical stability. The saying "start low, and go slow" serves as a good guideline in approaching a malnourished patient.

Protein is not restricted during nutritional support. Several studies show that high protein intake spares lean muscle mass and helps in its restoration.[3]

Sodium and fluids should be restricted during the initial period of refeeding to prevent fluid overload, especially in a patient at risk for RFS, whose cardiac function may be compromised. Palesty and Dudrick[3] recommend restricting sodium to 20 mEq/d and total fluid to 1000 mL/d or less.

Electrolyte deficiencies should be corrected before starting enteral or parenteral support (**Tables 2–4**). Of note, the National Institute for Health and Clinical Excellence 2006 guidelines in England and Wales state that correction of fluid and electrolyte abnormalities need not be done before refeeding and can be done while refeeding.[15] Most other investigators advocate correcting electrolyte imbalances before feeding a patient, however. Further, in light of reports of persistent biochemical imbalances despite conservative measures, correcting these abnormalities is prudent before initiating feeds. Miller[2] pointed out that refeeding hypophosphatemia can still occur despite cautious introduction of carbohydrate. Dunn and colleagues[10] also demonstrated similar findings in pediatric patients who showed electrolyte imbalances

Table 2
Guidelines for phosphate replacement

	Intravenous Replacement Dose (Administer Over 6–12 Hours)
Children	0.08–0.24 mmol/kg Maximum single dose: 15 mmol Maximum daily dose: 1.5 mmol/kg
Adults	Initial dose: 0.08 mmol/kg if recent uncomplicated or mild hypophosphatemia[a] 0.16 mmol/kg if prolonged or severe hypophosphatemia[b] Increase dose by 25%–50% if persistent hypophosphatemia Maximum dose: 0.24 mmol/kg per dose

Oral absorption may be unreliable, and the oral form may cause diarrhea.
Serum phosphate should be obtained 2 to 4 hours after completion of infusion.
Patients with impaired renal function should start at 50% or less of the initial dose.[13]
[a] Mild hypophosphatemia: 2.3 to 2.7 mg/dL, moderate hypophosphatemia: 1.5 to 2.2 mg/dL.
[b] Severe hypophosphatemia: less than 1.5 mg/dL. This level of hypophosphatemia can lead to severe neurologic, cardiac, respiratory, and hematologic abnormalities and possibly to death.[13]
Data from Huang T, Wo S, et al. 2007–2009 Housestaff manual Lucile Packard Children's Hospital at Stanford. 8th edition. Hudson (OH): Lexi-Comp, Inc; p. 564–5.

despite using conservative guidelines for nutritional support. Depletion of phosphate in severely malnourished patients is the likely culprit, and higher requirements may be needed. Supplements for potassium and magnesium may be required as well. Sodium and fluid should be limited, however, because there is an inclination to retain these during the initial feeding period. Daily measurement of electrolytes is recommended until stability is met. Weekly prealbumin and albumin levels have also been suggested. Supplementation with multivitamins and thiamine is advisable (**Table 5**).

Close monitoring can help to avoid or minimize the complications of RFS. A patient should be placed on a cardiorespiratory monitor during the initial phase of nutritional support. A patient's neuromuscular and mental status should be assessed on a regular basis. Fluid intake and output should also be carefully measured to avoid fluid overload and its potential sequelae to the cardiorespiratory system. Checking daily weight also ensures proper fluid balance; the goal of weight gain should be no more than 1 kg/wk. Any weight gain greater than this is likely attributable to fluid retention.

Table 3
Guidelines for magnesium replacement

	Intravenous Replacement Dose (Administer Over 4 Hours)
Children	25–50 mg/kg per dose (0.2–0.4 mEq/kg per dose) Maximum single dose: 2000 mg (16 mEq)
Adults	1 g every 6 hours for four doses for mild-moderate hypomagnesemia 8–12 g/d in divided doses for severe hypomagnesemia

Replacement is in the form of magnesium sulfate.
Patients with impaired renal function should start at 50% or less of the initial dose.
Mild hypomagnesemia: 1.5 to 1.8 mg/dL.
Moderate hypomagnesemia: 1 to 1.5 mg/dL.
Severe hypomagnesemia: less than 1 mg/dL.[13]
Data from Huang T, Wo S, et al. 2007–2009 Housestaff manual Lucile Packard Children's Hospital at Stanford. 8th edition. Hudson (OH): Lexi-Comp, Inc; p. 562–4.

Table 4
Guidelines for potassium replacement

	Intravenous Replacement Dose (Administer Over at Least 1 Hour)
Children	0.3–0.5 mEq/kg per dose Maximum dose: 30 mEq per dose
Adults	0.3–0.5 mEq/kg per dose Maximum dose: 30 mEq per dose

Serum potassium should be obtained within 2 hours of completion of infusion.
Ensure that urine output is greater than 0.5 mL/kg/h and that patient is on a cardiac monitor.
Mild to moderate hypokalemia: 2.5 to 3.4 mEq/L.
Severe hypokalemia: less than 2.5 mEq/L or if symptomatic.[13]
Data from Huang T, Wo S, et al. 2007–2009 Housestaff manual Lucile Packard Children's Hospital at Stanford. 8th edition. Hudson (OH): Lexi-Comp, Inc; p. 562–4.

Should the signs and symptoms of RFS emerge, nutritional support should be stopped immediately. Electrolyte abnormalities should be corrected without delay, and supportive measures should be administered, such as the administration of intravenous thiamine for encephalopathy, vasopressors for hypotension, oxygen for respiratory distress, and diuretics for fluid overload. Once these conditions are addressed, nutrition can be restarted. Previous publications recommend restarting nutrition at 50% or less of the previous rate that led to the development of symptoms.[3,13] Health care providers should examine the patient frequently for signs and symptoms of refeeding phenomena. Electrolyte abnormalities usually occur within the first days of initiating feeds, cardiac complications occur within the first week, and altered mental status typically occurs thereafter.[12]

Table 5
Guidelines for thiamine replacement

	Beriberi or Thiamine Deficiency	Deficiency After Bariatric Surgery	Wernicke's Encephalopathy (use Intravenous form)	Dietary Supplement
Children	10–25 mg/d administered IV or IM if extremely ill or 10–50 mg per dose administered PO every day for 2 weeks and then 5–10 mg/d for 1 month	(adolescents) 50 mg/d		0.5–1 mg/d
Adults	5–30 mg per dose 3 times per day, administered IV or IM if extremely ill, and then 5–30 mg/d administered PO for 1 month		Initial:100 mg IV followed by 50–100 mg/d until regular diet resumes	1–2 mg/d

Abbreviations: IM, intramuscular; IV, intravenous; PO, per os.
Data from Huang T, Wo S, et al. 2007–2009 Housestaff manual Lucile Packard Children's Hospital at Stanford. 8th edition. Hudson (OH); Lexi-Comp, Inc; p. 661.

Caring for a malnourished patient requires a well-coordinated multidisciplinary team for optimal results. Nutrition support teams may not be available in some institutions, however. Therefore, raising and maintaining awareness of the dangers of aggressively feeding a starved patient is imperative. Further, it is essential to review the patient's actual nutritional intake (which includes additional glucose, sodium, and fluids in medications) and to compare it with the intended nutritional orders for the patient. Discrepancies between the actual and intended nutritional intake may put the patient at risk, as shown in a study conducted by Dunn and colleagues.[10]

In facing a malnourished patient, there is an instinctive drive for health care providers to re-establish nutrition as rapidly as possible. Starvation develops over time as the body attempts to adapt to the lack of adequate calories and does not occur over a period of hours. Therefore, treatment should not be executed with haste. The pivotal article by Weinsier and Krumdieck[5] opened our eyes to the dangers of aggressive refeeding, and many subsequent papers have continued to echo the same message. Yet, the incidence of refeeding remains significant.[3,7,16] Improved awareness and understanding of RFS, along with a well-coordinated plan of care, are vital in delivering safe and effective nutritional rehabilitation.

SUMMARY

RFS is the result of aggressive enteral or parenteral feeding in a malnourished patient. Hypophosphatemia is the hallmark of RFS. Other metabolic abnormalities, such as hypokalemia and hypomagnesemia, may also occur, along with sodium and fluid retention. Refeeding should be started at a low caloric requirement and advanced slowly only in the setting of metabolic stability. Important in management is close monitoring of signs and symptoms of RFS, in addition to monitoring electrolyte levels while providing electrolyte replacement and vitamin supplementation as needed. Identifying patients at risk for RFS is key to prevention, and awareness of the potential complications involved in reintroducing feeds to an undernourished patient is crucial.

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