Refeeding Syndrome or Refeeding Hypophosphatemia: A Systematic Review of Cases

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Refeeding Syndrome or Refeeding Hypophosphatemia: A Systematic Review of Cases

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Abstract

Nutrition support clinicians refer to the abnormalities in laboratory data and changes in clinical signs and symptoms that follow refeeding of starved or malnourished patients as *refeeding syndrome*. Theoretical descriptions of refeeding syndrome include a complex and extensive list of changes, such as hypophosphatemia, hypomagnesemia, hypokalemia, hyponatremia, hypocalcemia, hyperglycemia, and vitamin deficiency—all of which are accompanied by clinical signs and symptoms. In practice, clinicians see asymptomatic refeeding hypophosphatemia more often than a full-blown syndrome with multiple laboratory and clinical abnormalities. Confusion results because there is no widely accepted or uniformly applied set of defining characteristics for diagnosing refeeding syndrome. To gain insight into the clinical characteristics of refeeding syndrome described in the literature, a systematic review of reported cases and case series was conducted. Since 2000, 20 authors described 27 cases that contained sufficient data for review. Hypophosphatemia occurred in 26 patients (96%). While 19 patients (71%) experienced at least 1 other laboratory abnormality, only 14 (51%) exhibited a consistent pattern of abnormally low phosphorus and magnesium levels. Seven patients had hypocalcemia (26%), and hyponatremia was reported in 3 patients (11%). There were no reports of hyperglycemia. Mean data reported in case series containing data from 63 patients showed that hypophosphatemia was a consistent finding but that other abnormalities were not consistently identified. Findings suggest that refeeding hypophosphatemia is not accompanied by a consistent pattern of biochemical or clinical abnormalities among case reports or case series of patients reported to have refeeding syndrome. (*Nutr Clin Pract.* 2012;27:34-40)

Keywords

refeeding syndrome; hypophosphatemia; complications; nutrition support; malnutrition; starvation

In 1990, Solomon and Kirby refocused the attention of the nutrition support community on the consequences of nutritional repletion in severely malnourished patients. In their review of the topic, they proposed an expanded definition of *refeeding syndrome*, which included "the metabolic and physiologic consequences of the depletion, repletion, compartmental shifts and interrelationships of phosphorus, potassium, magnesium, glucose metabolism, vitamin deficiency and fluid resuscitation."[1] Since then, authors have expanded the definition of refeeding syndrome to include secondary complications or all adverse events occurring during nutrition rehabilitation of malnourished patients.[2,3] These definitions incorporate a broad, theoretical approach to the metabolic basis of refeeding syndrome, which is reviewed in detail elsewhere.[4,5] Briefly, refeeding syndrome is believed to result when oral, enteral, or parenteral nutrients, primarily carbohydrate, fluid, and sodium, are administered to starved or malnourished patients in amounts greater than a weakened cardiopulmonary system can accommodate. The excess fluid and sodium intake results in volume expansion and fluid retention, which precipitate heart failure.[6] Carbohydrate administration increases the demand for intracellular phosphorus to synthesize adenosine triphosphate, which results in acute phosphorus depletion and ensuing neuromuscular, cardiovascular, and respiratory compromise. In addition, carbohydrate metabolism may increase the demand for magnesium and thiamine, which may also result in neuromuscular complications. Arrhythmias induced by hypokalemia, tetany associated with hypocalcemia, and hyperglycemia related to carbohydrate excess are also mentioned.[7]

While refeeding syndrome is usually presented as a single entity, in practice, patients present a variety of responses to feeding initiation. Some patients experience an asymptomatic decline in phosphorus, and others have multiple laboratory abnormalities, some of which are accompanied by moderate to severe symptoms. Thus, broad definitions are not always easy

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to measure clinically and are subject to wide interpretation. Specific criteria that are easy to apply may result in more widespread recognition and earlier treatment of complications associated with refeeding.

One defining characteristic of refeeding syndrome is that it appears within the first 2 to 5 days after feedings begin. Another component of refeeding syndrome is that it occurs among chronically and severely malnourished famine victims or anorectics. More recently, it has been described in patients starved for as little as 48 hours before feeding.[8] Since biochemical data are readily available, low or declining levels of phosphorus, magnesium, potassium, sodium, and calcium, as well as hyperglycemia and vitamin deficiency that occur after feeding, are often used as surrogate markers for clinical signs and symptoms. However, there is inconsistency among clinicians concerning the specific serum levels of nutrients that constitute refeeding syndrome.[9-11] While the word *syndrome* suggests multiple anomalies, there is no agreement on the number of abnormalities required to diagnose refeeding syndrome or whether an abnormal level of a single nutrient such as phosphorus is sufficient.[3,9,12,13] Individual clinicians use locally or personally defined criteria to diagnose refeeding syndrome; however, a more widely agreed-upon definition of refeeding syndrome could standardize diagnosis and provide a basis for developing and validating management protocols. As one step toward a clinically measurable description of refeeding syndrome, I systematically reviewed case reports and case series describing the signs and symptoms of refeeding syndrome. The intent of the review is to determine if a consistent pattern of clinical markers of refeeding syndrome are present in patients diagnosed with refeeding syndrome and to describe those markers.

Methods

I searched the English literature using the PubMed search engine and the search terms *refeeding syndrome* and *syndrome, refeeding*. The timeframe, 2000 to 2011, was consistent with an era where nutrition support clinicians were aware of refeeding syndrome, avoided the once-common practice of hyperalimentation, and used enteral and parenteral formulas with a complete array of electrolytes, vitamins, and minerals. In addition to the literature search, I hand-searched office files and bibliographies of the most recent review articles to identify additional cases.[7,14] Copies of papers were requested from authors whose work was unavailable in electronic format. Articles were reviewed and included if the interval between feeding initiation and the onset of refeeding signs and symptoms was ≤ 5 days. Attention was also given to whether patients were free of preexisting signs and symptoms of refeeding syndrome prior to feeding initiation. Articles without sufficient information to address these issues were excluded from this review. Normal serum ranges for phosphorus, magnesium, potassium, calcium, and blood glucose were used to evaluate reported serum levels of these nutrients. Reports of clinical signs and symptoms attributed to refeeding were noted so that they could be reviewed for trends.

Results

Twenty-seven cases reported by 20 authors contained descriptions of changes in laboratory and clinical data following feeding initiation. All described the appearance of refeeding signs and symptoms within the first 5 days of feeding. A number of the patients were adolescents with anorexia nervosa. As pediatric and adult clinicians use the same description of refeeding syndrome, these cases were included in the review.[14] Thus, the patients ranged in age from 10 to 90 years. Most patients received enteral nutrition administered orally or by feeding tube. Six patients received at least some parenteral nutrition. All patients demonstrated low body mass index or impaired oral intake for > 48 hours prior to refeeding. Table 1 contains a summary of the cases reviewed. Almost all patients experienced hypophosphatemia (n = 26, 96%). The majority of patients (n = 19, 71%) also experienced at least 1 other laboratory abnormality. About half the patients experienced hypomagnesemia (n = 14, 51%), and almost half had hypokalemia (n = 12, 46%). Twelve patients (46%) had low levels of phosphorus and magnesium. Eleven patients (42%) had 3 or more laboratory abnormalities. Seven patients had hypocalcemia (27%), and hyponatremia was reported in 2 patients (8%).

In 1 of the cases, a low thiamine level was present prior to feeding initiation. In at least 1 other case, the author reported signs and symptoms associated with thiamine deficiency but did not confirm the diagnosis by obtaining a nutrition history or a thiamine level. Thus, thiamine deficiency associated with refeeding was confirmed in a single patient (4%). There were no reports of hyperglycemia. Authors inconsistently reported clinical signs and symptoms, with several authors limiting their reports to laboratory data alone. Six patients (22%) had edema or weight gain in excess of that expected from lean tissue accretion. Paresthesias, a symptom of phosphorus deficiency, were reported in 4 patients (15%). Two patients had tachycardia, 2 had bradycardia (8% each), and 1 each had ventricular ectopy and heart failure.

Three case series providing mean data on electrolyte abnormalities in another 63 patients with refeeding syndrome are summarized in Table 2. Findings suggest that most patients had hypophosphatemia, but other laboratory abnormalities were not consistently observed. In 1 series of 8 patients, mean calcium levels below normal were reported. However, mean levels of magnesium and potassium remained normal or were not reported in all 3 series. Hyperglycemia was not reported in any series. Thus, no case was identified consistent with "metabolic and physiologic consequences of the depletion, repletion, compartmental shifts and interrelationships of phosphorus,

Table 1. Abnormal Laboratory Data and Clinical Findings Reported Within 5 days of Feeding Initiation in Patients Diagnosed With Refeeding Syndrome

Author	Nadir Phosphorus, < 2.4 mg/dL[a]	Nadir Magnesium, < 1.7 mg/dL[a]	Nadir Potassium, < 3.7 mg/dL[a]	Nadir Calcium, < 8.6 mg/dL[a]	Hyperglycemia	Other Signs and Symptoms
Afzal[38]	Yes	NR	No	No	NR	2.9 kg of weight gain in 72 h, fever
Akobeng[39]						
Case 1	Yes	No	No	No	NR	Acute breathlessness and tachycardia
Case 2	Yes	No	No	No	NR	Serum sodium of 133
Assiotisa[40]	Yes	Yes	Yes	No	NR	Serum sodium of 129, confusion, ventricular ectopy
Caplan[41]	Yes	Yes	NR	NR	NR	Confusion
Chiarenza[42]	Yes	Yes	NR	NR	NR	Thrombocytopenia, anemia, respiratory alkalosis
De Caprio[43]	Yes	NR	Yes	Yes	NR	Liver failure present prior to refeeding
Fisher[44]	Yes	NR	Yes	No	NR	Hyponatremia, hypochloremia; no clinical signs and symptoms reported
Fotheringham[45]	Yes	No	No	Yes	NR	Profound weakness, numbness, and paresthesias involving hands and feet
Fung[46]	Yes	Yes	Yes	No	NR	Lower limb myalgia, severe paresthesia of hands, severe diarrhea
Goldstein[47]	Yes	Yes	NR	Yes	No	NR
Hernando[48]	Yes	NR	Yes	NR	NR	NR
Jordaan[49]	Yes	Yes	NR	NR	NR	NR
Korbonits[50]	Yes	NR	NR	NR	NR	Low levels of vitamins B_1, B_2, and B_6 on admission corrected to normal by day 3
Lin[51]	Yes	NR	Yes	Yes	NR	Cardiac arrest in a patient with chronic renal failure
Mallet[52]	Yes	Yes	Yes	Yes	NR	Diarrhea
O'Connor[53]	Yes	Yes	NR	Yes	No	Hypotension, bradycardia, cardiac arrhythmias
Patel[54]	Yes	Yes	Yes	NR	NR	Difficulty breathing, abdominal pain, tachypnea, tachycardia
Stanga[55]						
Case 1	Yes	Yes	Yes	NR	NR	Vertigo, vertical nystagmus, fluid retention
Case 3	Yes	Yes	NR	NR	NR	Tachypnea, orthopnea, dependent edema, tachycardia, declining blood pressure, pulmonary edema, cardiomegaly, lactic acidosis

(continued)

Table 1. (continued)

Author	Nadir Phosphorus, < 2.4 mg/dL[a]	Nadir Magnesium, < 1.7 mg/dL[a]	Nadir Potassium, < 3.7 mg/dL[a]	Nadir Calcium, < 8.6 mg/dL[a]	Hyperglycemia	Other Signs and Symptoms
Case 4	Yes	Yes	Yes	NR	NR	Dependent edema, ascites, dyspnea, sinus tachycardia
Case 5	NR	Yes	NR	NR	NR	Ophthalmoplegia, diplopia, hypotension, thiamine deficiency
Case 6	Yes	No	Yes	NR	NR	Fluid retention, peripheral edema, pleural effusions, generalized muscle weakness, thrombocytopenia, gastrointestinal bleeding
Case 7	Yes	Yes	Yes	NR	NR	Numbness and paresthesia in the hands and feet, abdominal cramps
Tresley[56]	Yes	No	No	No	No	Bradycardia
Tripathy[57]	Yes	NR	Yes	Yes	NR	Profound weakness (flaccid paralysis), paresthesias, carpopedal spasms, hallucinations, severe diarrhea, coagulopathy
Vincent[58]	Yes	NR	NR	NR	NR	12 kg of weight gain in 5 d, lower back pain, severe bilateral pitting edema of the legs extending to the abdomen and upper limbs, deranged liver function

NR, not reported; WNL, within normal limits.
[a]Within 96 hours of feeding initiation.

Table 2. Mean Electrolyte Abnormalities in Case Series of Patients With Refeeding Syndrome

Author	n	Nadir Phosphorus, < 2.4 mg/dL[a]	Nadir Magnesium, < 1.7 mg/dL[a]	Nadir Potassium, < 3.7 mg/dL[a]	Nadir Calcium, < 8.6 mg/dL[a]
Faintuch[59]	8	Yes	No	No	Yes
Fan[31]	15	Yes	No	No	No
Lubart[60]	40	Yes	No	No	NR

For all studies, hyperglycemia was not reported (NR), and other signs and symptoms included normal serum sodium.
[a]Within 96 hours of feeding initiation.

potassium, magnesium, glucose metabolism, vitamin deficiency and fluid resuscitation."[1]

Discussion

Nutrition support clinicians often describe refeeding syndrome as a common complication of oral, enteral, and parenteral nutrition that is not widely known outside nutrition support circles.[4,15,16] However, the number of evaluable cases in this systematic review seems small compared to the total hospital population for the same period. The numbers of malnourished or starved patients in whom oral, enteral, or parenteral feedings are initiated is unknown. Based on conservative estimates of 30 million hospital discharges annually and a 10% malnutrition rate, an estimated 33 million starved or malnourished patients could have received oral, enteral, or parenteral feedings between 2000 and 2011.[17] During that period, there was no case report that described a patient with hypophosphatemia, hypomagnesemia, hypokalemia, hyperglycemia, fluid overload, thiamine deficiency, and possible mineral deficiency.[2,5,7,14,16,18] The

low incidence of refeeding syndrome may be the result of under recognition or of insufficient monitoring. In several case reports, it appeared that serum levels of phosphorus, magnesium, and calcium were monitored every third or fourth day rather than daily during feeding initiation. Serum thiamin levels were rarely measured. It is also possible that dietitians and other nutrition support clinicians trained to recognize refeeding syndrome are not available to monitor patients on a daily basis.[19,20] In institutions where clinicians see patients every 3 days or within 72 hours of feeding initiation, it is possible that abnormal levels of nutrients associated with refeeding syndrome are corrected before nutrition support clinicians return to evaluate the patients. Thus, it is possible that refeeding syndrome exists and remains undocumented.

Hypophosphatemia was almost universally present and was clearly used as a surrogate or perhaps the only marker for refeeding syndrome in the reports reviewed. Phosphorus is found in nature combined with oxygen in the phosphate form (PO_4^{-3}). In humans, 85% of phosphorus is in bone, with the remainder circulating through soft tissues.[21] Human blood and extracellular fluid contain about 465 mg (15 mmol) of inorganic phosphorus.[21] This inorganic phosphorus pool is the repository for phosphorus absorbed from food and reabsorbed from bone and is the source of phosphorus excreted in urine. Energy production temporarily increases the need for intracellular phosphorus, which is obtained from the pool of inorganic phosphorus and used during the conversion of adenosine diphosphate to adenosine triphosphate. Depending on the demand for phosphorus as glucose is converted to energy, serum phosphorus levels can drop precipitously before additional phosphorus is extracted from bone. Phosphorus levels that fall below normal are associated with poor outcomes.[10,13,22] Hematologic changes include hemolysis and rhabdomyolysis. Neurologic changes associated with hypophosphatemia include paresthesias and tremors. Perhaps the most dangerous of complications is energy deficit in the form of ATP depletion that has resulted in cardiac failure and pulmonary failure.[23]

In the US population, comparison of nutrient intake data and estimated requirements suggest that phosphorus intake is adequate in all age groups; thus, phosphorus deficiency is considered rare except in cases of starvation.[21,24] Even with decreased oral phosphorus intake combined with phosphate-binding antacids, phosphorus deficiency develops slowly over a period of several months.[25] While hospitalized patients often have inadequate food and nutrient intake, which tends to worsen as length of stay increases, it is unlikely that patients develop a phosphorus deficit during the typical 4- to 5-day hospital stay.[26-28] In the general hospital population, the incidence of hypophosphatemia may be 0.2% to 3.1%, depending on how it is defined.[29] The incidence of hypophosphatemia in geriatric facilities is reported to range from 14% to 37%.[10,30] In a series of patients with gastrointestinal fistula, 3% experienced hypophosphatemia, and in postoperative patients the incidence was 44%.[31,32] In patients with hepatic resection, the reported incidence of hypophosphatemia was 100%.[33] Thus, advancing age and illness severity may be associated with a greater incidence of hypo-phosphatemia.

Given that many reported causes of hypophosphatemia are present in patients receiving nutrition support, it is appropriate to carefully evaluate the adequacy of current and recent phosphorus intake, to determine the cause of hypophosphatemia, and rule out other causes before attributing hypophosphatemia to refeeding.[13] The higher incidence of hypophosphatemia found among intensive care unit patients is attributed to mechanical ventilation, sepsis, and respiratory alkalosis.[10,13,23] The incidence of hypophosphatemia is also greater in neoplastic disease and diabetes mellitus.[13,10] In a review of more than 45,000 reports of hypophosphatemia, almost half were attributed to intensive care unit admission, while only about 10% were attributed to refeeding.[13] Hypophosphatemia is attributed to medications, including insulin, epinephrine, dopamine, salbutamol, xanthine derivatives, erythropoietin, and GM-CSF.[34] It is tempting to speculate that the absence of hyperglycemia among patients in this review is the result of insulin administered to achieve tight glucose control that also resulted in hypophosphatemia, and that idea may be worthy of consideration.

As with any systematic review, this one is limited by the volume and quality of the available data. It is possible that additional cases are available but were not identified by the selected search engine. The data reviewed are from a small number of patients (n = 90); however, the definitions of refeeding syndrome used in practice were likely based on an even smaller number of patients. Clinical signs and symptoms were described by more than 20 clinicians, which is likely a source of inconsistency. Normal laboratory values were used as diagnostic criteria for refeeding syndrome. However, some clinicians favor treating only patients with severe depletion or those who are symptomatic, while others recommend treating normal but decreasing levels of serum phosphorus.[12,23,35-37] Since none of the patients in this analysis exhibited all the signs and symptoms of refeeding syndrome, it may be appropriate to designate specific levels and a minimum number of criteria to define refeeding syndrome. It may also be clinically relevant to diagnose refeeding hypophosphatemia rather than refeeding syndrome. Clearly, more information and discussion are needed to develop a consensus definition of refeeding syndrome.

Conclusions

This systematic review may represent the first comparison of data from case reports with published descriptions of refeeding syndrome. The assembled information suggests that refeeding syndrome, as described in the literature, is rare based on reports published since 2000. This review raises the question of whether there is a need to further define refeeding syndrome and to distinguish it from refeeding hypophosphatemia. The data also raise the question of whether a greater incidence of refeeding syndrome would be identified with more frequent

laboratory monitoring and more frequent follow-up by clinicians. Controlled studies with adequate follow-up and detailed descriptions of nutrient intake as well as detailed monitoring are needed to further define and distinguish refeeding syndrome and refeeding hypophosphatemia.

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