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Recognizing and Preventing Refeeding Syndrome

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Refeeding syndrome is an uncommon but potentially fatal phenomenon that can occur in patients receiving parenteral, enteral, or oral feedings after a period of sustained malnutrition or starvation. This syndrome is characterized by hypophosphatemia, hypokalemia, and hypomagnesemia. The purpose of this article was to bring an acute awareness of refeeding syndrome to the critical care nurse. The recognition, pathogenesis, clinical manifestations, potential life threatening complications, and treatment are presented.
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Refeeding syndrome is an uncommon but potentially fatal phenomenon that can occur in patients receiving parenteral, enteral, or oral feedings after a period of sustained malnutrition or starvation.[1-3] Refeeding syndrome was first recognized in starved World War II prisoners of war and victims of famine. Fatal cardiac complications transpired after rapid initiation of nutrition.[4,5] Refeeding syndrome is manifested by acute fluid and electrolyte disturbances. Hypophosphatemia is the abnormality most associated with refeeding syndrome; however, hypokalemia, hypomagnesemia, hyperglycemia, and thiamine deficiency may simultaneously exist.[1-3] The purpose of this article was to bring an acute awareness of refeeding syndrome to the critical care nurse. The recognition, pathogenesis, clinical manifestations, potential life-threatening complications, and treatment are presented. A case study illustrates key points in the diagnosis and treatment of refeeding syndrome.

CASE STUDY

Initial Presentation

A 63-year-old white female with a history of non–small-cell lung cancer was admitted to the hospital with severe nausea, vomiting, and repeated falling secondary to dizziness 1 week after completion of chemotherapy with cisplatin and etoposide. Her medical history is significant for chronic obstructive lung disease, hypertension, and depression. Six months before admission, she had a right pneumonectomy. Her current medications include atenolol, promethazine, and lorazepam. She admits to prior use of alcohol and tobacco but denies any use in the last 6 months. She has no known allergies.

Upon review, the patient was unable to quantify the amount of vomiting. She described her appetite as poor but denied any recent weight loss. She denied having diarrhea, abdominal pain, hematemesis, rectal bleeding, or cardiopulmonary symptoms. She appeared cachexic and fatigued. Vital signs revealed heart rate of 90 beats per minute, blood pressure of 100/43 mm Hg, and respiratory rate of 20 breaths per minute. She was oriented and cognitively intact. Physical examination revealed dry oral mucosa and poor skin turgor. Breath sounds were absent on the right lung, with no adventitious sounds on the left lung. A chest x-ray was completed and showed no infiltrates. All laboratory values are presented in Table 1. Electrocardiogram showed normal sinus rhythm with no changes from prior studies.

TABLE 1 Laboratory Values

Laboratory Values	Normal	Initial	Day 3	Day 4	Day 5	Day 10
Phosphorus, mg/dL	2.4-4.4	unknown	3.0	3.1	1.0	4.2
Potassium, mEq/L	3.5-5.0	3.3	3.5	2.8	2.7	4.4
Magnesium, mg/dL	1.5-2.4	unknown	2.2	1.8	1.1	2.0
Albumin, mg/dL	3.5-5.0	2.5	2.4	2.8		3.0
Prealbumin, mg/dL	18.0-38.0		8.9			14.5
Hematocrit, %	38-50	33.8				
Blood urea nitrogen, mg/dL	12-25	42	6			
Creatinine, mg/dL	0.5-1.2	1.3	0.6			
Sodium, mEq/L	135-145	134	133			
Glucose, mg/dL	60-110	133	63			

Patient sample is composite and does not reflect individual protected healthcare information.

The patient was admitted to the oncology unit with a diagnosis of dehydration secondary to vomiting. Vomiting was attributed to adverse effects of the chemotherapy. The treatment plan included volume repletion with normal saline and promethazine as needed for nausea.

Day 3

The patient's nausea persisted with occasional vomiting. She continued to have a diminished appetite with inadequate oral intake. In addition, her weight had dropped from 60 kg on admission to 56.5 kg. A peripherally inserted central catheter was placed, and parenteral nutrition was initiated. Again, all laboratory values can be found in Table 1. Actual body weight was used to determine her caloric intake.

Day 4

Acute onset of dyspnea, manifested with subsequent tachypnea, tachycardia, and hypoxia, occurred the morning after initiation of parenteral nutrition. Differential diagnoses included aspiration, pulmonary embolism, and exacerbation of chronic obstructive lung disease. See Table 1 for her laboratory values. She was treated with oxygen and aerosols and was transferred to the intensive care unit for closer monitoring. A computed tomography scan was completed, with no evidence of pulmonary embolism. She received additional potassium, and the parenteral electrolyte supplementation was adjusted.

Day 5

Thirty-six hours after initiation of parenteral nutrition, laboratory values revealed significant derangements in potassium, magnesium, and phosphorus (Table 1). Her differential diagnosis now included refeeding syndrome. Phosphorus, magnesium, and potassium supplements were given intravenously. Her chest x-ray showed congestion, and the patient required increased oxygen support. Over the next several days, serum electrolyte levels were evaluated daily and supplemented as needed, the pulmonary and cardiac symptoms subsided, and her condition improved.

Day 10

The patient continued to tolerate oral intake, and parenteral nutrition was successfully weaned. Her electrolytes were within normal limits (see Table 1). She was discharged home with her daughter.

PATHOGENESIS

Starvation

During periods of malnutrition or starvation, the body uses alternative means for energy. Initially, liver stores of glycogen are broken down to provide a source of glucose, and skeletal muscles provide amino acids for new glucose production. After several days, insulin production decreases, a decrease in metabolic rate occurs, and energy is derived from ketone production resulting from lipolysis, resulting in the preservation of the body's store of protein.[6]

Refeeding syndrome occurs when patients are reintroduced to carbohydrates as the primary source of energy. Glucose metabolism causes an increase in the use of phosphate, which is used to produce adenosine triphosphate (ATP) and 2,3-diphosphoglycerate.[1,7] This demand causes an increased cellular uptake of phosphorus, resulting in a low serum phosphorus level.[1] Hypophosphatemia is the predominate phenomenon associated with refeeding syndrome. In addition, carbohydrates stimulate pancreatic insulin secretion, resulting in increased cellular glucose uptake and protein synthesis.[4,8] These changes precipitate intracellular movement of phosphorus, potassium, and magnesium, worsening hypophosphatemia and causing hypokalemia and hypomagnesemia.[4,8]

Hypophosphatemia is the predominate phenomenon associated with refeeding syndrome.

CLINICAL MANIFESTATIONS

The clinical manifestations associated with refeeding syndrome coincide with fluid and electrolyte disturbances.[1] Although laboratory results are more likely to guide the diagnosis, clinical manifestations of electrolyte derangements guide the urgency of the treatment. When clinical manifestations of hypokalemia, hypophosphatemia, and hypomagnesemia are severe or life threatening, rapid intravenous repletion is essential. When clinical symptoms are absent or mild, slower oral or nasogastric repletion is implemented. A review of the actions of phosphorus, potassium, and magnesium on body systems and the clinical effects of the key refeeding electrolytes, hyperglycemia, and low thiamine levels is detailed in the next section.

Hypophosphatemia

Phosphorus is primarily located intracellularly, with only 1% of the total body phosphorus located extracellularly. Therefore, serum levels may not accurately reflect total body phosphorus levels.[9] Normal serum phosphorus concentration is 2.5 to 4.5 mg/dL.[10] Phosphorus is essential in the formation of ATP for cellular energy production and 2,3-diphosphoglycerate, which is necessary for oxygen unloading in the tissues.[6,10]

Multiple studies have been completed to evaluate the precedence of hypophosphatemia in the hospitalized patient.[11] The findings indicate that as many as 42% of hospitalized patients, especially those in critical care units, have experienced hypophosphatemia.

Hypophosphatemia is identified as a level less than 2.5 mg/dL and is most common within 2 to 4 days of refeeding.[5] Mild to moderate hypophosphatemia is often asymptomatic and may go unrecognized.[10] However, significant events occur if phosphorus levels drop below 1.5 mg/dL.[3] Symptoms associated with hypophosphatemia include weakness, respiratory distress, rhabdomyolysis, heart failure, or death.[10] Diaphragmatic contractility can be impaired in patients with low phosphate levels.[4] In addition, hypophosphatemia can cause neurological symptoms including parasthesias, confusion, and seizures. Hematologic consequences of hypophosphatemia include thrombocytopenia and leukocyte dysfunction.[10]

Although hypophosphatemia is the hallmark sign of refeeding syndrome,[3] there are a multitude of other causes of hypophosphatemia. These include malabsorption, volume repletion, hemodialysis, diabetic ketoacidosis, sepsis, and use of a plethora of medications.[12]

Hypokalemia

Ninety-eight percent of total body potassium is intracellular. The remaining 2% is located in the extracellular compartment. The normal serum potassium level is 3.5 to 5.0 mEq/L.[13] Potassium is critical for maintaining cell membrane action potential, especially in the myocardium. In addition, potassium functions in cell metabolism, glycogen synthesis, and protein synthesis.[9]

Hypokalemia is most often associated with patients receiving diuretics and can be present without symptoms. Other common causes of hypokalemia include diarrhea, vomiting, and alkalosis. More than 20% of hospitalized patients develop hypokalemia.[14] Hypokalemia interferes with action potential movement across cell membranes, thereby impairing muscle contraction.[1] Complications associated with hypokalemia range from mild weakness to life-threatening arrhythmias.[15] The response to hypokalemia is patient-specific and depends upon concomitant electrolyte and acid-base imbalances.[15] Magnesium is essential for potassium uptake and maintenance of intracellular potassium. Magnesium depletion should be suspected in refractory hypokalemia although potassium is being replaced.[15]

Hypomagnesemia

Magnesium primarily exists intracellularly. Only 1% of total magnesium is found in extracellular fluid.[1] Normal serum concentration of magnesium is 1.5 to 2.4 mg/dL.[9]

Magnesium inhibits the release of acetylcholine at neuromuscular junctions[15] and is involved in the phosphorylation of ATP.[16] Hypomagnesemia may increase cardiac irritability and worsen cardiac arrhythmias.[16] In addition to cardiac effects, hypomagnesemia may exhibit as insomnia, hyperactive reflexes, muscle cramps, tetany, and seizures.[2,15] In addition to refeeding syndrome, hypomagnesemia can be attributed to chronic alcoholism, malnutrition, chronic diarrhea, pancreatitis, diabetic ketoacidosis, diuretic therapy, and the use of multiple medications.[12,15]

Hyperglycemia

The initiation of glucose initially suppresses gluconeogenesis; however, continuation results in hyperglycemia.[4] Others[7,17,18] have studied cases of refeeding syndrome. In these cases (all females), the women were severely malnourished and chronically ill. After initiation of parenteral nutrition, cardiopulmonary failure ensued, resulting in their deaths.[16] In each case, profound hypophosphatemia[7] developed, as well as hyperglycemia. The glucose levels peaked between 700 and 1,200 mg/dL. Hyperglycemia can lead to osmotic diuresis, dehydration, and hyperosmolar nonketotic coma.[17]

Thiamine Deficiency

Thiamine is a water-soluble vitamin, which can easily be depleted in patients who are malnourished.[8,12] Thiamine

is required in carbohydrate metabolism and glucose utilization.[8,12] With a deficiency in thiamine, there is a disruption in the conversion of pyruvate to acetyl coenzyme-A. Instead, the pyruvate is converted to lactate. If lactic acid is in excess, lactic acidosis and death can result.[8,12] Another complication associated with prolonged thiamine deficiency is Wernicke encephalopathy.[8,12]

DIAGNOSTIC CRITERIA AND DIFFERENTIAL DIAGNOSES

A diagnosis of refeeding syndrome is based on the presence of multiple predisposing factors; initiation of oral, parenteral, or enteral feedings; and the subsequent development of fluid and electrolyte imbalances.[1,3,4] Development of clinical manifestations after significant changes in the electrolytes further supports refeeding syndrome.[1,3,4] When considering the differential diagnosis of hypophosphatemia, hypokalemia, and hypomagnesemia related to refeeding syndrome, diagnoses of alkalosis, sepsis, alcoholism, surgery, diarrhea, vomiting, cirrhosis, drugs, diuretic therapy, and hypercalcemia must be included.[10] Critically ill patients present with multiple diagnoses. Many of these diagnoses disrupt electrolyte balance. Refeeding syndrome further complicates the management of these other disease processes and electrolyte imbalances.

INCIDENCE

Patients at risk of developing refeeding syndrome present with some degree of malnutrition. The disease processes that accompany this condition are multivariant and include anorexia nervosa, chronic alcoholism, metastatic cancer, malabsorption diseases, and uncontrolled diabetes.[2,10] Risk factors that may precipitate refeeding syndrome include prolonged vomiting and diarrhea, homelessness when associated with inadequate nutrition and alcoholism, hunger strikes, prolonged fasting, surgery, and depression.[2,10]

Refeeding syndrome often goes unrecognized.[5] Cancer patients have a unique set of problems, which increases their vulnerability to refeeding syndrome.[5] Many patients undergoing chemotherapy present with decreased appetite, nausea, and vomiting. In addition, oral stomatitis, a frequent complication in patients radiated for head and neck cancer or in recipients of chemotherapy, hinders oral intake.

Cancer patients have a unique set of problems, which increases their vulnerability to refeeding syndrome.

The exact prevalence of refeeding syndrome is unknown. The development of refeeding syndrome has been reported as high as 25% in cancer patients receiving nutritional support.[4,19] However, in patients receiving parenteral supplementation, hypophosphatemia can occur in 30% to 38% who are receiving phosphate. This percentage can increase up to 100% in patients not receiving phosphate.[20]

Kagansky and colleagues[11] conducted a case-control study of all patients aged 65 years or older who were hospitalized in a geriatric division between January 1, 2001 and December 31, 2001. The study investigated episodes of hypophosphatemia. Of the 2,307 patients, 14.1% had documented hypophosphatemia. The patients who had developed hypophosphatemia while hospitalized had a higher incidence of weight loss before admission and had lower albumin levels, and a large portion had received intravenous glucose solutions or food supplementation while hospitalized.[11] These data suggest that these patients presented with a less than optimal nutritional status and that the elderly are at risk for refeeding syndrome.

PREVENTION

The first step in the prevention of refeeding syndrome is identifying those patients at risk. The patient identified in the case study depicts a typical presentation of a patient at risk for the development of refeeding syndrome. An astute practitioner is instrumental in identifying a patient who is vulnerable to this scenario, thereby facilitating the implementation of nutrition appropriately and avoiding the detrimental consequences of refeeding. A thorough history and physical assessment can identify risk factors and characteristics of malnutrition (eg, muscle wasting, dry skin, thin hair).

Laboratory assessment includes obtaining serum albumin and prealbumin levels. Serum albumin with a 2- to 3-week half-life is highly sensitive for malnutrition but has a low specificity.[21] Prealbumin is used as an indicator in monitoring the effectiveness of nutrition replacement. Because of its short half-life of 1.9 days, it is a more sensitive indicator of nutritional status.[16] Transferrin with a 1-week half-life can also be used.[21] However, in a critical illness, these markers of nutrition marginalize and travel from the vascular system to the liver and other organs for synthesis of protein used during acute illness.[16] As a result, serum markers of nutrition are not consistent during injury, systemic inflammation, or exacerbation of chronic conditions.

Patients at risk for refeeding syndrome need monitoring of electrolytes, particularly phosphorus, magnesium, and potassium, before feeding and daily for 4 to

7 days.[3] If electrolyte abnormalities are present, oral replacements are preferred if they can be tolerated. Alternatively, intravenous supplements can be used in patients who are unable to take oral supplements. Nutrition implementation is delayed until electrolytes are normalized.[22]

Several strategies for beginning feedings have been proposed. A conservative recommendation[7,9] suggests starting at 25% of goal on day 1 and gradually increasing over several days. Recommendations that are more common advocate 20 kcal/kg per day or 1,000 kcal/d.[5,12,18] In severely malnourished patients, 15 kcal/kg per day is appropriate.[12] Goal rate can be accomplished within 5 to 7 days. Any additional caloric sources need to be included, such as diprovan or dextrose infusions.[12] The macronutrient values of these must be counted as well. There are no data to support protein restriction, and thus, it is included in standard total parenteral nutrition.[18] Thiamine and multivitamins are important supplements when beginning nutritional support and are included in most standard formulas.[3] Patients at risk of refeeding syndrome should be provided with thiamine doses of 50 to 100 mg/d intravenously or 100 mg/d orally.[7]

Patients identified to be at an elevated risk for refeeding syndrome should be closely monitored for signs and symptoms associated with hypophosphatemia, hypokalemia, and hypomagnesemia.[7] In addition, fluid balance and daily weights must be monitored.[7]

TREATMENT

The treatment for refeeding syndrome involves immediate management of any respiratory compromise and prompt correction of electrolyte abnormalities.[12] Hypophosphatemia treated orally requires 1 to 2 mg daily in divided doses or treated intravenously with 2 mg/kg infused over 6 hours.[12,23] Hypokalemia is treated immediately if associated with cardiac arrhythmias. Intravenous doses of 20 to 80 mEq can be safely infused at 10 mEq/h through a central live if properly diluted.[1,23] Doses greater than 20 mEq/h are reserved for emergent situations.[1] Cardiac monitoring is imperative when infusing 10 mEq/h of potassium intravenously in acutely ill adults.[23] In patients with hypophosphatemia and hypokalemia, potassium phosphate can provide dual replacement.[24] Hypomagnesemia requires correction in conjunction with hypokalemia, lest replacement of potassium will be ineffective.[23,24] The daily requirement for magnesium is 0.4 mEq/kg per day. Hypomagnesemia can be corrected with 1 to 2 g of magnesium sulfate at the rate of 2 g over 4 hours.[23,24] Too rapid infusion is associated with respiratory depression characterized by decreased respiratory rate and tidal volume. Patients with renal impairment require individualized adjustments when replacing electrolytes.[23,24] Hyperglycemia can be effectively treated with insulin.[25] Multiple studies have been completed regarding the efficacy of management of hyperglycemia in the critically ill patient.[26-28]

Nutritional support teams can provide direct and consultative roles in caring for patients who are at risk for refeeding syndrome. A consultation with a nutrition team would be beneficial when initiating therapy to prevent the potential complications of refeeding syndrome. A small study completed in a Spanish Hospital used a nutrition support team and individualized parenteral nutrition on 11 at-risk patients. Of these 11 patients, only mild electrolyte disturbances were noted. The study suggests that parenteral nutrition needs to be adjusted on an individual basis.[29]

A consultation with a nutrition team would be beneficial when initiating therapy to prevent the potential complications of refeeding syndrome.

In the case study presented, the patient had only mild electrolyte deficiencies initially and, despite correction, had further deterioration and electrolyte imbalance requiring a more aggressive treatment and intensive care management. The patient presented with multiple characteristics of malnutrition and consequently was a candidate to develop refeeding syndrome upon initiation of parenteral nutrition.

SUMMARY

Refeeding syndrome is an overlooked but a clinically significant phenomenon that has potentially fatal complications. Patients receiving oral, parenteral, or enteral feedings after having periods of starvation or who are severely malnourished are at an increased risk of developing hypophosphatemia, hypokalemia, and hypomagnesemia. Intensive care patients may have additional underlying electrolyte derangements that increase their risk for refeeding syndrome. The pivotal intervention for refeeding syndrome is prevention. Early recognition of at-risk patients targets electrolyte normalization before feeding and provides glycemic control during feeding. Each clinician must be aware and knowledgeable regarding the pathophysiology and the complications associated with this uncommon diagnosis.[25]

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