CASE REPORT

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Management of refeeding syndrome in a severely wasted complex case pediatric patient in a limited facilities hospital: a case report

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ABSTRACT

Backgrounds: Refeeding syndrome is a life-threatening metabolic complication that may occur during nutritional rehabilitation in patients with severe malnutrition. Diagnosing this condition is particularly challenging in resource-limited settings due to the lack of comprehensive laboratory facilities, although prompt recognition and treatment are essential to prevent serious outcomes.

Case Presentation: We report an 11-month-old boy presenting with pallor, generalized weakness, severe wasting, a senile facial appearance, prominent ribs, abdominal distention, phimosis, pedal edema, and global developmental delay. Pulmonary auscultation revealed fine crackles bilaterally. Laboratory investigations demonstrated very severe anemia (hemoglobin 2.7 g/dL), leukocytosis, hypoalbuminemia, and the presence of *Ascaris lumbricoides* ova. Chest radiography suggested pulmonary tuberculosis. The patient was treated with packed red cell transfusions, broad-spectrum intravenous antibiotics, multivitamins, folic acid, and first-line anti-tuberculosis drugs. Nutritional support was initiated cautiously following stabilization, but on day five, the patient developed recurrent pedal edema, hypokalemia, hypernatremia, and a further decline in serum albumin. Based on the consensus criteria of the American Society for Parenteral and Enteral Nutrition (ASPEN), a diagnosis of refeeding syndrome was established. Electrolyte abnormalities were corrected, and nutritional support was restarted from the stabilization phase with close monitoring. Anthelmintic and iron therapy were administered during the rehabilitation phase. The patient showed gradual clinical improvement and was discharged on day 26.

Conclusion: Early diagnosis of refeeding syndrome is feasible in resource-limited hospitals using the updated diagnostic criteria provided by ASPEN. Careful nutritional repletion, timely administration of thiamine and multivitamins, correction and monitoring of electrolytes, and vigilant clinical observation are essential for the effective management of pediatric refeeding syndrome.

Keywords: Refeeding Syndrome, Case Report, Pediatrics, Wasting Syndrome, Severe Acute Malnutrition.
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INTRODUCTION

Refeeding syndrome is a condition of reduced electrolytes, fluid retention, and changes in glucose balance that occurs in patients who experience nutritional deficiencies during nutritional restoration either orally, enterally, or parenterally. Refeeding syndrome starts when hyperglycemia occurs upon refeeding in a body that was previously undergoing starvation.[1] Electrolytes then shift intracellularly as glucose becomes available once again to facilitate cellular processes. This causes a drop in the serum concentration of the already depleted supply of electrolytes in the body. Hypokalemia and hypomagnesemia are caused by the rapid shift of potassium and magnesium along with glucose and amino acids. Hypophosphatemia reflects the increase in adenosine triphosphate production from the newly available carbohydrate.[1]

Reports regarding the incidence of refeeding syndrome in children are quite rare. A cohort study conducted in 2003 of 164 patients in intensive care who were given parenteral nutrition. It was found that 15 patients were at risk of experiencing refeeding syndrome, with the incidence of decreased serum electrolytes in the first 72 hours of nutrition was 27% of the total population, and 8 of 15 were at risk, even though nutrition had been provided carefully.[2-5]

Previously, there was no standard definition of refeeding syndrome; moreover, research and studies discussing refeeding syndrome were still limited. Apart from that, in the past, hypophosphatemia was often considered the primary marker of this syndrome.[1-6] In contrast, many hospitals with limited laboratory facilities are unable to carry out phosphate tests. Hence, refeeding syndrome is often difficult to diagnose and treat early in hospitals with limited facilities, which could be fatal to the patient. Therefore, a fast and precise way is needed to diagnose refeeding syndrome, especially in limited facilities, so that this syndrome can be treated immediately and does not have fatal consequences.

The American Society of Parenteral and Enteral Nutrition (ASPEN) previously issued a consensus on refeeding syndrome in 2020.[7] The recommendations were formulated by an interprofessional task force aiming to provide clinical guidance regarding the prevention and management of refeeding syndrome. The recommendations also supplied guidance on prompt diagnosis and treatment of refeeding syndrome in limited facilities with simpler laboratory tests, so that immediate treatment can be carried out on patients. Based on those mentioned above, this case study aims to evaluate the complex case of refeeding syndrome in a severely wasted pediatric patient treated in a resource-limited facility.

CASE PRESENTATION

An 11-month-old boy presented to the emergency department with complaints of generalized pallor, shortness of breath, and weakness. According to his mother, the patient had appeared pale for the past seven months, with worsening pallor over the preceding two weeks. He had also exhibited progressive dyspnea during the same two-week period, appearing to struggle with breathing. Additionally, the patient had become increasingly weak and was reportedly unable to sit or roll over; he mainly remained bedridden.

The mother stated that the child's early growth and developmental milestones were initially appropriate for age. He began social smiling and turning his head from side to midline by two months, was able to lie in the prone position by three months, and lift his head by four months. However, developmental regression was noted starting at five to six months of age, as he was unable to sit even with assistance. Since becoming malnourished, the patient had demonstrated marked hypotonia and failed to achieve further motor milestones. In terms of language development, the patient was only capable of cooing sounds such as "aah" and "ooh."

The patient received both breast milk and formula during the first two months of life. Starting at three months of age, the mother exclusively breastfed, discontinuing formula due to the infant's apparent refusal to take it. From the age of four months, breastfeeding was provided solely from the right breast, as the mother reported no milk production from the left breast. Complementary feeding was introduced at six months of age, consisting primarily of rice porridge with vegetables such as carrots and spinach, and occasionally supplemented with animal protein sources like eggs. The complementary food was given 2–3 times daily, with an estimated portion size of 2–3 tablespoons per meal. At the time of admission, the mother reported breastfeeding approximately 10–12 times per day, exclusively from the right breast. She had never expressed or pumped her breast milk.

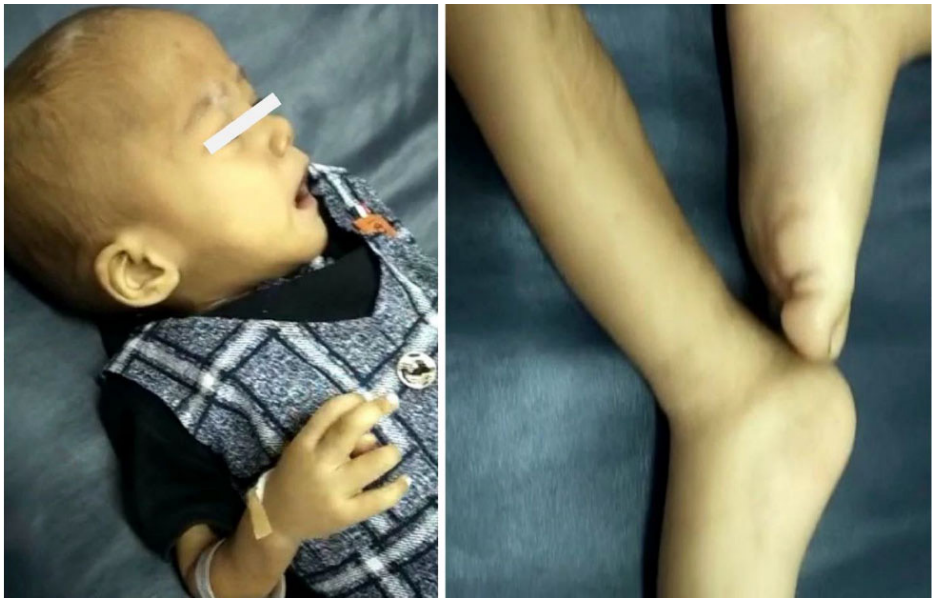

Figure 1. The patient's clinical picture at the initial presentation showed the telltale older adult facies along with pedal edema.

Table 1. Baseline laboratory values

Examination	Results	Unit	Normal
Complete blood count			
Hemoglobin	2.7	g/dL	13.2–17.3
Hematocrit	8	%	35–47
Leukocyte	11.5	$\times 10^3/mm^3$	3.8–10.6
Thrombocyte	209	$\times 10^3/\mu L$	150–440
Erythrocyte	0.9	$\times 10^3/mm^3$	4.4–5.9
Differential count			
Eosinophil	0	%	2–4
Basophil	0	%	0–1
Band neutrophil	2	%	3–5
Segmented neutrophil	14	%	50–70
Lymphocyte	83	%	20–40
Monocyte	1	%	2–8
Blood chemistry			
Ureum	18	mg/dL	15–36
Creatinine	0.3	mg/dL	0.9–1.3
Glucose (random)	76	mg/dL	<200
Albumin	2.8	g/dL	3.5–5.2
AST	12	U/L	<41
ALT	14	U/L	<35

On physical examination, the patient was alert but appeared weak, pale, and dyspneic. His vital signs revealed tachypnea. The patient exhibited a characteristic "old man facies," and his palpebral conjunctivae were pale, suggestive of severe anemia (Figure 1). Thoracic examination revealed prominent ribs,

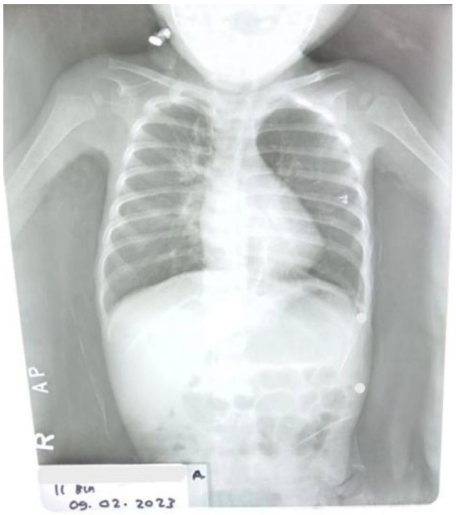

Figure 2. The patient's initial chest x-ray showed bilateral suprahilar, perihilar, and paracardial infiltrates, which were suggestive of pulmonary tuberculosis.

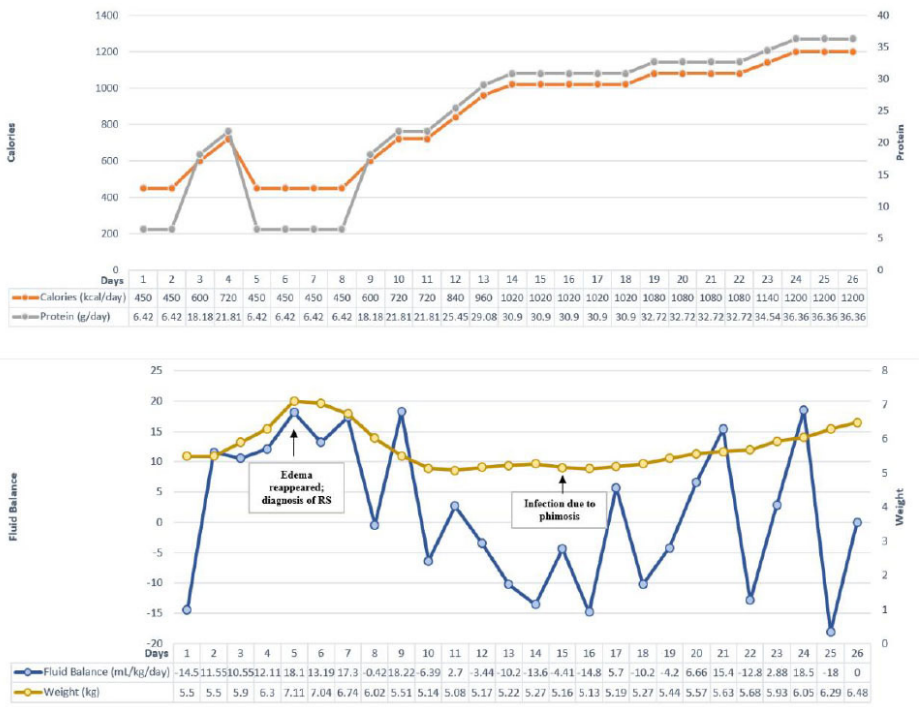

Figure 3. A timeline of the patient's weight, caloric intake, protein intake, fluid balance, and significant events throughout hospitalization.

and fine crackles were auscultated in both lung fields. The abdomen was distended, and marked gluteal muscle wasting was observed, giving a "baggy pants" appearance. Pitting edema was present in both lower extremities. Laboratory findings (Table 1) demonstrated severe anemia with a hemoglobin level of 2.7 g/dL and leukocytosis with a total leukocyte count of 11,500/mm^3. Chest radiography (Figure 2) revealed findings suggestive of pulmonary tuberculosis.

Anthropometric assessment revealed that the patient had a body weight of 5.5 kg, a body length of 70 cm, a head circumference of 44 cm, and a mid-upper arm circumference (MUAC) of 11 cm. When plotted against the 2006 World Health Organization (WHO) growth standards, the patient's z-scores were as follows: weight-for-age −4.66 SD, length-for-age −2.17 SD, weight-for-length −5.16 SD, head circumference-for-age −1.49 SD, and MUAC-for-age −3.6 SD. These findings indicate that the patient was severely underweight, severely wasted, stunted, and normocephalic.

In the emergency department, the patient received oxygen therapy via nasal cannula at a flow rate of 0.5 L/min. Initial stabilization measures included confirmation of normoglycemia, as well as ensuring the absence of hypothermia and dehydration. Empirical antibiotic therapy was initiated with intravenous (IV) ampicillin-sulbactam at a dose of 275 mg four times daily and IV gentamicin at 45 mg once daily. Anti-tuberculosis treatment was commenced using a fixed-dose combination (FDC) pediatric formulation for the intensive phase, administered as one tablet once daily.

The patient also received a packed red blood cell (PRC) transfusion with 28 mL administered over the first hour, followed by 55 mL over the subsequent three hours, adhering to a total transfusion volume not exceeding 15 mL/kg/day. Adjunctive nutritional support included vitamin A (100,000 IU, single dose), folic acid (5 mg on the first day, followed by 1 mg once daily thereafter), vitamin B complex (½ tablet once daily), vitamin C (50 mg once daily), and zinc supplementation (20 mg once daily).

Figure 3 describes the patient's clinical course throughout hospitalization, highlighting changes in caloric intake, protein intake, fluid balance, and weight trajectory. After clinical stabilization, nutritional therapy was initiated with 450 kcal/day and 6.42 g/day of protein using F75 formula delivered at 50 mL every 2 hours via a nasogastric tube. On the second day, there was a visible reduction in pedal edema, followed by complete resolution by the third day. The patient entered the transition phase on day 3 with an increased intake of 600–720 kcal/day and 18.18–21.81 g/day of protein on days 3–4. However, by day 5, there was a reappearance of pedal edema, abdominal distension, and a significant weight gain from 6.3 kg to 7.11 kg, along with a fluid balance spike of +18.1 mL/kg/day. These signs coincided with laboratory findings of hypokalemia (2.7 mmol/L), hypernatremia (153.3 mmol/L), and hypoalbuminemia (2.1 g/dL), leading to the clinical diagnosis of refeeding syndrome based on ASPEN criteria. The patient was immediately returned to the stabilization phase (Figure 4).

On day 6, fluid balance remained high at +17.3 mL/kg/day, and the patient received albumin and potassium correction. By day 7, edema began to subside, the abdomen became less distended, and the corrected potassium level improved to 3.2 mmol/L, although albumin remained low (2.1 g/dL), prompting a second albumin infusion. By day 8, the patient's weight had decreased to 6.02 kg, edema had further reduced, and albumin slightly increased to 2.3 g/dL. By day 9, pedal edema had resolved completely.

On day 15, the patient developed a

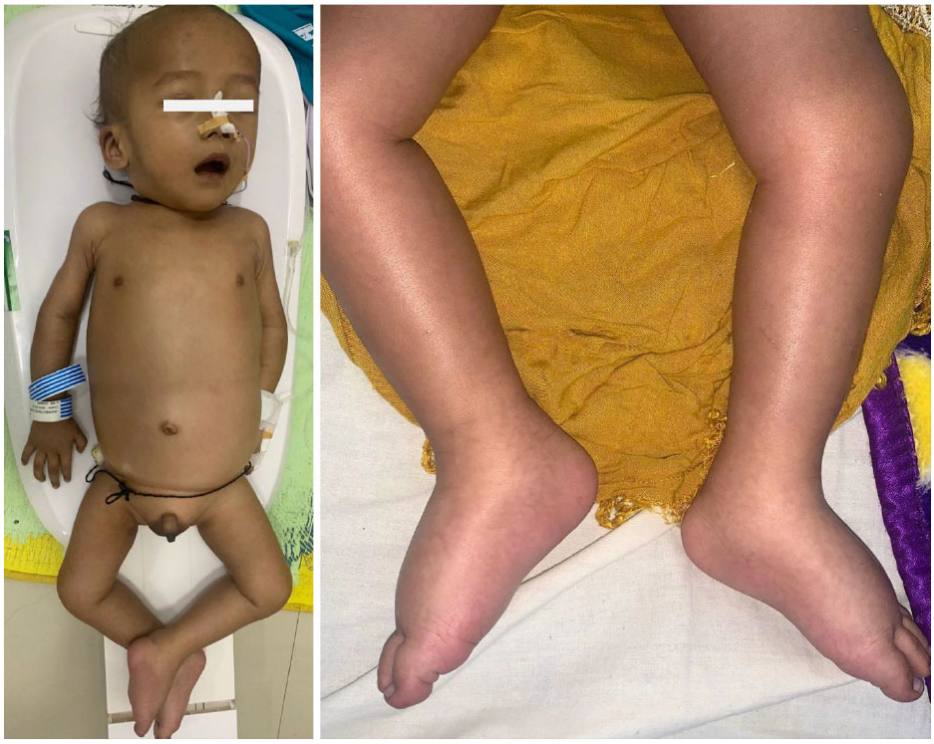

Figure 4. Reappearance of edema and abdominal enlargement on the fifth day of treatment warrants an evaluation for refeeding syndrome.

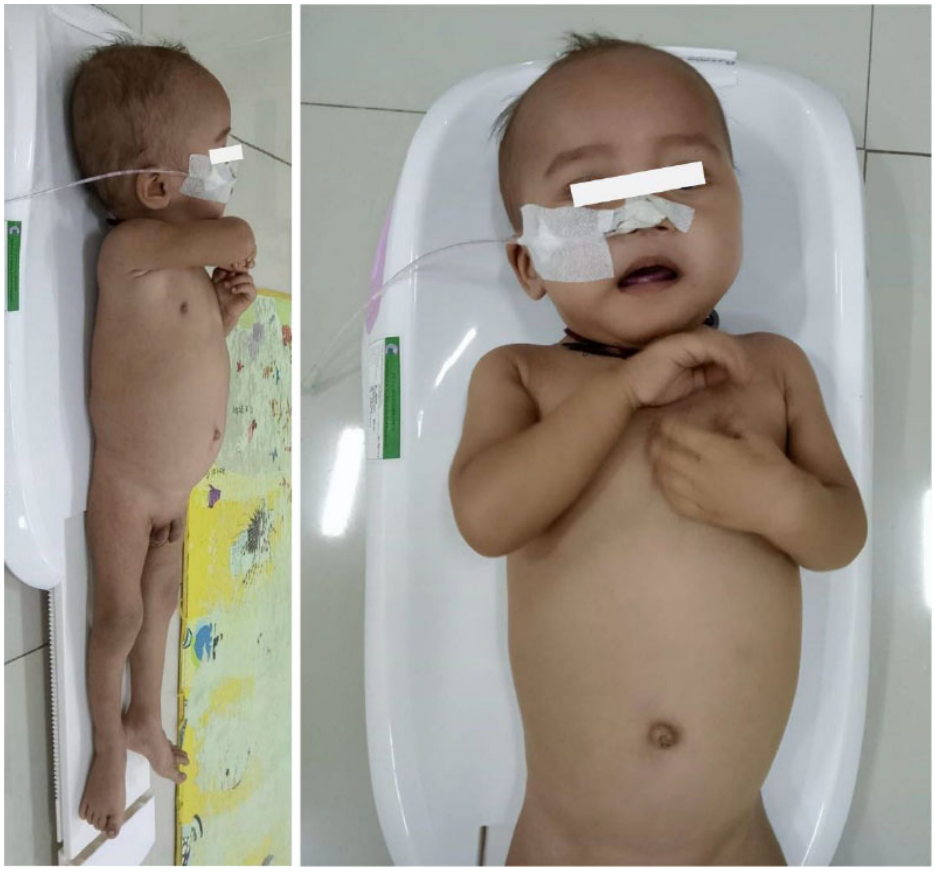

Figure 5. The patient's clinical picture at discharge showed the disappearance of the older adult's facies, pedal edema, and prominent ribs.

fever (38.3°C), coinciding with weight loss to 5.16 kg and leukocytosis (17,100/mm^3). The suspected cause was a urinary tract infection secondary to phimosis. Despite this, fluid balance remained within manageable limits (around -4.41 mL/kg/day), and with appropriate antibiotic therapy, the patient's clinical condition improved. From day 17 onward, nutritional intake was gradually increased to 1,200 kcal/day and 36.36 g/day of protein, with stabilization of weight (up to 6.48 kg on day 26) and maintenance of fluid balance within acceptable ranges. The patient was discharged on day 26 in improved condition and continued with outpatient follow-up (Figure 5).

DISCUSSION

When undergoing starvation, the body adapts by restricting growth, losing fat, muscle, and visceral mass, and reducing basal metabolic rate to reduce the total energy expenditure. Various hormones and organ systems become affected, which are detrimental to the child's growth and development. As glycogen storage is used up, rapid gluconeogenesis happens with consequent loss of skeletal muscle and fat.[3]

Along with other organ systems, the immune system (specifically cellular immunity) is also affected by malnutrition. This leads to an increased susceptibility to infections.[3] This secondary malnutrition has implications for tuberculosis, an infection highly prevalent in Indonesia. Indonesia accounts for 8.5 percent of global tuberculosis cases, with undernutrition being one of the top three risk factors for tuberculosis in Indonesia. Pediatric tuberculosis makes up about 12% of the total cases in Indonesia, with around 87,000 children developing tuberculosis yearly.[4] Tuberculosis can lead to decreased appetite, nutrient malabsorption, and altered metabolism, which leads to wasting. On the other hand, malnutrition also renders patients prone to tuberculosis due to lymphoid tissue atrophy, alterations in T-cell functions and associated cytokines, and potentiation of further immunosuppression from the production of transforming growth factor-beta (TGF-β).[5]

Refeeding syndrome is a fatal complication that can occur in severely

malnourished patients during nutritional reintroduction. A study conducted in South Africa stated that the incidence of refeeding syndrome in children with severe acute malnutrition was 8.7% even though the WHO guidelines for managing severe acute malnutrition had been implemented. Furthermore, this study also states that the incidence of refeeding syndrome is significantly related to electrolyte disturbances in patients when admitted to the hospital, such as hypophosphatemia and hypokalemia. In patients who experienced refeeding syndrome, it was also found that edema conditions before hospital admission were more common compared to those who did not, although this was not statistically significant.[6] In our case, the patient was severely malnourished. There was edema on his legs when he was admitted to the hospital. Based on the McLaren score, it was found that the patient had edema, and the initial serum albumin was 2.8 g/dl with a total score of 6, so it could be concluded that the patient had marasmic kwashiorkor malnutrition. However, serum electrolytes were not evaluated at admission due to laboratory limitations.

After stabilization with WHO-recommended nutritional support, the patient received formula F75 at 450 kcal/day (approximately 48% of target energy), consistent with safe initiation. By day five, during transition to higher calorie feeding, the patient developed peripheral edema, abdominal distension, and demonstrated biochemical abnormalities: hypokalemia (2.7 mmol/L), hypernatremia (153.3 mmol/L), and worsening hypoalbuminemia (2.1 g/dL). These findings raised a strong suspicion of refeeding syndrome.

The diagnosis of refeeding syndrome in this patient is supported by a combination of clinical, biochemical, and temporal findings that align with the ASPEN consensus criteria, after careful exclusion of alternative causes of clinical deterioration. While progression of pulmonary tuberculosis was initially considered, there was no evidence of worsening respiratory symptoms or systemic inflammatory response during the critical period. Similarly, deteriorating malnutrition was unlikely given the temporal correlation between nutritional advancement and the onset of edema and weight gain, which reversed upon stabilization of nutritional intake and electrolyte correction. Cumulative fluid balance over the first seven days showed only mild to moderate positive values, without signs of fluid overload such as respiratory distress or hepatomegaly, and thus did not support iatrogenic fluid overload as a primary cause. The development of peripheral edema, abdominal distension, and significant hypokalemia (25% decrease) on day five, following transition to higher caloric feeding, and subsequent improvement after reduction in caloric intake and electrolyte correction, strongly indicates refeeding syndrome as the most plausible explanation.[7-9]

Based on ASPEN consensus recommendations regarding refeeding syndrome, the diagnostic criteria for refeeding syndrome are a decrease of either 1, 2, or 3 of serum phosphorus, potassium, and/or magnesium levels by 10%-20% (mild RS), 20%-30% (moderate RS) or >30% and/or organ dysfunction which is the result of a decrease in one of these chemicals and/or caused by a deficiency of thiamin (vitamin B1) (severe RS) and occurs within the first 5 days of reinitiating or substantially increasing energy provision.[7] In this case, we found the patient had hypokalemia with a decrease in serum potassium of 25%; moreover, we also found signs and symptoms of organ dysfunction such as weakness, dyspnea, hematological abnormalities (anemia), and fluid retention (edema). Based on these criteria, in this case, the patient meets the criteria for severe refeeding syndrome.

Recent studies have reported a notably high incidence of pediatric refeeding syndrome, particularly during the transition from stabilization to rehabilitation in severely malnourished children. In a prospective cohort of 115 children aged 6–59 months at Mulago Hospital, Uganda, the cumulative incidence of refeeding syndrome, defined as a drop in serum phosphorus of more than 0.3 mmol/L, was 34.8%, with low baseline serum sodium and the presence of edema being significant risk factors.[10] Similarly, a retrospective analysis in South Africa found a refeeding syndrome incidence of 8.7% among infants with severe acute malnutrition, with admission hypophosphatemia, hypokalemia, and hyponatremia significantly associated with its development.[11] In contrast, a study in pediatric intensive care units in France reported that among 199 undernourished critically ill children, 46.7% of those at risk developed probable refeeding syndrome per the ASPEN criteria, with the majority classified as severe.[12] These findings echo our case in several aspects: (1) the timing of manifestations during the transition phase; (2) the presence of electrolyte derangements and edema; and (3) classification as severe refeeding syndrome. However, our case differs in being a single-case report with limited phosphate data due to resource constraints, in contrast to the cohort-level analyses. This underlines the value of case-level data for recognizing refeeding syndrome in resource-limited settings and emphasizes that refeeding syndrome remains a critical and under-recognized complication across diverse pediatric populations and health systems.

One of the significant limitations encountered in this case was the inability to assess serum phosphate levels due to the unavailability of testing in our resource-limited hospital setting. Phosphate is a central biochemical marker in the diagnosis of refeeding syndrome, and hypophosphatemia is often the earliest and most specific laboratory indicator of this condition, as outlined in the ASPEN consensus recommendations.[7] The absence of phosphate testing inevitably constrained our ability to fulfill the full diagnostic criteria. However, in such contexts, clinicians must adopt a syndromic approach, integrating other suggestive biochemical abnormalities such as hypokalemia and hypoalbuminemia with the clinical course, particularly deterioration that coincides with initiation of nutritional therapy. In this patient, the close temporal association between increased caloric intake and the recurrence of edema, alongside a significant drop in serum potassium and signs of organ dysfunction, provided sufficient clinical evidence to support the diagnosis of refeeding syndrome despite the missing phosphate data.

In similar low-resource environments, practical alternatives include: (1) identifying high-risk patients using clinical and anthropometric markers (e.g., weight-for-length < -3 SD, presence of edema, chronic illness), (2) initiating conservative caloric refeeding at ≤50% of energy targets, as recommended by WHO and ASPEN guidelines, and (3) presumptively supplementing phosphate and thiamine when laboratory monitoring is unavailable, especially in severely malnourished children.[7,13,14] In such scenarios, frequent bedside monitoring of weight gain, hydration status, neuromuscular signs, and vital signs remains critical. This pragmatic, evidence-informed approach allows early recognition and management of refeeding syndrome even in settings with diagnostic constraints.

There are several criteria according to the ASPEN consensus to detect pediatric patients at risk of refeeding syndrome, namely weight-for-age z score (age 1-24 months) or BMI-for-age z score (age 2-20 years), degree of weight loss, energy intake, abnormal concentrations of potassium, phosphate, and magnesium before nutrition, presence of high-risk diseases, loss of subcutaneous fat, and loss of muscle mass.[7] Based on these criteria, in this case, it can be concluded that the patient has a significant risk of refeeding syndrome. Criteria that meet include patients with a weight-for-length z score of -5.16 SD, mid-upper arm circumference z score of -3.6 SD, decreased potassium concentration 25% below the normal limit, as well as the presence of high-risk comorbidities, namely failure to thrive in the patient.

In its consensus recommendations, ASPEN also explains the avoidance and management of refeeding syndrome in at-risk pediatric patients, which is divided into several aspects of care. The first aspect is when starting nutrition. Nutrition starts with a maximum of 40%-50% of the calorie target, but usually starts the glucose infusion rate at around 4-6 mg/kg/minute and increases it by 1-2 mg/kg/minute per day if needed until it reaches a maximum of 14-18 mg/kg/minute, including enteral and parenteral glucose. The second aspect is electrolytes. In this aspect, ASPEN recommends checking the serum electrolytes potassium, magnesium, and phosphorus before starting nutrition. Then monitor every 12 hours for the first 3 days in high-risk patients and more frequently based on the patient's clinical picture. Electrolyte correction is also carried out based on existing standards. There are no specific recommendations regarding the dose of prophylactic electrolytes that should be given if serum electrolyte levels were previously normal. If electrolytes are difficult to correct or decrease drastically when starting nutrition, reduce calories/grams of dextrose by 50% and increase dextrose/calories by around 33% of the goal every 1-2 days based on the patient's clinical condition. Recommendations can be adjusted based on the doctor's assessment and consideration, and discontinuation of nutritional support may be considered if electrolyte levels are severely and/or life-threateningly low or dropping precipitously.[7]

The next aspect is thiamine and multivitamins. Thiamine is administered at 2 mg/kg up to a maximum of 100-200 mg/day before starting nutrition or before administering intravenous fluids containing dextrose in high-risk patients. Thiamine supplementation is given for 5-7 days or more in patients with severe starvation, chronic alcoholism, or other high risk for deficiency and/or signs of thiamine deficiency. Regular checking of thiamine levels is not necessary. Injectable multivitamins are added to daily parenteral nutrition, unless there are contraindications, as long as parenteral nutrition is still being given. In patients receiving oral and enteral nutrition, add a complete oral/enteral multivitamin once per day for 10 days or more based on the patient's clinical condition. If the patient is in the adult weight range, then refer to the recommended adult multivitamin. The final aspect is monitoring and long-term care. It is recommended to check vital signs every 4 hours for the first 24 hours after the start of nutrition in at-risk patients. Cardiorespiratory monitoring is recommended in unstable patients or patients with severe deficiencies based on established standards of care. Check the patient's body weight daily by monitoring intake and output. Calculate the energy requirements needed to administer oral nutrition to the patient. Evaluate short-term and long-term goals for providing daily nutrition for the first few days until the patient is considered stable and based on the service standards of each institution.[7]

In this case, we started giving the patient calories with 450 kcal/day which is equivalent to 48% of the patient's target calories. However, in patients, we still find the condition of refeeding syndrome even though we have given low calories and carefully, this could be because the patient is a population at significant risk for refeeding syndrome. After we concluded that the patient had refeeding syndrome, we returned the patient to the stabilization phase and reduced the calories to 48% of the target calories. We did not carry out an initial electrolyte examination because there was no serum electrolyte examination available at the hospital. Serum electrolyte examination at an external hospital could only be carried out on the 5th day of treatment after refeeding syndrome was suspected in the patient. The results of the electrolyte examination showed hypokalemia in the patient. We corrected the hypokalemia and the results of the potassium examination after correction improved.

We also provided thiamine and multivitamin supplementation to patients before the start of providing nutrition to patients. We administered vitamin B1 (thiamine) at a dose of 10 mg once daily for 7 days following the diagnosis of refeeding syndrome, along with a multivitamin supplement for 14 days, in accordance with established clinical guidelines based on the guidebook for the prevention and management of malnutrition in toddlers published by the Indonesian Ministry of Health. However, the detailed dosage that must be given was not stated. This thiamine administration is still slightly below the recommended thiamine dose for refeeding syndrome patients according to ASPEN consensus (2 mg/kg/day). The dosage was determined with the consideration that the patient's body weight during the onset of refeeding syndrome was not an accurate representation of his true weight, as it was confounded by fluid retention secondary to edema. We also monitored the patient's

vital signs, fluid balance, and weight increment every day in accordance with recommendations from ASPEN.

In this patient, we also found a very severe anemia condition. We gave packed red cells transfusions to patients with 5 mL/kg in the first hour, and the rest was spent 3 hours later with a total blood dose not exceeding 15 mL/kg/day. After the transfusion, the patient's hemoglobin rose to 8 gr/dl. From examination of the peripheral blood smear, it was shown that the patient had microcytic hypochromic anemia which could be caused by iron deficiency. However, the patient was not tested for serum iron, TIBC, and ferritin due to limited laboratory tests. This condition may be worsened by the presence of *Ascaris lumbricoides* infection found in the patient. This parasitic infection can interfere with the patient's absorption of nutrients, including iron. This was further exacerbated by the condition of malnutrition in patients which was also caused by inadequate nutritional intake and the presence of comorbidities in patients. A study conducted in Indonesia stated that there was a relationship between single soil-transmitted helminth infection and the incidence of stunting and anemia in preschool children. *Ascaris lumbricoides* will absorb nutrients from the host's intestinal lumen, which in moderate to severe infections will cause anemia and malnutrition.[15,16]

This patient also had mild hypoalbuminemia on initial admission with an albumin of 2.8 g/dl. However, on the 5th day of treatment, we found that the patient's pedal edema had returned, so we did another albumin examination and found that the albumin had dropped to 2.1 g/dl. We gave a transfusion of 30 ml of 20% albumin, but we still got post-correction albumin at 2.1 g/dl. We administered a second albumin correction with the same amount. From the results of the second albumin correction, it was found that albumin increased to 2.3 g/dl and the patient's edema decreased. Hypoalbuminemia often occurs in children with malnutrition; this may be due to decreased albumin synthesis. Moreover, malnutrition can also cause other comorbid conditions, such as infections, which can worsen the illness and ultimately contribute to hypoalbuminemia. Hypoalbuminemia occurs more frequently in patients with kwashiorkor than marasmus. Several mechanisms can cause hypoalbuminemia in malnutrition, including adaptation to nutritional deficiencies, hormonal responses to stress, and the release of pro-inflammatory cytokines. Improved nutrition can improve hypoalbuminemia conditions in patients with protein-energy malnutrition through the mechanism of reducing albumin catabolism. However, patients with kwashiorkor and marasmus can still experience hypoalbuminemia even years after their nutritional status has improved, which can be caused by several factors, including the degree of malnutrition and the patient's liver synthesis ability.[17] We gradually increase the calorie target and nutritional intake. The patient's condition improved, and the patient's weight increased. The patient was sent home on the 26th day of treatment and then continued to outpatient services.

CONCLUSION

Healthcare providers could use the latest consensus recommendations regarding refeeding syndrome from ASPEN to help make a diagnosis quickly, even in limited facilities. The occurrence of complications of refeeding syndrome must be watched out for, especially in high-risk patients. Quick recognition and appropriate treatment of this complication are significant so that fatal consequences do not occur in malnourished patients who are receiving nutritional therapy. Careful nutritional administration, correction and evaluation of serum electrolytes, thiamine and multivitamin supplementation, and evaluation of the patient's clinical and vital signs are important aspects in the prevention and management of refeeding syndrome.

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CONFLICT OF INTEREST

None declared.

PATIENT CONSENT

The authors confirm that written informed consent was obtained from the patient's parent for publication of this case report, including any clinical details and images.

AUTHORS CONTRIBUTIONS

All authors contributed equally to the conception, design, data collection, analysis, drafting, and critical revision of the manuscript. All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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None.

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