



NUTRITIONAL MANAGEMENT OF CLINICALLY EVIDENT NUTRITIONAL DEPLETION DESPITE NORMAL BMI IN A PATIENT WITH DYSPHAGIA AND ACUTE KIDNEY DYSFUNCTION: A CASE REPORT

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ABSTRACT

Background:

Malnutrition in hospitalized patients may remain unrecognized when nutritional assessment relies primarily on body mass index (BMI). Nutritional management becomes particularly challenging when reduced nutritional intake coexists with dysphagia, acute illness, and kidney dysfunction. This case describes the nutritional management of a patient with clinically evident nutritional depletion despite a normal-range BMI in the setting of multiple medical and nutritional challenges.

Case presentation:

A 57-year-old man was admitted with severe pneumonia, hypovolemia with hypernatremia, acute kidney injury with a differential diagnosis of acute-on-chronic kidney disease, neurological impairment, and flaccid paraplegia. He had experienced markedly reduced food intake for approximately one

month because of generalized weakness, cough, and dysphagia. Nutritional assessment estimated a body weight of 55 kg and BMI of 20.95 kg/m². Despite a normal-range BMI, nutrition-focused physical examination demonstrated moderate muscle wasting and loss of subcutaneous fat. Nasogastric enteral nutrition was initiated at approximately 15 kcal/kg/day and progressively advanced toward an estimated target of 1,650 kcal/day. Protein provision was initially 1.0 g/kg/day and subsequently reduced to 0.6 g/kg/day in the context of kidney dysfunction. Thiamine supplementation, potassium correction, and monitoring of feeding tolerance, renal function, fluid status, and electrolytes were performed during nutritional advancement.

Outcome:

Estimated energy intake increased from approximately 600 kcal/day at the initial assessment to 1,387 kcal/day by nutrition day 6, corresponding to approximately 84% of the estimated target. Transient nausea, vomiting, and epigastric discomfort occurred during nutritional advancement but subsequently improved. Urine output increased to approximately 1,400 mL/24 hours, and hypovolemia was documented as resolved. However, objective reassessment of body weight, muscle mass, and functional capacity was unavailable during the short hospitalization.

Conclusion:

This case illustrates that clinically relevant nutritional depletion may occur despite a normal-range BMI. Comprehensive nutritional assessment and individualized enteral nutrition were important in addressing markedly reduced intake and dysphagia while accounting for kidney dysfunction and fluid-electrolyte disturbances. Nutritional management required progressive

reassessment of energy and protein provision, feeding tolerance, and biochemical status rather than reliance on BMI or kidney function alone.

Keywords: Malnutrition; Nutritional assessment; Muscle wasting; Enteral nutrition; Dysphagia; Acute kidney injury; Refeeding risk.

INTRODUCTION

Disease-related malnutrition is a common clinical problem among hospitalized medical patients and is associated with increased morbidity, impaired recovery, and other adverse clinical outcomes. Early identification and individualized nutritional support are therefore important components of the management of hospitalized patients at nutritional risk. The European Society for Clinical Nutrition and Metabolism (ESPEN) recommends timely nutritional screening followed by individualized nutritional assessment and intervention in polymorbid medical inpatients.¹

Importantly, malnutrition may not be adequately recognized by body mass index (BMI) alone. The Global Leadership Initiative on Malnutrition (GLIM) diagnostic framework incorporates three phenotypic criteria—non-volitional weight loss, low BMI, and reduced muscle mass—and two etiologic criteria—reduced food intake or assimilation and disease burden/inflammation. A diagnosis requires at least one phenotypic and one etiologic criterion. The 2025 GLIM update further emphasizes that assessment of reduced muscle mass should be adapted to available clinical resources and that clinical judgment may contribute to the assessment of disease burden and inflammation.²

Nutritional management becomes particularly challenging when nutritional depletion occurs in the setting of acute illness and impaired kidney function. Patients hospitalized with acute kidney injury (AKI), acute kidney disease, or chronic kidney disease have heterogeneous metabolic characteristics and nutritional requirements. The current ESPEN practical guideline recommends an individualized approach incorporating nutritional assessment, regular reassessment of nutritional requirements, appropriate selection of the feeding route, and monitoring to avoid both underfeeding and overfeeding.³ In this context, nutritional rehabilitation may be further complicated by dysphagia, impaired oral intake, fluid and electrolyte disturbances, and the need to balance nutritional requirements with renal considerations.

We present a case of a 57-year-old man with severe acute illness, markedly reduced oral intake, dysphagia, clinically evident muscle wasting despite a normal-range BMI, kidney

dysfunction, and hypovolemic dehydration with hypernatremia, who required nasogastric enteral nutrition. Nutritional therapy was progressively advanced with concurrent monitoring of feeding tolerance, renal function, fluid status, and electrolytes, while the protein prescription was adjusted during hospitalization. This case highlights the importance of comprehensive nutritional assessment beyond BMI and the challenges of individualized nutritional support in an acutely ill patient with concomitant kidney dysfunction and electrolyte disturbances.

CASE PRESENTATION

A 57-year-old man was admitted to the Regional General Hospital of Klungkung, Bali, Indonesia, after being previously hospitalized at another hospital since 7 September 2026. He was referred with progressive generalized weakness for approximately one month, which had worsened during the preceding week. During this period, he became increasingly dependent and remained bedridden for approximately one month. He also reported pain in both upper and lower extremities, cough, and reduced oral intake. During the week before referral, he reportedly developed difficulty speaking and subsequently continued to experience difficulty swallowing. At evaluation in the emergency department, he was conscious, able to follow commands, and able to speak, although swallowing difficulty persisted.

The initial medical assessment documented severe pneumonia (CURB-65 score 4), a post-shock state suspected to be secondary to hypovolemia, with septic shock as a differential diagnosis, severe dehydration with hypernatremia, acute kidney injury with a differential diagnosis of acute-on-chronic kidney disease in the setting of stage IV chronic kidney disease, communicating hydrocephalus, flaccid paraplegia, small-vessel cerebrovascular disease, muscle wasting, and intercritical gouty arthritis. He was initially managed with intravenous fluid therapy and antimicrobial treatment, while nutritional support was subsequently initiated and monitored by the clinical nutrition team.

At the initial nutritional assessment, the patient reported markedly reduced food intake for approximately one month. During the first two weeks of illness, he consumed only approximately

2–3 tablespoons of rice per meal, followed by approximately 2–3 tablespoons of porridge per meal during the subsequent two weeks. Oral intake remained inadequate because of weakness, cough, and difficulty swallowing, and enteral nutrition through a nasogastric tube (NGT) was therefore used. Approximately 600 kcal/day was being delivered through enteral feeding at the initial assessment. The patient also reported weight loss, although the amount could not be quantified because previous body-weight measurements were unavailable.

Anthropometric assessment estimated body weight at 55 kg and height at 162 cm based on a knee height of 50 cm, corresponding to a BMI of 20.95 kg/m². Mid-upper arm circumference was 26 cm, and estimated ideal body weight was 55.8 kg. Despite the BMI being within the conventional normal range, nutrition-focused physical examination demonstrated moderate muscle wasting (+2) and loss of subcutaneous fat (+2), without peripheral edema or ascites. The patient was bedridden, with bowel sounds present and no NGT feeding residue documented. These findings were recorded by the clinical nutrition team as protein-energy malnutrition.

Initial laboratory investigations on 11 September 2026 showed hemoglobin 10.3 g/dL, leukocyte count $14.13 \times 10^3/\mu\text{L}$, neutrophils 91%, urea 197 mg/dL, creatinine 1.3 mg/dL, sodium 149 mmol/L, potassium 3.8 mmol/L, chloride 107 mmol/L, AST 57 U/L, ALT 26 U/L, and random blood glucose 148 mg/dL. During hospitalization, urea and creatinine increased to 215 mg/dL and 1.6 mg/dL, respectively, on 14 September. Serum sodium subsequently increased to 150 and then 155 mmol/L, while potassium decreased to 2.8 mmol/L before partially improving to 3.4 mmol/L. Chloride increased to 111 mmol/L.

Nutritional support was initiated cautiously through the NGT. The initial estimated target was 1,650 kcal/day and 55 g protein/day (1.0 g/kg/day). Enteral energy provision was progressively increased during nutritional follow-up, while protein prescription was subsequently reduced to 0.6 g/kg/day in the context of kidney dysfunction. The enteral formula was changed from Peptibren to a renal-specific formula, and thiamine supplementation and potassium correction were provided. During nutritional advancement, the patient experienced transient episodes of nausea, vomiting, and epigastric discomfort, which subsequently improved. By nutrition day 6, estimated energy intake had increased to 1,387 kcal/day, representing approximately 84% of the estimated target, with no

further nausea or vomiting. Urine output was approximately 1,400 mL/24 hours, and hypovolemia was documented as resolved.

Table 1. Baseline Clinical and Nutritional Characteristics at Initial Nutritional Assessment

Domain	Parameter	Finding
Patient characteristics	Age, years	57
	Sex	Male
	Functional status	Bedridden for approximately 1 month
Nutritional history	Duration of reduced intake	Approximately 1 month
	Usual intake	First 2 weeks: 2–3 tablespoons of rice per meal Next 2 weeks: 2–3 tablespoons of porridge per meal
	Reported weight loss	Present, not quantitatively measured
	Feeding route at assessment	Nasogastric tube (NGT)
Anthropometry and nutrition-focused physical examination	Estimated body weight, kg	55
	Estimated height, cm	162
	Body mass index, kg/m ²	20.95 (normal range)
	Mid-upper arm circumference, cm	26
	Muscle wasting (clinical assessment)	+2 (moderate)
	Loss of subcutaneous fat (clinical assessment)	+2 (moderate)
	Edema	Absent
	Ascites	Absent
Disease-related factors	Major acute illness	Severe pneumonia (CURB-65 score 4)
	Hemodynamic status	Post-shock state suspected to be secondary to hypovolemia, with septic shock as a differential diagnosis
	Neurological condition	Communicating hydrocephalus, flaccid paraplegia, small-vessel cerebrovascular disease, dysphagia
	Kidney dysfunction	Acute kidney injury with a differential diagnosis of acute-on-chronic kidney disease in the setting of stage IV chronic kidney disease
	Fluid and electrolyte disturbance	Severe dehydration with hypernatremia
	Other conditions	Intercritical gouty arthritis
Initial nutritional prescription	Energy target	1,650 kcal/day ($\approx$ 30 kcal/kg/day)
	Protein target	55 g/day ($\approx$ 1.0 g/kg/day)
	Enteral formula	Peptibren®

Note: Body weight and height were estimated. Clinical assessment of muscle wasting and loss of subcutaneous fat was performed using standardized nutrition-focused physical examination and graded as +2 (moderate).

Nutritional Assessment

Nutritional assessment identified clinically evident nutritional depletion despite a BMI within the conventional normal range. The patient had a history of markedly reduced food intake for

approximately one month, with intake reduced to approximately 2–3 tablespoons of rice per meal during the first two weeks and 2–3 tablespoons of porridge per meal during the subsequent two weeks. At the time of nutritional assessment, approximately 600 kcal/day was being provided through nasogastric enteral feeding. Nutrition-focused physical examination (NFPE) demonstrated moderate muscle wasting (+2) and loss of subcutaneous fat (+2), while edema and ascites were absent. The patient was also bedridden and had substantial functional dependence. The documented muscle wasting provided clinical evidence of nutritional depletion and served as a clinical surrogate for the GLIM phenotypic criterion of reduced muscle mass.

The nutritional findings were considered in the context of the Global Leadership Initiative on Malnutrition (GLIM) diagnostic framework. GLIM requires at least one phenotypic criterion and one etiologic criterion for a diagnosis of malnutrition. The phenotypic criteria include non-volitional weight loss, low BMI, and reduced muscle mass, whereas the etiologic criteria include reduced food intake or assimilation and disease burden/inflammation.² In this patient, the reported weight loss could not be quantified because previous body-weight measurements were unavailable. His BMI was 20.95 kg/m², which does not fulfill the GLIM low-BMI criterion for an Asian adult younger than 70 years, for whom the suggested cutoff is <18.5 kg/m².² The patient nevertheless had a phenotypic finding consistent with reduced muscle mass based on the NFPE findings described above. He also clearly fulfilled the reduced food intake criterion, with markedly inadequate intake persisting for approximately one month, and had a substantial acute disease burden, including severe pneumonia and neurological disease.²

Taken together, the combination of a phenotypic finding suggestive of reduced muscle mass and etiologic criteria of markedly reduced food intake and acute disease burden supported the clinical diagnosis of protein-energy malnutrition documented by the clinical nutrition team. However, the severity of malnutrition was not formally graded according to GLIM because the degree of weight loss was unavailable and muscle mass was assessed clinically rather than quantitatively. The 2025 GLIM update emphasizes that assessment of reduced muscle mass should be adapted to available clinical expertise and resources, while recognizing the limitations of clinical assessment compared with objective body-composition techniques.²

Nutritional Intervention and Follow-up

Nutritional support was initiated through a nasogastric tube (NGT) because oral intake remained inadequate in the setting of persistent dysphagia and generalized weakness. The initial estimated nutritional target was 1,650 kcal/day (30 kcal/kg/day) and 55 g protein/day (1.0 g/kg/day). Given the patient's prolonged markedly reduced intake and clinically evident muscle wasting, enteral feeding was initiated at approximately 750 kcal/day (15 kcal/kg/day) using Peptibren, with six feeds of 30 g diluted in 100 mL per feeding. Nutritional intake, gastrointestinal tolerance, vital signs, functional status, and NGT residue were monitored during the subsequent follow-up. The initial low-calorie approach was consistent with the clinical concern for nutritional complications during reintroduction of nutrition in a patient with prolonged inadequate intake.

On nutrition day 3 (14 September 2026), the previous 24-hour enteral intake was approximately 750 kcal. One episode of vomiting containing yellowish fluid was reported and was associated with coughing. Enteral nutrition was subsequently advanced to approximately 1,100 kcal/day (20 kcal/kg/day) using Peptibren 45 g six times daily in 175 mL per feeding. A small oral trial of half a portion of rice-flour porridge (*bubur sumsum*) was also introduced, while NGT feeding remained the primary route of nutritional support. Repeat blood urea nitrogen and serum creatinine measurements were planned.

On nutrition day 4 (15 September 2026), the previous 24-hour intake was approximately 1,100 kcal. The patient had experienced one episode of vomiting containing milk. Urine output was approximately 1,000 mL/24 hours and was described as concentrated. At this assessment, the documented protein target was reduced from 1.0 g/kg/day to 0.6 g/kg/day (33 g/day), while the energy target remained 1,650 kcal/day. The enteral formula was changed from Peptibren to Nephrisol, initially providing approximately 1,300 kcal/day (24 kcal/kg/day). Potassium correction was performed according to the treating medical team, and thiamine 100 mg twice daily was administered 30 minutes before feeding.

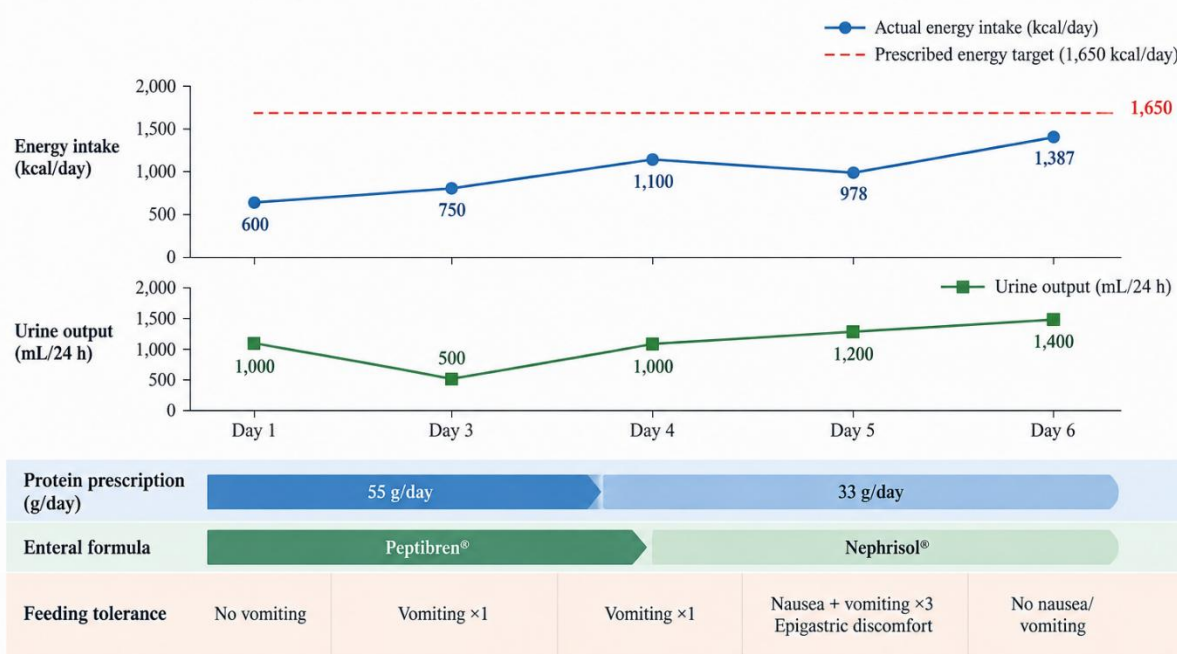
On nutrition day 5 (16 September 2026), the previous 24-hour intake was approximately 978 kcal, with the patient receiving Peptibren 30 g three times and Nephrisol 45 g three times through the NGT. The patient developed nausea, three episodes of watery vomiting, and epigastric

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discomfort. Urine output was approximately 1,200 mL/24 hours. Serum sodium was 155 mmol/L, potassium 3.4 mmol/L, and chloride 111 mmol/L. Enteral nutrition was advanced to approximately 1,500 kcal/day (27 kcal/kg/day) using Nephrisol 55 g six times daily in 200 mL per feeding. Potassium correction and thiamine supplementation were continued, with ongoing monitoring of intake, symptoms, vital signs, functional status, and NGT residue.

By nutrition day 6 (17 September 2026), the previous 24-hour enteral intake had increased to approximately 1,387 kcal/day, corresponding to approximately 84% of the estimated energy target. The patient was receiving Nephrisol 45 g twice and 55 g four times daily, with 200 mL per feeding through the NGT. No nausea or vomiting was reported. Urine output increased to approximately 1,400 mL/24 hours, and the medical record documented resolution of hypovolemia. The nutritional plan was to continue Nephrisol at 61 g six times daily in 230 mL per feeding, corresponding to the documented nutritional target.

Figure 1. Nutritional progression, feeding tolerance, and fluid status during hospitalization



Note: Actual energy intake refers to the amount of enteral nutrition delivered in the previous 24 hours. The prescribed energy target was 1,650 kcal/day. Protein prescription was 55 g/day on Days 1–3 and 33 g/day on Days 4–6. Enteral formula was changed from Peptibren® to Nephrisol® on Day 4.

Outcome

During nutritional follow-up, energy delivery progressively increased from approximately 600 kcal/day at the initial assessment to 1,387 kcal/day by nutrition day 6, corresponding to approximately 84% of the estimated energy target of 1,650 kcal/day. Enteral nutrition through the NGT remained the primary route of nutritional support because swallowing difficulty persisted. Although transient nausea, vomiting, and epigastric discomfort occurred during nutritional advancement, feeding tolerance improved by the final nutritional assessment, with no further nausea or vomiting reported.

Urine output increased from approximately 1,000 mL/24 hours at the initial nutritional assessment to 1,400 mL/24 hours on nutrition day 6. The medical record documented that hypovolemia had resolved by 17 September 2026. The patient remained bedridden and continued to report generalized weakness, pain in the extremities, cough, and difficulty swallowing at the final assessment. No objective reassessment of body weight, muscle mass, or functional capacity was available during the hospitalization. Therefore, nutritional recovery could not be quantitatively established. On 17 September 2026, discharge planning was initiated, with follow-up arranged at the neurology and internal medicine clinics.

DISCUSSION

Malnutrition Despite a Normal-Range BMI

This case illustrates that a normal-range body mass index (BMI) does not exclude clinically relevant nutritional depletion in hospitalized adults. Although the patient's BMI was 20.95 kg/m², nutritional assessment demonstrated a markedly reduced dietary intake for approximately one month, together with clinically evident muscle wasting and loss of subcutaneous fat. These findings are clinically important because BMI provides information about body weight relative to height but does not adequately characterize body composition or distinguish between preserved adipose tissue and loss of lean tissue. In patients with acute or chronic kidney disease, this limitation may be further accentuated by alterations in fluid status, which can obscure changes in nutritional status when body weight is interpreted in isolation.^{2,3}

The Global Leadership Initiative on Malnutrition (GLIM) framework emphasizes that malnutrition should be diagnosed using both phenotypic and etiologic criteria rather than BMI alone. Phenotypic criteria include non-volitional weight loss, low BMI, and reduced muscle mass, while etiologic criteria include reduced food intake or assimilation and disease burden or inflammation.² In the present case, the patient had prolonged and markedly reduced food intake and a substantial acute disease burden, while physical examination demonstrated muscle wasting despite a BMI within the conventional normal range. However, the absence of a quantitatively measured weight loss and objective body-composition assessment limited formal characterization of the severity of malnutrition. This case therefore reinforces the importance of comprehensive nutritional assessment, particularly in patients whose BMI may appear reassuring despite evidence of nutritional depletion.

Dysphagia and the Need for Enteral Nutrition

Dysphagia was an important contributor to the patient's inadequate oral intake. The patient had neurological abnormalities, including communicating hydrocephalus, flaccid paraplegia, and small-vessel cerebrovascular disease, and continued to experience difficulty swallowing during hospitalization. At the initial nutritional assessment, oral intake was markedly reduced and enteral feeding through a nasogastric tube (NGT) was already being used. In patients with neurological disorders, dysphagia may impair the ability to consume sufficient food and fluids and may consequently increase the risk of nutritional and hydration deficits.⁴

In this case, the decision to maintain enteral nutrition was clinically relevant because oral intake was insufficient to meet the patient's nutritional requirements and swallowing difficulty persisted. The ESPEN guideline on clinical nutrition in neurology recommends enteral nutrition when adequate oral intake cannot be achieved in patients with neurological conditions and prolonged dysphagia. In the acute phase of stroke with insufficient oral intake, nasogastric feeding is recommended as the preferred enteral route.⁴ Although the precise etiology and severity of dysphagia in this patient were not formally characterized using instrumental swallowing assessment, the combination of persistent swallowing difficulty, markedly reduced oral intake, and neurological impairment provided a clinical rationale for continued NGT feeding.

A small oral trial was subsequently documented on nutrition day 3, while NGT feeding remained the primary source of nutrition. However, because no formal swallowing evaluation was documented, the safety of oral intake and the patient's aspiration risk could not be objectively assessed. This represents an important limitation of the nutritional assessment in this case and supports the need for formal swallowing assessment when clinically indicated in patients with persistent neurogenic dysphagia.

Stepwise Nutritional Rehabilitation and Refeeding Risk

The patient's prolonged and markedly reduced nutritional intake, together with clinically evident muscle and subcutaneous fat loss and severe acute illness, raised concern for complications associated with the reintroduction of nutrition. According to the American Society for Parenteral and Enteral Nutrition (ASPEN) consensus recommendations, prolonged inadequate energy intake and loss of muscle mass or subcutaneous fat are among the characteristics associated with increased risk of refeeding syndrome (RS). The patient had consumed very small amounts of food for approximately one month before nutritional assessment and had documented muscle wasting and loss of subcutaneous fat, supporting the need for cautious nutritional rehabilitation.⁵

In this case, enteral nutrition was initiated at approximately 750 kcal/day (15 kcal/kg/day) and progressively advanced to 1,100, 1,300, and 1,500 kcal/day before reaching an actual intake of approximately 1,387 kcal/day on nutrition day 6. This stepwise approach was consistent with the principle of conservative initiation and gradual advancement recommended by ASPEN for adults at risk of RS. ASPEN recommends initiating nutrition at approximately 10–20 kcal/kg during the first 24 hours and advancing caloric provision progressively while monitoring for electrolyte abnormalities.⁵

Electrolyte monitoring was particularly relevant because the patient's serum potassium decreased from 3.8 mmol/L to 2.8 mmol/L during nutritional advancement, followed by partial correction to 3.4 mmol/L. In the context of nutritional reintroduction, this decline in serum potassium may represent a biological signal of a refeeding-related intracellular electrolyte shift. Potassium replacement was subsequently provided, and thiamine 100 mg twice daily was administered before feeding. ASPEN recommends assessment of potassium, magnesium, and

phosphorus before initiation of nutrition and close monitoring during the initial period of refeeding, together with thiamine supplementation in patients at risk.⁵

However, the available findings were insufficient to establish a diagnosis of refeeding syndrome. The ASPEN consensus definition requires a decrease in serum phosphorus, potassium, and/or magnesium occurring within five days of reinitiating or substantially increasing energy provision, with severity based on the magnitude of the electrolyte decline and/or associated organ dysfunction.⁵ In this case, phosphorus and magnesium measurements were not available, and the observed hypokalemia alone could not establish the diagnosis of RS. Therefore, the nutritional course is more appropriately described as cautious nutritional rehabilitation in a patient at risk for refeeding complications, with the observed hypokalemia interpreted as a possible refeeding-related electrolyte shift rather than definite refeeding syndrome.

Protein Prescription in the Setting of Acute Kidney Injury and Possible Chronic Kidney Disease

Protein prescription represented an important challenge during nutritional support in this patient. The medical record documented acute kidney injury with a differential diagnosis of acute-on-chronic kidney disease in the setting of stage IV chronic kidney disease. During hospitalization, serum urea increased from 197 to 215 mg/dL and creatinine from 1.3 to 1.6 mg/dL. The initial protein target was 55 g/day (1.0 g/kg/day), which was subsequently reduced to 33 g/day (0.6 g/kg/day), accompanied by a switch from Peptibren to a renal-specific enteral formula.

Protein requirements in hospitalized patients with kidney dysfunction should be determined according to the underlying clinical condition and metabolic state rather than kidney function alone. The 2024 ESPEN practical guideline recommends 0.8–1.0 g/kg/day for patients with AKI or AKI on CKD without acute or critical illness. For patients with AKI, AKI on CKD, or CKD accompanied by acute or critical illness who are not receiving kidney replacement therapy, protein intake should be initiated at approximately 1.0 g/kg/day and progressively increased toward 1.3 g/kg/day if tolerated.³

In the present case, the initial protein prescription of 1.0 g/kg/day was therefore consistent with the starting intake recommended by ESPEN for patients with kidney dysfunction and

concomitant acute or critical illness who are not receiving kidney replacement therapy. Following the increase in urea and creatinine, the documented protein target was reduced to 0.6 g/kg/day. This reduction can be interpreted as an individualized, transient, and conservative measure in response to the acute azotemic state rather than as a long-term protein target. Once hemodynamic status and urea levels have stabilized, protein intake should be titrated upward toward approximately 1.0–1.2 g/kg/day, as clinically tolerated, to support nutritional rehabilitation and minimize further muscle wasting.

This case highlights the need to balance adequate protein provision to address nutritional depletion and muscle wasting against concerns regarding nitrogenous waste accumulation. However, the optimal protein prescription cannot be determined retrospectively with certainty because pre-hospitalization renal function and the patient's baseline renal status were not fully documented.

Fluid and Electrolyte Disturbances During Nutritional Rehabilitation

Fluid and electrolyte disturbances were important considerations during nutritional support in this patient. At the initial nutritional assessment, the patient had documented hypovolemia and hypernatremia, with a serum sodium concentration of 149 mmol/L. During subsequent monitoring, serum sodium increased to 150 and then 155 mmol/L, while potassium decreased from 3.8 to 2.8 mmol/L before partially improving to 3.4 mmol/L following potassium replacement. Urine output ranged from approximately 500 to 1,400 mL/24 hours, and hypovolemia was documented as resolved by the final nutritional assessment.

Patients with AKI or CKD may develop disturbances in water and electrolyte homeostasis because of impaired renal function and the underlying acute illness. The ESPEN practical guideline emphasizes that nutritional management in these patients should be individualized according to the clinical condition, including consideration of fluid status, urine output, and electrolyte abnormalities.³ In this case, these parameters were monitored concurrently while enteral nutrition was progressively advanced.

The patient's hypernatremia occurred in the context of documented hypovolemia and markedly reduced oral intake. Therefore, the increase in serum sodium during hospitalization

should not be attributed solely to enteral nutrition. Changes in serum sodium may reflect the interaction of water balance, fluid therapy, ongoing losses, renal function, and nutritional intake. Because serial urine sodium, urine osmolality, calculated free-water deficit, and quantified ongoing or insensible losses were unavailable, the specific contribution of these factors could not be determined retrospectively.

Hypokalemia emerged during nutritional advancement and required potassium replacement. In the context of the patient's renal dysfunction and ongoing fluid and electrolyte disturbances, potassium levels were monitored and corrected according to the treating team's management.

The switch to a renal-specific enteral formula was also documented during the period of renal and electrolyte disturbances. ESPEN does not recommend routine use of renal-specific formulas in all patients with AKI or CKD but supports individualized selection according to clinical circumstances, including fluid and electrolyte considerations.³ In this case, the change in formula should therefore be interpreted as an individualized nutritional intervention rather than evidence that renal-specific formulas are universally indicated.

By the final nutritional assessment, urine output had increased to approximately 1,400 mL/24 hours, no further nausea or vomiting was reported, and hypovolemia was documented as resolved. However, because subsequent sodium and potassium measurements were unavailable, biochemical correction of the electrolyte abnormalities could not be established.

Clinical Integration and Limitations

This case illustrates the complexity of nutritional management when multiple barriers to adequate nutritional delivery coexist. The patient had markedly reduced oral intake, dysphagia, clinically evident muscle wasting, severe acute illness, hypovolemia with hypernatremia, and kidney dysfunction. These conditions required nutritional therapy to be individualized and repeatedly reassessed rather than determined by a single anthropometric or laboratory parameter.

The nutritional strategy included enteral nutrition through an NGT because oral intake was inadequate, gradual advancement of energy provision, adjustment of protein prescription in the context of kidney dysfunction, thiamine supplementation and potassium correction during nutritional advancement, and monitoring of gastrointestinal tolerance, urine output, renal

biochemical parameters, and electrolytes. This approach is consistent with the principle of individualized nutritional support and ongoing reassessment recommended by ESPEN.^{1,3}

By nutrition day 6, estimated energy intake had increased to approximately 1,387 kcal/day, corresponding to 84% of the estimated energy target, and no further nausea or vomiting was reported. Hypovolemia was also documented as resolved. However, these short-term changes should not be interpreted as complete nutritional recovery. The patient remained bedridden and continued to experience weakness and dysphagia, while objective reassessment of body weight, muscle mass, and functional capacity was unavailable.¹

Given the persistent neurological dysphagia and planned transition to outpatient care, bedside swallowing screening, such as the Gugging Swallowing Screen (GUSS), or referral to Physical Medicine and Rehabilitation/swallowing therapy should be considered to support a safe transition from NGT feeding to oral intake.

Several limitations should be acknowledged. Previous body weight was unavailable, preventing quantitative assessment of weight loss. Muscle wasting was assessed clinically rather than using objective body-composition or quantitative muscle measurements. No formal instrumental swallowing assessment was documented, limiting objective characterization of dysphagia and aspiration risk. Baseline renal function and complete longitudinal renal data were unavailable, limiting definitive distinction between AKI and acute-on-chronic kidney dysfunction. In addition, phosphorus and magnesium measurements were unavailable, preventing definitive assessment of the biochemical criteria for refeeding syndrome. Finally, the short observation period prevented assessment of longer-term changes in nutritional status, muscle mass, and functional recovery.

Despite these limitations, this case emphasizes that nutritional management in medically complex patients should be based on repeated clinical and nutritional reassessment rather than BMI or kidney function alone. The coexistence of nutritional depletion, dysphagia, acute illness, renal dysfunction, and electrolyte disturbances requires simultaneous consideration of energy and protein provision, feeding tolerance, hydration, and biochemical monitoring.

CONCLUSION

This case illustrates that clinically relevant nutritional depletion may occur despite a normal-range body mass index in a medically complex patient. Comprehensive nutritional assessment identified markedly reduced food intake, muscle wasting, and loss of subcutaneous fat in the presence of persistent dysphagia, severe acute illness, and kidney dysfunction. Nasogastric enteral nutrition was initiated cautiously and progressively advanced with monitoring of feeding tolerance, renal function, fluid status, and electrolytes. Protein prescription required individualized consideration because of the coexistence of nutritional depletion and acute kidney dysfunction. By nutrition day 6, energy intake had increased to approximately 84% of the estimated target and feeding tolerance had improved, although objective nutritional and functional recovery could not be established during the short hospitalization. This case emphasizes the importance of individualized and repeatedly reassessed nutritional support rather than reliance on BMI or kidney function alone in nutritionally vulnerable patients with acute illness.

DECLARATION

Patient Consent

Written informed consent was obtained from the patient's legal representative for publication of this case report and accompanying images.

Ethics Approval

This case report complied with the ethical requirements of the local hospital.

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