Management Goals for Asymptomatic Persistent Hypophosphatemia in Malnourished Patients with Cancer: A Case Report and Clinical Reflections

Xiaoxi Wu, Xia Zhang, Yang Ling*
Affiliated Hospital of Hebei University, Baoding, Hebei, China

Abstract: This article reports the case of a 68-year-old patient with colon cancer and severe malnutrition who was at high risk of refeeding syndrome and had persistent hypophosphatemia before and after admission, with a nadir serum phosphate level of 0.21 mmol/L. After combined intravenous and oral phosphate supplementation, the serum phosphate level was maintained at 0.4–0.5 mmol/L. The patient had no arrhythmia, decreased muscle strength, bone pain, or neurological abnormalities. This report suggests that the management goal for asymptomatic severe hypophosphatemia in malnourished patients with cancer should not be limited to rapid correction of serum phosphate; rather, attention should be paid to acute safety, tolerance of progressive nutritional advancement, and long-term etiological evaluation.

***Keywords*:** hypophosphatemia; malnutrition; cancer; refeeding syndrome; phosphate supplementation

1. Introduction

Hypophosphatemia is not uncommon among hospitalized patients, particularly in those with malnutrition, malignancy, prolonged inadequate intake, critical illness, and those receiving nutritional support. Severe hypophosphatemia may lead to muscle weakness, respiratory muscle fatigue, reduced myocardial contractility, arrhythmia, hemolysis, rhabdomyolysis, impaired consciousness, seizures, and osteomalacia [1].

However, in the context of cancer-related wasting and chronic malnutrition, symptoms associated with hypophosphatemia may be masked by fatigue, reduced activity, or manifestations of the primary disease. Some patients may lack typical clinical manifestations even when serum phosphate is markedly decreased. This article reports the management process of persistent hypophosphatemia in a patient with colon cancer and severe malnutrition and discusses the staged management goals for asymptomatic persistent hypophosphatemia.

2. Clinical Data

The patient was a 68-year-old male admitted for "colon cancer diagnosed more than 3 months ago and generalized jaundice for 1 week." He was 173 cm in height, weighed 72 kg, and had a BMI of 24.1 kg/m². Over the preceding 3 months, he had lost approximately 15 kg, and his food intake was less than 50% of his estimated requirements. Outside the hospital, his serum phosphate level in January 2026 was approximately 0.4 mmol/L. After admission, his serum phosphate level was below 0.32 mmol/L, with a nadir of 0.21 mmol/L.

His NRS 2002 score was 4, indicating nutritional risk; the Patient-Generated Subjective Global Assessment (PG-SGA) indicated severe malnutrition; and he met the NICE criteria for high risk of refeeding syndrome [2]. Phosphate supplementation was prioritized before initiation of nutritional therapy.

Intravenous sodium glycerophosphate was administered at 10 mL per dose, twice daily, providing approximately 20 mmol/day of intravenous phosphate. Meanwhile, oral sodium fructose diphosphate was given at 10 mL per dose, three times daily, providing approximately 15 mmol/day of oral phosphate, with a total daily phosphate supplementation of approximately 35 mmol. Over the subsequent 4 weeks, based on the patient's serum phosphate level, renal function, and clinical tolerance, and with reference to recommendations from reviews on phosphate replacement therapy for hypophosphatemia, the total daily phosphate supplementation was maintained at 30–40 mmol [3]. After phosphate supplementation, the patient's serum phosphate level did not rapidly return to normal and fluctuated mostly between 0.4 and 0.5 mmol/L. Enteral nutrition was then gradually initiated and increased.

During treatment, the patient reported only mild fatigue, without obvious worsening compared with admission. Neurological examination showed no reduction in deep tendon reflexes or sensory abnormalities; muscle strength assessment showed no significant decline; and there was no bone pain, convulsions, impaired consciousness, or dyspnea. Electrocardiographic monitoring showed no arrhythmia or prolongation of the QTc interval. Renal function was normal, with

no oliguria or acute kidney injury. Due to limited conditions, urinary phosphate, tubular maximum reabsorption of phosphate adjusted for glomerular filtration rate (TmP/GFR), and fibroblast growth factor 23 (FGF23) were not further evaluated.

3. Etiological Analysis of Hypophosphatemia

The hypophosphatemia in this patient may have resulted from multiple contributing factors. First, patients with cancer often have inadequate phosphate intake and absorption due to decreased appetite, impaired gastrointestinal function, or treatment-related adverse effects. In this case, the patient had a marked reduction in food intake, and inadequate intake may have been an important reason for the persistence of hypophosphatemia. Second, the patient had malignant tumor-related wasting, prolonged inadequate intake, and significant weight loss, suggesting possible depletion of systemic phosphate stores. Even with phosphate supplementation, serum phosphate may fail to normalize rapidly because of intracellular replenishment and redistribution within tissue phosphate pools [4]. Third, after phosphate supplementation, the serum phosphate level could only be maintained at a relatively low level, suggesting the possibility of persistent phosphate consumption or renal phosphate wasting. If conditions permit, further evaluation of urinary phosphate, TmP/GFR, parathyroid hormone (PTH), and FGF23 should be performed. Fourth, tumor-induced osteomalacia is usually associated with elevated FGF23 and may present with hypophosphatemia, increased urinary phosphate excretion, bone pain, fractures, and muscle weakness.

4. Management Goals and Treatment Strategies

The features of this case were markedly reduced serum phosphate without typical symptoms, failure of serum phosphate to normalize rapidly after supplementation, and the ongoing need to advance nutritional therapy. Therefore, the management goal should not simply be set as "correcting serum phosphate to the normal range as quickly as possible," but should instead adopt staged and composite goals.

4.1 Short-Term Goal: Exit from the Acute High-Risk State

When serum phosphate is below 0.32 mmol/L, prevention of acute complications related to severe hypophosphatemia should be the first priority. Short-term goals include: serum phosphate rising out of the severe hypophosphatemia range; no new-onset arrhythmia or QTc prolongation; no respiratory muscle weakness, dyspnea, or deterioration of oxygenation; no progressive muscle weakness, dysphagia, impaired consciousness, or convulsions; no evidence of rhabdomyolysis or hemolysis; relative stability of serum potassium, magnesium, and calcium; stable renal function; and no adverse reactions during phosphate supplementation, such as hyperphosphatemia, hypocalcemia, or an increased calcium-phosphate product.

Therefore, in the early stage of this case, immediate normalization of serum phosphate was not pursued blindly. Instead, maintaining serum phosphate above 0.32 mmol/L and ensuring the stability of electrocardiographic findings, respiration, muscle strength, renal function, and other electrolytes were used as short-term safety goals.

4.2 Medium-Term Goal: Ensure Tolerance of Progressive Nutritional Advancement

For malnourished patients with cancer, long-term maintenance on low caloric intake can aggravate wasting, whereas excessively rapid caloric escalation may induce refeeding syndrome. Therefore, the focus of medium-term management is to establish a safe boundary for progressive nutritional advancement. In this case, serum phosphate was stably maintained at 0.4–0.5 mmol/L, while electrocardiographic findings, muscle strength, respiratory status, and changes in other electrolytes were closely monitored. In the absence of obvious hypophosphatemia-related symptoms, arrhythmia, or renal instability, nutritional therapy was gradually advanced.

4.3 Long-Term Goal: Oral Maintenance and Etiological Evaluation

When the patient's nutritional intake gradually stabilizes and the risk of refeeding syndrome decreases, intravenous phosphate supplementation should be gradually reduced, with transition to oral phosphate supplementation and dietary intake. Long-term goals include: maintaining serum phosphate near the lower limit of normal or within the normal range; regularly monitoring serum calcium, phosphate, magnesium, PTH, vitamin D, and alkaline phosphatase; observing bone pain, muscle strength, activity capacity, and fall risk; and, when necessary, completing tests such as urinary phosphate, TmP/GFR, and FGF23 to determine whether renal phosphate wasting or FGF23-related hypophosphatemia is present.

In summary, for patients with persistent hypophosphatemia but no obvious symptoms, phosphate supplementation should not simply be increased. Instead, the causes of hypophosphatemia, nutritional intake, absorption status, medication-related factors, and renal tubular phosphate excretion should be reassessed.

5. Discussion

The therapeutic goal should shift from "correcting numerical values" to "establishing safety boundaries." The traditional approach to treating hypophosphatemia tends to focus on serum phosphate levels; however, the management of malnourished patients with cancer is more complex. On the one hand, severe hypophosphatemia may entail acute risks; on the other hand, overly rapid or excessive phosphate supplementation may lead to hypocalcemia, hyperphosphatemia, soft tissue calcification, and an increased burden on renal function, warranting particular caution in patients with unstable renal function.

At the same time, nutritional therapy should be designed in parallel with phosphate supplementation. In patients at risk of refeeding, phosphate supplementation cannot be carried out in isolation. The nutritional support strategy itself directly affects changes in serum phosphate. Clinically, attention should be paid to the following: the initial caloric intake should not be excessively high; the proportion of carbohydrates should not be increased rapidly; vitamin B1 should be supplemented before or concurrently with nutritional support; potassium, magnesium, and phosphate supplementation should be considered simultaneously; electrolytes should be closely monitored during the first 3–5 days or even for a longer period; and if serum phosphate decreases markedly again, caloric escalation should be suspended or slowed rather than simply increasing the amount of nutrition provided [5]. In the present case, nutritional support was gradually increased on the basis of phosphate supplementation, and the patient did not develop evident cardiopulmonary or neuromuscular complications, suggesting that this strategy has a certain degree of clinical rationality.

6. Conclusion

This case suggests that asymptomatic severe hypophosphatemia in malnourished patients with cancer is not a low-risk condition, but its management should not take normalization of serum phosphate as the sole objective. A more reasonable strategy is to establish staged and composite therapeutic goals: in the short term, to prevent acute cardiopulmonary and neuromuscular complications; in the medium term, to ensure the safe progressive advancement of nutritional therapy; and in the long term, to restore the ability to maintain phosphate orally and complete the etiological evaluation of hypophosphatemia. Clinical practice should shift from "correcting a laboratory value" to "maintaining functional safety and the sustainability of nutritional therapy."

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Author Bio

Xiaoxi Wu, female (born September 18,1987), Han ethnicity, native of Baoding, Hebei Province, Master's degree holder, attending physician; Research focus: Obesity.