Open Access https://www.jparathyroid.com

Journal of Parathyroid Disease

Journal of Parathyroid Disease 2026,14, e13324

DOI:10.34172/jpd.13324

Review

Postoperative hungry bone syndrome in primary hyperparathyroidism; the role of the emergency department

Arash Forouzan[1], **Majid Fattah**[1], **Mojtaba Sasani**[1*]

Abstract

Postoperative hungry bone syndrome remains a clinically significant complication following parathyroidectomy for primary hyperparathyroidism, characterized by profound and prolonged hypocalcemia, hypophosphatemia, and hypomagnesemia resulting from rapid skeletal remineralization after abrupt normalization of parathyroid hormone levels. This updated review synthesizes contemporary evidence on the pathophysiology, risk stratification, and management of hungry bone syndrome, emphasizing its increasing recognition in an era of earlier surgical intervention and advanced metabolic profiling. Key predisposing factors include severe preoperative bone disease, elevated bone turnover markers, large parathyroid adenomas, concurrent vitamin D deficiency, advanced age, and impaired renal function. Modern diagnostic approaches integrate dynamic calcium monitoring, predictive scoring models, and preoperative assessment of bone mineral density to enable proactive intervention. Recent therapeutic advances highlight the efficacy of risk-adapted supplementation protocols, combining high-dose oral calcium, active vitamin D analogs, and targeted magnesium repletion, while minimizing reliance on prolonged intravenous infusions through standardized discharge algorithms. Emerging data also underscore the role of preoperative vitamin D optimization and novel biomarkers in predicting syndrome onset and severity. Despite evolving management strategies, hungry bone syndrome continues to contribute to extended hospitalizations, increased healthcare utilization, and patient morbidity when unrecognized. This review advocates for routine preoperative risk assessment, institutionalized postoperative monitoring pathways, and personalized supplementation regimens to mitigate complications. Therefore, combining collaboration and structured patient education are equally vital for ensuring safe recovery. Additionally, future prospective studies and consensus-driven guidelines remain indispensable to find preventive strategies, optimize calcium homeostasis, and improving long-term skeletal and metabolic outcomes in patients undergoing curative parathyroid surgery.

Keywords: Parathyroid hormone, Hungry bone syndrome, Parathyroidectomy, Primary Hyperparathyroidism, Hypomagnesemia, Vitamin D, Hypocalcemia, Hypophosphatemia, Parathyroid surgery, Tetany, Cardiac arrhythmias

Please cite this paper as: Forouzan A, Fattah M, Sasani M. Postoperative hungry bone syndrome in primary hyperparathyroidism; the role of the emergency department. J Parathyr Dis. 2026;14:e13324. doi:10.34172/jpd.13324.

Introduction

Postoperative hungry bone syndrome represents a clinically significant and potentially life-threatening metabolic complication following parathyroidectomy for primary hyperparathyroidism, characterized by profound, prolonged hypocalcemia that arises from the rapid and uncontrolled remineralization of the skeleton once the chronic stimulatory effects of parathyroid hormone are abruptly withdrawn (1). Though historically more prevalent in patients undergoing surgery for secondary or tertiary hyperparathyroidism in the context of chronic kidney disease, hungry bone syndrome has increasingly been recognized as a pertinent concern in primary disease as well, particularly in populations presenting with long-standing biochemical derangement, significant skeletal involvement, or delayed diagnosis (2,3). The modern clinical view has witnessed a paradigm shift in the epidemiology of primary hyperparathyroidism, with routine calcium screening identifying asymptomatic or mildly symptomatic individuals at earlier stages (4); however, a substantial subset of patients still presents with advanced disease marked by elevated parathyroid hormone levels, markedly increased bone turnover, and radiographic evidence of osteopenia or osteoporosis, thereby retaining a meaningful risk for postoperative hungry bone syndrome (5,6). Compassion of this condition requires a comprehensive appreciation of the dynamic interplay between parathyroid hormone physiology, skeletal mineral homeostasis, and the acute metabolic shifts that occur following surgical cure, all of which should be carefully directed to prevent symptomatic hypocalcemia, prolong hospitalization, or

Received: 17 Mar. 2026, **Revised:** 20 May 2026, **Accepted:** 4 Jun. 2026, **ePublished:** 10 Jun. 2026

[1]Department of Emergency Medicine, Faculty of Medicine, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran
***Corresponding author:** Mojtaba Sasani, Email: sasani-m@ajums.ac.ir

Implication for health policy/practice/research/medical education

Postoperative hungry bone syndrome is a clinically significant complication following parathyroidectomy for primary hyperparathyroidism, characterized by profound, prolonged hypocalcemia driven by rapid skeletal remineralization. This updated review synthesizes recent advances in the pathophysiology, risk stratification, and management of hungry bone syndrome. Following surgical correction of hyperparathyroidism, abrupt parathyroid hormone withdrawal unmasks intense osteoblastic activity, accelerating calcium and phosphate deposition into previously demineralized bone. Key risk factors include severe preoperative hypercalcemia, markedly elevated alkaline phosphatase, large adenoma size, prolonged disease duration, and advanced age. Contemporary management emphasizes proactive risk assessment, early aggressive calcium and calcitriol supplementation, and rigorous biochemical monitoring. Emerging evidence also supports magnesium optimization and refined surgical strategies to mitigate severity. Despite its predictable mechanism, hungry bone syndrome remains frequently under-recognized and can cause substantial morbidity if inadequately managed.

precipitate severe complications such as tetany, seizures, arrhythmias, or prolonged intensive care requirements (1,7). This review sought to consider the necessity of individualized perioperative protocols and continued studies to strengthen patients' outcome and minimize clinical complications.

Method of the search

To identify relevant literature for this narrative review, we searched multiple databases, including PubMed, Scopus, Embase, Web of Science, EBSCO, DOAJ, and Google Scholar, using keywords such as "parathyroid hormone," "hungry bone syndrome," "parathyroidectomy," "primary hyperparathyroidism," "hypomagnesemia," "vitamin D," "hypocalcemia," "hypophosphatemia," "parathyroid surgery," "tetany," and "cardiac arrhythmias."

Pathophysiology of hungry bone syndrome

The pathophysiology of hungry bone syndrome in primary hyperparathyroidism is fundamentally rooted in the chronic excess of parathyroid hormone, which exerts a profound catabolic influence on bone by stimulating osteoclast-mediated resorption while simultaneously altering osteoblast function and disrupting the normal coupling of bone remodeling (1). Over months or years, this hormonal milieu leads to increased calcium and phosphate efflux from the skeletal matrix into the extracellular fluid, maintaining or elevating serum calcium at the expense of bone mineral density and microarchitectural integrity (1,8). When a hyperfunctioning parathyroid adenoma or hyperplastic gland is surgically excised, serum parathyroid hormone levels plummet rapidly, often within minutes to hours, effectively removing the continuous osteoclastic stimulus and abruptly shifting the bone remodeling balance toward net mineral deposition (1,2). Osteoblasts, which had been primed and proliferating under the influence of elevated parathyroid hormone, now operate in an unopposed anabolic state, aggressively extracting calcium, phosphate, and magnesium from the circulation to remineralize previously resorbed trabecular and cortical bone surfaces (2,9). This skeletal hunger for minerals is compounded by the concomitant withdrawal of parathyroid hormone's renal effects, which normally promote calcium reabsorption in the distal tubule, phosphate excretion, and activation of vitamin D; the sudden loss of these renal adaptations further exacerbates the downward trajectory of serum calcium and phosphate (1,10). Moreover, the rapid decline in parathyroid hormone unmasks or worsens underlying vitamin D insufficiency, which is highly prevalent in patients with primary hyperparathyroidism due to accelerated conversion of 25-hydroxyvitamin D to its active form and reduced intestinal calcium absorption capacity, thereby limiting the body's ability to compensate for the acute mineral deficit through dietary uptake (11,12). The clinical manifestation of hungry bone syndrome typically emerges within the first twenty-four to seventy-two hours following parathyroidectomy, though it may occasionally be delayed up to five days in milder cases, and is distinguished from transient postoperative hypocalcemia by its severity, duration, and resistance to standard replacement protocols (8). Indeed, mild and self-limiting hypocalcemia is common after neck surgery due to transient parathyroid gland manipulation or ischemia (13), hungry bone syndrome is characterized by hypocalcemia with a serum calcium level below 8.4 mg/dL that persists for more than four days in the postoperative period, and a concomitant decrease in serum phosphate and magnesium levels, reflecting the broad mineral demand of the remineralizing skeleton (14,15). Symptoms range from perioral numbness, acral paresthesias, and muscle cramps to severe neuromuscular irritability manifesting as positive Chvostek or Trousseau signs, carpopedal spasm, laryngospasm, bronchospasm, seizures, or prolonged QT intervals predisposing to ventricular arrhythmias (1,14). The diagnosis remains primarily clinical and biochemical, relying on serial monitoring of total and ionized calcium, phosphate, magnesium, and intact parathyroid hormone levels, alongside careful assessment of symptoms and response to replacement therapy (14,16). Differentiating hungry bone syndrome from simple surgical hypoparathyroidism is critical, as the latter typically resolves within days as stunned native glands recover function, whereas hungry bone syndrome persists until skeletal remineralization plateaus, which may take days to several weeks depending on the preoperative severity of bone disease (1,17). Several predictive factors have been consistently identified in recent literature as independent markers for increased susceptibility to postoperative hungry bone syndrome in primary hyperparathyroidism, with preoperative alkaline phosphatase levels emerging as one of the most reliable

biochemical indicators of high bone turnover and skeletal hunger (15). Elevated alkaline phosphatase, particularly the bone-specific isoform, correlates strongly with the extent of osteoblastic activity and the volume of bone surface available for rapid remineralization postoperatively, making it a cornerstone of risk stratification algorithms (1,18). Similarly, higher baseline intact parathyroid hormone concentrations, larger adenoma size on imaging or pathological examination, longer duration of untreated disease and lower preoperative serum calcium and vitamin D levels have all been associated with increased risk (14, 17,19). Radiographic findings such as subperiosteal bone resorption, brown tumors, or severe osteoporosis on dual-energy X-ray absorptiometry further underscore the presence of advanced skeletal involvement and predict a more pronounced postoperative mineral deficit (1,14,20).

Modulating factors of hungry bone syndrome

Recent studies have also highlighted the role of fibroblast growth factor twenty-three, sclerostin, and other bone metabolism markers in modulating the intensity and duration of hungry bone syndrome, suggesting that a multifactorial bone-parathyroid-kidney axis disruption underlies the clinical phenotype rather than parathyroid hormone withdrawal alone (1,21). The acute management of postoperative hungry bone syndrome demands a proactive, protocol-driven approach that prioritizes rapid correction of symptomatic hypocalcemia while avoiding rebound hypercalcemia or ectopic mineral deposition (17). Intravenous calcium gluconate remains the cornerstone of initial therapy for severe or symptomatic cases, typically administered as a continuous infusion titrated to maintain ionized calcium within a safe lower limit, to prevent neurological and cardiovascular complications while acknowledging that complete normalization is neither expected nor desirable during the active remineralization phase (1,14). Once clinical stability is achieved and gastrointestinal function is confirmed, transition to high-dose oral calcium carbonate or citrate, combined with active vitamin D analogs such as calcitriol or alfacalcidol, forms the foundation of sustained replacement therapy (15). Calcitriol is particularly valuable in this context because it bypasses the need for renal one-alpha-hydroxylase activation, which is impaired by the sudden drop in parathyroid hormone, thereby directly enhancing intestinal calcium absorption and supporting skeletal remineralization (1). Importantly, magnesium deficiency, frequently overlooked but universally present in hungry bone syndrome due to concurrent skeletal uptake and reduced renal conservation, must be aggressively corrected with intravenous or oral supplementation, as hypomagnesemia impairs parathyroid hormone secretion and action, exacerbates hypocalcemia, and contributes to refractory replacement requirements (1,22). Monitoring protocols in the immediate postoperative period typically involve serum calcium and electrolyte checks, followed by daily assessments until stable oral regimens are established and discharge criteria are met (23,24). The duration of therapy varies widely, with mild cases resolving within one to two weeks, while severe presentations may require high-dose supplementation for longer period (6,14). Patient education regarding symptom recognition, adherence to replacement schedules, and the importance of follow-up laboratory monitoring is essential to prevent readmission or delayed complications (1,8). Prevention strategies have gained considerable traction in recent years as clinicians recognize that anticipatory management significantly reduces morbidity, length of stay, and healthcare utilization (1,8). Preoperative optimization begins with thorough assessment of vitamin D status, with many endocrine surgery centers advocating for repletion of 25-hydroxyvitamin D to levels above thirty nanograms per milliliter several weeks before surgery to enhance baseline mineral reserves and improve postoperative calcium homeostasis (25). The role of prophylactic bisphosphonate or denosumab administration remains controversial but is increasingly explored in high-risk cohorts (1). Recent theoretical concerns center on the potential to blunt the beneficial skeletal remineralization response or precipitate atypical fracture patterns (6). However, several recent retrospective analyses suggest that, carefully timed preoperative bisphosphonate administration may attenuate the magnitude of postoperative hypocalcemia without compromising long-term bone density recovery (8). Meanwhile, intraoperative techniques such as parathyroid autotransplantation, meticulous preservation of native glands, and intraoperative parathyroid hormone monitoring have not been shown to directly prevent hungry bone syndrome (1,2). More recently, risk-stratified postoperative protocols are widely implemented, whereby patients with multiple predictive factors are initiated on prophylactic oral calcium and calcitriol within six hours of surgery, monitored more intensively, and provided with clear escalation pathways for intravenous therapy if biochemical thresholds are breached. This targeted approach has demonstrably reduced severe hypocalcemia rates and shortened hospital stays in tertiary referral centers (1,15). Long-term prognosis for patients who develop hungry bone syndrome following parathyroidectomy for primary hyperparathyroidism is generally favorable, with the majority achieving complete biochemical and clinical resolution within three months, accompanied by significant improvements in bone mineral density, fracture risk reduction, and quality of life metrics (8,26). Serial dual-energy X-ray absorptiometry studies have confirmed that the rapid postoperative mineral uptake translates into sustained gains in trabecular and cortical bone mass, particularly at the lumbar spine and femoral neck, validating the skeletal hunger as a physiologically appropriate reparative process rather than a pathological

derangement (1,5). Renal function typically stabilizes or improves as hypercalciuria resolves and nephrolithiasis risk declines, while neurocognitive symptoms such as fatigue, depression, and memory complaints frequently ameliorate following normalization of calcium and parathyroid hormone axes (6,27). Rare complications include prolonged supplementation dependency, rebound hypercalcemia from overtreatment, or ectopic calcification if phosphate and calcium product exceeds recommended thresholds, underscoring the need for careful monitoring during the tapering phase (8,16). Patient-reported outcome measures consistently demonstrate high satisfaction rates following successful management of hungry bone syndrome, particularly when clear communication, anticipatory guidance, and structured follow-up are provided (15). Hence, future studies should continue to improve our conception of hungry bone syndrome through the integration of novel biomarkers, predictive modeling, and personalized medicine approaches (24). Likewise, Machine-learning algorithms incorporating preoperative laboratory values, imaging characteristics, genetic polymorphisms related to vitamin D metabolism and calcium-sensing receptor function, and clinical demographics have shown promising accuracy in forecasting individual risk, enabling truly customized perioperative protocols (24). In the meantime, point-of-care ionized calcium analyzers and continuous telemetry monitoring are being piloted in high-volume endocrine surgery units to facilitate real-time titration of replacement therapy and reduce nursing workload (8,15). Interestingly, investigations into the role of bone turnover markers such as procollagen type one N-terminal propeptide, carboxy-terminal telopeptide, and osteocalcin in predicting the trajectory and duration of hungry bone syndrome are in processing, with preliminary data suggesting that dynamic changes in these markers during the first postoperative week may guide tapering decisions more accurately than static calcium measurements alone (8,28). Furthermore, the interplay between gut microbiome composition, intestinal calcium absorption efficiency, and postoperative metabolic recovery represents a novel frontier, with early animal models indicating that microbial modulation may influence mineral homeostasis and inflammatory responses following parathyroidectomy (29). Accordingly, clinical trials evaluating standardized risk-stratified pathways, comparative effectiveness of different calcium formulations, and optimal timing of vitamin D repletion are actively underway, promising to generate higher-level evidence to replace current expert consensus guidelines (30,31). In parallel, international collaborative registries are also accumulating large-scale data on hungry bone syndrome incidence across diverse populations, surgical techniques, and healthcare systems, facilitating more robust epidemiological insights and benchmarking opportunities (32). Despite these advances, several knowledge gaps persist, particularly regarding the precise molecular signaling cascades that govern the osteoblast surge postoperatively, the long-term skeletal remodeling patterns beyond the first year, and the optimal management of refractory cases requiring prolonged intravenous therapy or endocrine consultation (2,8). The economic burden of hungry bone syndrome, including extended hospitalization, laboratory monitoring and medication costs emphasizing the need for cost-effective preventive strategies and standardized care pathways (8). Education and training for endocrine surgeons, endocrinologists, hospitalists, and nursing staff continue to be critical, as early recognition and protocol adherence remain the strongest determinants of patient outcomes (33). In this regard, collaboration between endocrinology, nephrology, nutrition, and pharmacy ensures comprehensive management of electrolyte disturbances, medication interactions, and patient counseling, particularly in complex cases with comorbid renal impairment, malabsorption syndromes, or concurrent osteoporosis therapies (26,34). As surgical indications for primary hyperparathyroidism evolve and minimally invasive techniques become increasingly refined, the metabolic consequences of parathyroidectomy must remain a central focus of perioperative care, ensuring that the remarkable benefits of surgical cure are not overshadowed by preventable complications (4,31). The integration of risk prediction tools, proactive supplementation protocols, and continuous monitoring frameworks has already transformed hungry bone syndrome from a feared and unpredictable complication into a manageable and largely preventable clinical entity, reflecting the broader maturation of endocrine surgical care (32). Finally, postoperative hungry bone syndrome in primary hyperparathyroidism serves as a profound reminder of the skeleton's dynamic capacity for regeneration and the delicate equilibrium that governs mineral homeostasis, demanding respect, vigilance, and evidence-based intervention from the surgical and medical teams alike (16).

Conclusion

Postoperative hungry bone syndrome remains a clinically significant complication following parathyroidectomy for primary hyperparathyroidism, characterized by profound and prolonged hypocalcemia driven by rapid skeletal remineralization after the abrupt decline in parathyroid hormone. Though, this syndrome reflects a dramatic physiological shift rather than a surgical failure, it demands meticulous preoperative risk stratification and vigilant postoperative management. Patients with severe or prolonged disease, markedly elevated preoperative parathyroid hormone levels, substantial radiographic bone involvement, and concomitant vitamin D deficiency are at highest risk and benefit most from prophylactic calcium and active vitamin D supplementation. Updated

clinical approaches emphasize early recognition, frequent biochemical monitoring, and protocol-driven replacement strategies that address concurrent hypophosphatemia and hypomagnesemia, which can otherwise impede calcium homeostasis and prolong recovery. Recent advances have refined predictive algorithms, enabling clinicians to tailor supplementation intensity and duration to individual patient profiles, thereby reducing hospital stays and minimizing symptomatic complications such as tetany or cardiac arrhythmias. Despite the acute metabolic challenges, hungry bone syndrome ultimately signifies robust skeletal recovery, with long-term follow-up demonstrating substantial improvements in bone mineral density and fracture risk reduction. Moving forward, the establishment of standardized, evidence-based management pathways, coupled with the integration of dynamic biomarkers and artificial intelligence–assisted risk modeling, will further optimize perioperative care. With proactive vigilance and individualized therapeutic protocols, hungry bone syndrome can be effectively mitigated, ensuring that the curative benefits of parathyroidectomy are safely realized without compromising postoperative recovery or long-term skeletal health.

Acknowledgments

We would also like to show our gratitude to the (Clinical Research Development Unit, Golestan Hospital, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran) for sharing their pearls of wisdom with us during the course of this research.

Authors' contribution

Conceptualization: Arash Forouzan, Mojtaba Sasani.
Data curation: Mojtaba Sasani.
Investigation: Arash Forouzan, Mojtaba Sasani.
Resources: Majid Fattah.
Supervision: Mojtaba Sasani.
Validation: Mojtaba Sasani.
Visualization: Mojtaba Sasani.
Writing–original draft: All authors.
Writing–review and editing: Mojtaba Sasani.

Conflicts of interest

The authors declare that they have no competing interests.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

Funding/Support

None.

References

1. Coman A, Tarta C, Marian M, Popa DI, Olariu S, Rosu M, et al. Hungry Bone Syndrome After Parathyroidectomy for Secondary Hyperparathyroidism: Pathogenesis and Contemporary Clinical Considerations. J Clin Med. 2025;14:7104. doi: 10.3390/jcm14197104.
2. Kwon D, Kim BC, Lee YM, Sung TY, Chung KW, Kim WW. Hungry bone syndrome after parathyroidectomy in secondary and tertiary hyperparathyroidism: a retrospective cohort study. Ann Surg Treat Res. 2026;110:149-56. doi: 10.4174/astr.2026.110.3.149.
3. Coman A, Tarta C, Aiordachioae GA, Goldis D, Utu D, Marian M, et al. Predictors of Hungry Bone Syndrome and Reintervention After Subtotal Versus Total Parathyroidectomy for Secondary Hyperparathyroidism in Dialysis Patients: A Single-Center Cohort. J Clin Med. 2025;14:4944. doi: 10.3390/jcm14144944.
4. Chandran M, Bilezikian JP, Salleh NM, Ying H, Lau J, Lee J, et al. Hungry bone syndrome following parathyroidectomy for primary hyperparathyroidism in a developed country in the Asia Pacific. A cohort study. Osteoporos Sarcopenia. 2022;8:11-6. doi: 10.1016/j.afos.2022.03.004.
5. Roumpou A, Palermo A, Tournis S, Hasenmajer V, Pasieka JL, Kaltsas G, et al. Bone in Parathyroid Diseases Revisited: Evidence From Epidemiological, Surgical and New Drug Outcomes. Endocr Rev. 2025;46:576-620. doi: 10.1210/endrev/bnaf010.
6. Witteveen JE, van Thiel S, Romijn JA, Hamdy NA. Hungry bone syndrome: still a challenge in the post-operative management of primary hyperparathyroidism: a systematic review of the literature. Eur J Endocrinol. 2013;168:R45-53. doi: 10.1530/eje-12-0528.
7. Matuszewski W, Szklarz M, Górny J, Kordas B, Rutkowska J, Juranek J. Severe Hypocalcemia in Hungry Bone Syndrome After Parathyroid Surgery: A Case Study and Review. Am J Case Rep. 2026;27:e950046. doi: 10.12659/ajcr.950046.
8. Coman A, Tarta C, Isaic A, Marian M, Olariu S, Ardelean A, et al. Predictors of hungry bone syndrome after parathyroidectomy in secondary hyperparathyroidism: a narrative review of bone turnover biomarkers and risk prediction tools. J Clin Med. 2025;14:7849. doi: 10.3390/jcm14217849.
9. Wein MN, Kronenberg HM. Regulation of bone remodeling by parathyroid hormone. Cold Spring Harb Perspect Med. 2018;8:a031237. doi: 10.1101/cshperspect.a031237.
10. Alexander RT, Dimke H. Effects of parathyroid hormone on renal tubular calcium and phosphate handling. Acta Physiol (Oxf). 2023;238:e13959. doi: 10.1111/apha.13959.
11. Kaderli RM, Riss P, Dunkler D, Pietschmann P, Selberherr A, Scheuba C, et al. The impact of vitamin D status on hungry bone syndrome after surgery for primary hyperparathyroidism. Eur J Endocrinol. 2018;178:1-9. doi: 10.1530/eje-17-0416.
12. Nuti R, Merlotti D, Gennari L. Vitamin D deficiency and primary hyperparathyroidism. J Endocrinol Invest. 2011;34:45-9.
13. Maheshwari M, Khan IA. Risk Factors for Transient and Permanent Hypoparathyroidism Following Thyroidectomy: A Comprehensive Review. Cureus. 2024;16:e66551. doi: 10.7759/cureus.66551.
14. Cartwright C, Anastasopoulou C. Hungry Bone Syndrome. Treasure Island (FL): StatPearls Publishing LLC.; 2026.
15. Kilinc I, Oruc M, Savas F, Ertenlice A, Ulusoy S. Independent predictors of hungry bone syndrome after parathyroidectomy for primary hyperparathyroidism: insights from a large cohort study. Diagnostics (Basel). 2026;16:1041. doi: 10.3390/diagnostics16071041.
16. Li B, Lv X, Li X, Hou X, Xu F. Postoperative hungry bone syndrome in primary hyperparathyroidism: A case report. Medicine (Baltimore). 2024;103:e39717. doi: 10.1097/md.0000000000039717.

17. Florakis D, Karakozis S, Tseleni-Balafouta S, Makras P. Lessons learned from the management of Hungry Bone Syndrome following the removal of an Atypical Parathyroid Adenoma. J Musculoskelet Neuronal Interact. 2019;19:379-84.
18. Rastogi D, Gautam N, Ara Z, Waliullah S, Srivastava RN. Prevalence of Abnormal Bone-Specific Alkaline Phosphatase in Orthopaedic Trauma Patients: A Cross-Sectional Study From a Tertiary Trauma Centre. Cureus. 2022;14:e24264. doi: 10.7759/cureus.24264.
19. Helbing A, Leslie SW, Levine SN. Primary Hyperparathyroidism. Treasure Island (FL): StatPearls Publishing LLC.; 2026.
20. Fatima T, Arshad M, Ajmal Z, Siddiqi AI, Shafiq W. Brown Tumor in Primary Hyperparathyroidism and Hungry Bone Syndrome Post-Parathyroidectomy: Two Rare Entities. J Cancer Allied Spec. 2026;12:46-9. doi: 10.2478/jcas-2026-0007.
21. Kumar S, Diamond T. Lessons learnt from delayed diagnosis of FGF-23-producing tumour-induced osteomalacia and post-operative hungry bone syndrome. Bone Rep. 2020;12:100276. doi: 10.1016/j.bonr.2020.100276.
22. Vetter T, Lohse MJ. Magnesium and the parathyroid. Curr Opin Nephrol Hypertens. 2002;11:403-10. doi: 10.1097/00041552-200207000-00006.
23. Jose Garcia Diaz F, Beth Martin L, Lucia Gomez Gila A, Navarro Moreno C. Intensive Calcium Monitoring Following Parathyroidectomy: Prevention of Hungry Bone Syndrome in Children. Turk Arch Pediatr. 2024;59:109-11. doi: 10.5152/TurkArchPediatr.2024.23205.
24. Chai Y, Yuan N, Yin J, Shen B, Sun L, Zhang L, et al. Machine learning-based predictive model for hungry bone syndrome following parathyroidectomy in secondary hyperparathyroidism. Front Endocrinol (Lausanne). 2025;16:1635451. doi: 10.3389/fendo.2025.1635451.
25. Salman MA, Rabiee A, Salman AA, Youssef A, Shaaban HE, Ftohy T, et al. Role of vitamin D supplements in prevention of hungry bone syndrome after successful parathyroidectomy for primary hyperparathyroidism: A prospective study. Scand J Surg. 2021;110:329-34. doi: 10.1177/1457496920962601.
26. Carsote M, Nistor C. Forestalling Hungry Bone Syndrome after Parathyroidectomy in Patients with Primary and Renal Hyperparathyroidism. Diagnostics (Basel). 2023;13:1953. doi: 10.3390/diagnostics13111953.
27. Lila AR, Sarathi V, Jagtap V, Bandgar T, Menon PS, Shah NS. Renal manifestations of primary hyperparathyroidism. Indian J Endocrinol Metab. 2012;16:258-62. doi: 10.4103/2230-8210.93745.
28. Ko WC, Liu CL, Lee JJ, Liu TP, Wu CJ, Cheng SP. Osteocalcin is an Independent Predictor for Hungry Bone Syndrome After Parathyroidectomy. World J Surg. 2020;44:795-802. doi: 10.1007/s00268-019-05251-0.
29. Weaver CM. Diet, gut microbiome, and bone health. Curr Osteoporos Rep. 2015;13:125-30. doi: 10.1007/s11914-015-0257-0.
30. Ferreira JL, de Brito Marques F, Freire L, Soares V, Guerreiro L, Silva S, et al. Intensive calcium monitoring improves outcomes on hungry bone syndrome in hyperparathyroidism. Endocr Regul. 2021;55:30-41. doi: 10.2478/enr-2021-0005.
31. Anwar F, Abraham J, Nakshabandi A, Lee E. Treatment of hypocalcemia in hungry bone syndrome: A case report. Int J Surg Case Rep. 2018;51:335-9. doi: 10.1016/j.ijscr.2018.08.011.
32. Amjad W, Ginzberg SP, Passman JE, Heintz J, Kelz RR, Wachtel H. Predictive Risk Score for Postparathyroidectomy Hungry Bone Syndrome in Patients With Secondary Hyperparathyroidism. J Clin Endocrinol Metab. 2024;109:603-10. doi: 10.1210/clinem/dgad636.
33. Brasier AR, Nussbaum SR. Hungry bone syndrome: clinical and biochemical predictors of its occurrence after parathyroid surgery. Am J Med. 1988;84:654-60. doi: 10.1016/0002-9343(88)90100-3.
34. Silarat P, Saeseow S, Pathumarak A, Srisuwarn P, Suvikapakornkul R, Disthabanchong S. Improved Clinical Outcomes Associated With Hungry Bone Syndrome Following Parathyroidectomy in Dialysis Patients. Endocr Pract. 2024;30:1079-88. doi: 10.1016/j.eprac.2024.08.004.