



Hungry Bone Syndrome in the Postoperative Management of Primary Hyperparathyroidism: Report of Two Cases

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Abstract

Introduction: Hungry Bone Syndrome (HBS) is a rare but potentially severe metabolic complication following parathyroidectomy, occurring mainly in patients with severe primary hyperparathyroidism (PHPT) and high bone turnover [1–3].

Case Reports: We report two cases of HBS occurring after parathyroidectomy for advanced primary hyperparathyroidism. Both patients presented with marked preoperative hypercalcemia, very high parathyroid hormone (PTH) levels, and clinical and biological features of increased bone turnover. The postoperative course was characterized by prolonged hypocalcemia associated with hypophosphatemia, requiring intensive calcium and vitamin D supplementation.

Conclusion: HBS should be anticipated in high-risk patients undergoing parathyroidectomy. Early recognition and appropriate management are essential to reduce postoperative morbidity.

Keywords: Hungry Bone Syndrome; Primary Hyperparathyroidism; Parathyroidectomy; Postoperative Hypocalcemia

Introduction

Primary hyperparathyroidism is a common endocrine disorder characterized by excessive secretion of parathyroid hormone (PTH), leading to hypercalcemia and increased bone turnover [4, 5]. Parathyroidectomy remains the definitive treatment for this condition [6, 7]. Hungry Bone Syndrome is a rare but serious postoperative complication, defined by rapid, profound, and prolonged hypocalcemia, often associated with hypophosphatemia and sometimes hypomagnesemia [1, 2]. It mainly occurs in patients with severe PHPT and high preoperative bone turnover [3].

We report two cases of HBS and discuss its pathophysiology, risk factors, and management.

Case Reports

Case 1

A 78-year-old woman with a history of cholecystectomy 12 years earlier was admitted to the emergency department for acute gastrointestinal symptoms. Laboratory investigations revealed hypercalcemia of 141 mg/L, hypophosphatemia of 19 mg/L, and an elevated PTH level of 392 pg/mL (approximately nine times the upper limit of normal).

Neck ultrasonography and cervicothoracic MRI confirmed a parathyroid lesion. Parathyroidectomy was performed, and histopathological examination confirmed a parathyroid adenoma.

Preoperative medical management included intravenous hydration, forced diuresis, and intravenous bisphosphonate therapy.

Case 2

A 46-year-old woman was admitted for generalized weakness and gait impairment in the context of progressively worsening chronic low back pain. Clinical examination revealed tachycardia (110 bpm) and chest wall deformity.

Laboratory tests showed severe hypercalcemia (185 mg/L), hypophosphatemia (19 mg/L), markedly elevated PTH levels (4936 pg/mL; normal range 15–65), and elevated alkaline phosphatase

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(478 IU/L; normal range 40–150), indicating intense bone turnover. Neck ultrasonography and sestamibi scintigraphy identified a localized parathyroid adenoma. Parathyroidectomy was performed. Histopathological examination confirmed a parathyroid adenoma weighing 4 g and measuring $3 \times 2 \times 0.6$ cm.

Postoperative Outcome

In both patients, the postoperative course was characterized by prolonged hypocalcemia associated with hypophosphatemia, with progressive normalization of PTH levels, consistent with the diagnosis of Hungry Bone Syndrome [1, 3].

Management required high-dose oral calcium supplementation (calcium carbonate) combined with active vitamin D (alfacalcidol), with close biochemical monitoring. Patients were discharged on prolonged calcium and vitamin D therapy.

Discussion

Hungry Bone Syndrome is characterized by rapid, profound, and prolonged hypocalcemia following parathyroidectomy, particularly in patients with severe primary hyperparathyroidism [1, 3, 8].

The pathophysiology involves abrupt withdrawal of excessive circulating PTH after surgery, leading to an immediate cessation of bone resorption while bone formation persists. This results in massive skeletal uptake of calcium and phosphate, causing severe hypocalcemia [3, 9].

Although no universal definition exists, HBS is generally defined as hypocalcemia below 8.4 mg/dL persisting for more than four days after surgery [2, 9].

Identified risk factors include very high preoperative PTH levels, severe hypercalcemia, elevated alkaline phosphatase, radiological evidence of osteitis fibrosa cystica, and large parathyroid adenomas [3, 9, 10].

The duration of HBS corresponds to the time required for skeletal remineralization, reflected by normalization of bone turnover markers, radiological improvement, and significant increases in bone mineral density [1, 11].

Treatment relies on aggressive and prolonged calcium supplementation combined with active vitamin D analogues, requiring close monitoring and gradual dose adjustment [2, 6, 10].

Conclusion

Hungry Bone Syndrome is a rare but serious complication of parathyroidectomy. Early recognition and appropriate management are crucial to prevent severe hypocalcemia-related complications and to reduce postoperative morbidity in patients with severe primary hyperparathyroidism.

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