



# PARATHYROIDECTOMY FOR TERTIARY HYPERPARATHYROIDISM IN A PATIENT WITH CHRONIC KIDNEY DISEASE AND RENAL OSTEODYSTROPHY: A CASE REPORT

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## ABSTRACT

**Background:** Chronic kidney disease-mineral and bone disorder (CKD-MBD) poses a major therapeutic challenge in hemodialysis patients. Untreated secondary hyperparathyroidism (SHPT) can progress to tertiary hyperparathyroidism (THPT), characterized by the autonomous secretion of parathyroid hormone (PTH) despite correction of biochemical abnormalities. **Case Presentation:** We report the case of a 59-year-old male with end-stage renal disease (ESRD) on hemodialysis for eight years, who developed severe THPT and a pathologic femoral neck fracture secondary to renal osteodystrophy. Laboratory results revealed markedly elevated PTH (4,876 pg/mL preoperatively and 8 pg/mL postoperatively), hypercalcemia, and hyperphosphatemia despite optimized medical therapy. The patient underwent subtotal parathyroidectomy (3½ glands removed) with histopathologic confirmation of nodular hyperplasia. Postoperatively, he developed hungry bone syndrome, successfully managed with intravenous and oral calcium and calcitriol. At three-month follow-up, PTH decreased to 215 pg/mL with normalization of calcium and phosphorus and significant clinical improvement. **Conclusion:** Parathyroidectomy remains the definitive treatment for refractory tertiary hyperparathyroidism, providing biochemical control and symptom relief. This case highlights the multidisciplinary management required in CKD-MBD, integrating nephrology, endocrinology, and orthopedic care.

**Keywords:** tertiary hyperparathyroidism; chronic kidney disease; parathyroidectomy; renal osteodystrophy; hungry bone syndrome.

## INTRODUCTION

Chronic kidney disease–mineral and bone disorder (CKD-MBD) represents one of the most challenging complications in dialysis patients, involving abnormalities in calcium, phosphorus, vitamin D metabolism, and parathyroid hormone (PTH) secretion (KDIGO, 2017). Secondary hyperparathyroidism arises due to persistent hypocalcemia, hyperphosphatemia, and calcitriol deficiency, which chronically stimulate parathyroid hyperplasia (Almquist et al., 2020). When PTH secretion becomes autonomous despite partial correction of these disturbances, tertiary hyperparathyroidism (THPT) develops (Patel et al., 2021).

THPT is particularly relevant among patients with advanced CKD or those undergoing long-term hemodialysis, where nodular parathyroid hyperplasia may become refractory to pharmacologic therapy (Nakamura et al., 2024). Clinical consequences include persistent hypercalcemia, bone pain, fractures, osteitis fibrosa cystica, and vascular calcification—all of which worsen prognosis and quality of life (Raudales et al., 2025).

International guidelines, such as those from *Kidney Disease: Improving Global Outcomes* (KDIGO), recommend tight control of calcium, phosphate, and PTH levels using phosphate binders, vitamin D analogues, and calcimimetics as first-line therapies. In refractory or severe skeletal or vascular cases, surgical intervention—parathyroidectomy—remains the definitive treatment (Hamed et al., 2021).

Recent studies show that parathyroidectomy achieves biochemical cure and normocalcemia rates exceeding 95%, outperforming medical therapy alone (Palumbo et al., 2021). However, debate persists regarding the optimal timing and surgical technique—subtotal versus total resection with or without autotransplantation—given risks such as permanent hypoparathyroidism or hungry bone syndrome (Madorin et al., 2012).

This report describes a hemodialysis patient with severe THPT and pathologic femoral fracture secondary to renal osteodystrophy, managed with subtotal parathyroidectomy, and discusses its clinical and therapeutic implications.

## CASE PRESENTATION

A 59-year-old male with end-stage renal disease (ESRD) on maintenance hemodialysis for eight years (three weekly sessions: Monday, Wednesday, Friday), with long-standing hypertension and diabetes, presented for surgical repair of a right femoral neck fracture. Medications included methyldopa 500 mg orally every 12 h, amlodipine 10 mg every 12 h, and gliclazide 30 mg daily. He reported an allergy to acetaminophen.

Radiographs revealed decreased bone density and a right femoral neck fracture consistent with severe bone fragility. During the orthopedic procedure, marked bone friability prevented adequate fixation, leading to abortion of arthroplasty. The patient was transferred to the intensive care unit for stabilization and metabolic reassessment.

Transthoracic echocardiography showed preserved left ventricular function and mild mitral stenosis. Neck computed tomography revealed enlarged parathyroid glands, more prominent on the right, without mediastinal extension. Laboratory tests demonstrated intact PTH 4,876 pg/mL preoperatively and 8 pg/mL postoperatively, along with hypercalcemia and hyperphosphatemia despite optimized therapy (cinacalcet 30 mg/day, paricalcitol 1 µg at each dialysis session, vitamin D 50,000 IU weekly, and calcium carbonate 1 g every 8 h). Alkaline phosphatase was markedly elevated, indicating intense osteoblastic activity.

Given persistent severe hyperparathyroidism and pathologic fracture, surgical intervention was indicated in accordance with KDIGO 2017 recommendations for PTH > 800 pg/mL with skeletal manifestations refractory to medical therapy.

Subtotal parathyroidectomy (3½ glands removed) was performed uneventfully. Histopathology confirmed diffuse nodular hyperplasia. Postoperatively, the patient developed symptomatic hypocalcemia consistent with hungry bone syndrome, managed with intravenous calcium gluconate followed by oral calcium and calcitriol supplementation.

At three months, laboratory tests demonstrated normalization of calcium with oral calcium supplementation. The patient remained on regular hemodialysis with improved functional status and stable biochemical parameters.

## **DISCUSSION**

Tertiary hyperparathyroidism (THPT) is a frequent and disabling complication of advanced CKD. Inadequate control of calcium–phosphate balance leads to progressive parathyroid hyperplasia and autonomous PTH secretion. KDIGO (2017) recommends initial medical management—phosphate binders, vitamin D analogues, and calcimimetics—with surgical parathyroidectomy reserved for refractory cases or those with severe skeletal involvement.

In this case, the patient developed a pathologic femoral fracture due to severe renal osteodystrophy and extremely high PTH levels (> 4,800 pg/mL), fulfilling surgical criteria.

Subtotal parathyroidectomy achieved marked biochemical and clinical improvement. Postoperative hungry bone syndrome, a common consequence of abrupt PTH withdrawal, was successfully managed conservatively.

Current evidence supports parathyroidectomy as the most effective treatment for biochemical normalization and symptom relief in THPT, with high remission rates and improved quality of life. Nonetheless, close multidisciplinary follow-up is essential to prevent hypocalcemia and ensure long-term metabolic stability.

## REFERENCES

ALMQUIST, M.; ISAKSSON, E.; CLYNE, N. The treatment of renal hyperparathyroidism. *Endocr Relat Cancer*, v. 27, n. 1, p. R21-R34, jan. 2020. Disponível em: <https://doi.org/10.1530/ERC-19-0284>.

HAMED, S. *et al.* Secondary hyperparathyroidism in chronic kidney disease: pathophysiology and management. *Cureus*, 2021.

KIDNEY DISEASE: Improving Global Outcomes (KDIGO). *Clinical Practice Guideline Update for CKD-MBD. Kidney International Supplements*, 2017.

MADORIN, C. *et al.* The surgical management of renal hyperparathyroidism. *Eur Arch Otorhinolaryngol*, v. 269, n. 6, p. 1565-1576, jun. 2012. Disponível em: <https://doi.org/10.1007/s00405-011-1833-2>.

NAKAMURA, M.; TAKIGUCHI, S.; UEHARA, S.; TOMITA, Y. Outcome of surgical parathyroidectomy for tertiary hyperparathyroidism in kidney transplant recipients: tertiary hyperparathyroidism should not be ignored, for the sake of precious allografts. *Renal Failure*, v. 46, n. 1, p. 2333919, dez. 2024. DOI: 10.1080/0886022X.2024.2333919.

PALUMBO, V. D. *et al.* Tertiary hyperparathyroidism: a review. *Clin Ter*, v. 172, n. 3, p. 241-246, 5 maio 2021. Disponível em: <https://doi.org/10.7417/CT.2021.2322>.

PATEL, A. *et al.* Parathyroidectomy for Tertiary Hyperparathyroidism: A Multi-Institutional Analysis of Outcomes. *J Surg Res.*, v. 258, p. 430-434, fev. 2021. Disponível em: <https://doi.org/10.1016/j.jss.2020.08.079>.

RAUDALES, B. M. *et al.* Severe consequences of uncontrolled tertiary hyperparathyroidism in a limited resources setting: a case report. *Front Nephrol*, v. 5, e1645622, 1 set. 2025. Disponível em: <https://doi.org/10.3389/fneph.2025.1645622>.