Subtotal Parathyroidectomy: A Comprehensive Review for Secondary Hyperparathyroidism Management

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Abstract

Secondary hyperparathyroidism (sHPT) is a common complication of chronic kidney disease (CKD), characterized by excessive secretion of parathyroid hormone (PTH) due to disrupted calcium-phosphate homeostasis. Subtotal parathyroidectomy (sPTx), involving the resection of 3.5 parathyroid glands, has emerged as an effective surgical option for patients with refractory sHPT, particularly when medical management fails. This review explores the role of sPTx in the management of sHPT, focusing on indications, surgical techniques, and outcomes. Evidence from recent studies highlights the procedure's success in achieving durable biochemical control, alleviating clinical symptoms, and improving quality of life. The long-term benefits of sPTx are contrasted with potential complications, including persistent or recurrent hyperparathyroidism and hypoparathyroidism. Furthermore, this article addresses the evolving landscape of sHPT management, emphasizing the need for individualized treatment plans and interdisciplinary care. By synthesizing current research and clinical insights, this review aims to provide a comprehensive understanding of the efficacy and limitations of subtotal parathyroidectomy in managing secondary hyperparathyroidism, contributing to optimized patient care and surgical decision-making.

Keywords: Subtotal Parathyroidectomy, Secondary Hyperparathyroidism

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Introduction

Secondary hyperparathyroidism (SHPT) is a condition characterized by excessive secretion of parathyroid hormone (PTH) secondary to chronic hypocalcemia, which often results from underlying conditions such as chronic kidney disease (CKD). The persistent stimulation of the parathyroid glands leads to their hyperplasia and elevated PTH levels, causing a wide range of metabolic disturbances. This condition is particularly prevalent among patients with CKD due to their impaired ability to maintain calcium and phosphate balance, as well as reduced vitamin D activation [1].

Surgical management of SHPT, known as parathyroidectomy, is often indicated when medical therapy fails to control PTH levels or when significant complications such as bone disease or vascular calcifications arise. Parathyroidectomy can be performed using different approaches, including subtotal parathyroidectomy, where most of the parathyroid tissue is removed, or total parathyroidectomy with autotransplantation of parathyroid tissue into another site such as the forearm [2].

The choice of surgical technique is influenced by several factors, including the extent of parathyroid hyperplasia, the patient's overall health, and the risk of recurrent disease. Subtotal parathyroidectomy leaves a small portion of parathyroid tissue in situ to maintain some PTH secretion, while total parathyroidectomy with autotransplantation aims to prevent recurrence while providing a controllable source of PTH [3].

Chronic kidney disease-mineral and bone disorder (CKD-MBD) is a key contributor to SHPT, as it disrupts calcium, phosphate, and vitamin D homeostasis. Reduced kidney function impairs phosphate excretion, leading to hyperphosphatemia, which stimulates PTH secretion. Additionally, decreased conversion of 25-hydroxyvitamin D to its active form, 1,25-dihydroxyvitamin D, reduces calcium absorption in the gut, further exacerbating hypocalcemia and hyperparathyroidism [4].

In addition to surgical interventions, medical management plays a critical role in controlling SHPT. Calcimimetics, such as cinacalcet, are often used to suppress PTH secretion by increasing the sensitivity of calcium-sensing receptors in the parathyroid glands. Other treatments include phosphate binders to manage hyperphosphatemia and vitamin D analogs to correct vitamin D deficiency [5].

Parathyroidectomy has shown significant benefits in alleviating the symptoms of SHPT and improving biochemical abnormalities. Patients often experience improvements in bone density, reduced bone pain, and a decrease in cardiovascular complications associated with SHPT, such as vascular calcifications. However, careful monitoring is required postoperatively to prevent complications like hypocalcemia or recurrent hyperparathyroidism [6].

The surgical approach to parathyroidectomy has evolved with advancements in imaging techniques and minimally invasive procedures. Preoperative localization of hyperplastic parathyroid glands using imaging modalities such as ultrasonography, sestamibi scans, or 4D computed tomography (CT) has significantly improved the precision of surgery and reduced operative times [7].

Minimally invasive parathyroidectomy, guided by preoperative imaging and intraoperative PTH monitoring, has gained popularity due to its reduced morbidity and shorter recovery time compared to traditional open surgery. This technique is particularly beneficial for patients with localized disease and minimal comorbidities [8].

Recurrent or persistent SHPT after initial parathyroidectomy poses a challenging clinical scenario. The recurrence rate depends on the surgical technique used and the completeness of gland removal. Reoperations are associated with increased risks, including injury to surrounding structures and difficulty in localization of residual parathyroid tissue [9].

Autotransplantation of parathyroid tissue during total parathyroidectomy aims to maintain long-term control of PTH levels. The transplanted tissue is typically placed in a site easily accessible for reoperation if necessary. Success rates of autotransplantation are high, but there is still a risk of graft hyperplasia and recurrent hyperparathyroidism [10].

In patients with SHPT secondary to CKD, the timing of parathyroidectomy is crucial. Surgery is generally considered when PTH levels remain refractory to medical treatment or when complications such as severe hypercalcemia, pruritus, or calciphylaxis develop. Early intervention can prevent irreversible complications and improve quality of life [11].

Postoperative management following parathyroidectomy involves close monitoring of calcium and phosphorus levels to prevent hypocalcemia and manage any residual hyperparathyroidism. Calcium and vitamin D supplementation are often required to stabilize serum calcium levels and support bone health [12].

The long-term outcomes of parathyroidectomy in SHPT are generally favorable, with significant reductions in PTH levels and improvements in symptoms and biochemical parameters. However, recurrence remains a concern, necessitating lifelong monitoring of calcium, phosphate, and PTH levels [13].

In addition to medical and surgical interventions, lifestyle modifications and dietary management play an important role in managing SHPT. Patients are often advised to limit dietary phosphate intake and ensure adequate calcium and vitamin D consumption to support overall metabolic balance [14].

Emerging therapies and novel surgical techniques continue to enhance the management of SHPT. Advances in intraoperative monitoring, including real-time PTH assays, and the development of less invasive approaches have improved surgical outcomes and reduced complication rates [15].

Calciphylaxis, a severe complication of SHPT characterized by vascular calcification and tissue necrosis, is a significant indication for parathyroidectomy. This life-threatening condition highlights the need for aggressive management of SHPT to prevent irreversible damage [16].

While parathyroidectomy offers a definitive treatment for SHPT, patient selection and preoperative planning are critical to achieving optimal outcomes. Multidisciplinary collaboration among nephrologists, endocrinologists, and surgeons ensures comprehensive care for patients with SHPT [17].

Research into the molecular mechanisms driving parathyroid hyperplasia and PTH secretion has opened new avenues for targeted therapies. Understanding the genetic and epigenetic factors contributing to SHPT may lead to more personalized and effective treatments in the future [18].

Secondary hyperparathyroidism remains a complex condition requiring a multifaceted approach to management. Advances in surgical techniques, medical therapies, and understanding of disease pathophysiology continue to improve outcomes for affected patients [19], secondary hyperparathyroidism is a significant clinical challenge, particularly in the context of CKD. While surgical intervention remains a cornerstone of treatment, it is complemented by medical

management and lifestyle modifications to achieve optimal patient outcomes. Continued research and innovation are essential to address the evolving needs of this patient population [20].

Secondary hyperparathyroidism (SHPT) is a common and severe complication in patients with chronic kidney disease (CKD), characterized by excessive secretion of parathyroid hormone (PTH) due to impaired calcium and phosphate homeostasis. This condition often leads to bone demineralization, vascular calcification, and cardiovascular morbidity. Subtotal parathyroidectomy (SPTX) has emerged as a surgical treatment option for patients with SHPT who do not respond to medical therapy. By preserving a portion of the parathyroid tissue, SPTX aims to maintain basal PTH levels while alleviating hyperparathyroidism-related complications [21].

SHPT results from a cascade of biochemical disturbances beginning with reduced kidney function, leading to decreased phosphate excretion and impaired vitamin D activation. The subsequent hypocalcemia and hyperphosphatemia stimulate PTH secretion, creating a vicious cycle of parathyroid gland hyperplasia and hormone overproduction. Understanding this pathophysiology underscores the importance of timely intervention, including surgical options [22].

Historically, medical management has been the cornerstone of SHPT treatment, involving dietary modifications, phosphate binders, and vitamin D analogs. However, these measures often fail in advanced cases, necessitating surgical intervention. SPTX, as opposed to total parathyroidectomy, offers a balance by reducing hormone levels while retaining some parathyroid function [23].

The decision to perform SPTX must consider the patient's overall health, disease severity, and response to prior treatments. Multidisciplinary collaboration ensures that the surgical approach is tailored to the patient's unique clinical scenario. Advances in imaging and intraoperative monitoring have further refined the success rates of this procedure [24].

Emerging research highlights the potential role of minimally invasive techniques and robotic-assisted surgeries in managing SHPT. These innovations aim to reduce operative time, postoperative complications, and recovery duration, though their long-term efficacy compared to traditional methods remains under investigation [25].

Indications

SPTX is indicated in patients with SHPT who exhibit persistently elevated PTH levels despite optimized medical management, including vitamin D analogs, calcimimetics, and phosphate binders. Other indications include severe hypercalcemia, refractory pruritus, calciphylaxis, or progressive bone disease. Patients with evidence of significant end-organ damage, such as cardiovascular calcifications, also benefit from this intervention. These indications are guided by clinical guidelines and individualized patient assessments to ensure maximal therapeutic benefit [26].

Patients presenting with debilitating symptoms that severely impact their quality of life often require SPTX. Symptoms such as severe bone pain, pathologic fractures, and unremitting pruritus are common clinical drivers for surgical intervention. These manifestations frequently correlate with high PTH levels and metabolic derangements, necessitating prompt surgical action [27].

Another important indication is the presence of resistant vascular calcifications, which contribute to cardiovascular morbidity and mortality in CKD patients. SPTX has been shown to attenuate the progression of vascular calcifications, improving both functional capacity and survival outcomes [28].

Recurrent SHPT following renal transplantation poses a unique challenge, as residual hyperplastic parathyroid tissue can continue to produce excessive PTH. In such cases, SPTX offers an effective solution, enabling the restoration of mineral homeostasis and protecting graft function [29].

While surgical criteria are generally well-defined, certain patient subsets may present atypically, requiring nuanced decision-making. For example, younger patients with rapidly progressive disease may benefit from early surgical intervention to prevent long-term complications [30].

Contraindications

Although SPTX is a valuable treatment modality, certain contraindications must be considered. Absolute contraindications include uncorrectable coagulopathy, severe cardiovascular instability, and active systemic infections. Relative contraindications may include advanced age, frailty, or poor functional status that heightens surgical risk. Additionally, patients with minimal disease burden or successful medical management should not undergo this procedure, as it might expose them to unnecessary risks [31].

Preoperative counseling is critical to evaluate surgical candidacy. This includes assessing the patient's understanding of potential benefits and risks, particularly in borderline cases. For instance, elderly patients with comorbidities may benefit more from continued medical management than from surgery [32].

The presence of significant anatomical anomalies, such as ectopic parathyroid glands or prior neck surgeries, can complicate the surgical approach. These factors require careful preoperative imaging and planning to mitigate the risk of incomplete resection or nerve damage [33].

Patients with psychosocial barriers, including non-adherence to postoperative care, may not be ideal candidates for SPTX. Effective postoperative management is critical for preventing complications and ensuring long-term success, necessitating a thorough evaluation of patient reliability [34].

Technological advancements, such as preoperative localization techniques and intraoperative PTH monitoring, are enhancing patient selection and minimizing contraindications. These tools allow for more precise surgical interventions, even in complex cases [35].

Preoperative Evaluation

A thorough preoperative evaluation is critical to identify suitable candidates for SPTX. This includes biochemical assessments, such as serum calcium, phosphate, alkaline phosphatase, and PTH levels, along with imaging studies like ultrasound and sestamibi scans to localize hyperactive glands. Cardiopulmonary evaluation and optimization of coexisting medical conditions are equally vital to minimize perioperative risk. Multidisciplinary collaboration involving nephrologists, endocrinologists, and surgeons ensures a comprehensive approach to patient management [36].

Biochemical markers provide essential insights into the severity of SHPT and guide surgical planning. Elevated PTH levels, in conjunction with hypercalcemia and hyperphosphatemia, often indicate advanced disease necessitating intervention. Serial measurements help monitor disease progression and treatment response [37].

High-resolution imaging, including four-dimensional CT and MRI, has revolutionized the preoperative assessment of parathyroid glands. These modalities enhance localization accuracy, particularly in cases of ectopic or supernumerary glands, thereby improving surgical outcomes [38].

The role of intraoperative PTH monitoring cannot be overstated, as it allows real-time assessment of surgical efficacy. A significant intraoperative drop in PTH levels correlates with successful gland excision, reducing the likelihood of persistent or recurrent disease [39].

Comprehensive preoperative preparation also involves patient education, addressing expectations, potential complications, and the importance of adherence to follow-up care. Such discussions foster informed decision-making and enhance overall satisfaction with surgical outcomes [40].

Surgical Approach and Technique

SPTX involves the removal of 3.5 parathyroid glands, leaving a small remnant of tissue to maintain basal PTH secretion. The procedure is typically performed under general anesthesia, employing a transverse cervical incision to access the parathyroid glands. Identification and preservation of the recurrent laryngeal nerve are paramount to avoid vocal cord dysfunction. Intraoperative PTH monitoring can guide the extent of resection and predict surgical success. The remaining parathyroid tissue is often implanted in a muscle pocket to allow easy access in case of future interventions [41].

The surgical team must meticulously identify and excise the hyperplastic glands while preserving essential structures, such as the thyroid gland and surrounding vasculature. A systematic approach minimizes the risk of incomplete resection or unintended tissue damage [42].

Adjunctive technologies, such as intraoperative ultrasound and fluorescence imaging, have emerged as valuable tools for improving gland localization and ensuring complete resection. These innovations enhance the precision of SPTX, reducing operative time and complications [43].

Muscle autotransplantation of the parathyroid remnant is a key component of the procedure. Common sites include the brachioradialis or sternocleidomastoid muscles, which provide an accessible location for future interventions if required [44].

The importance of a multidisciplinary team cannot be overstated. Collaboration among surgeons, anesthesiologists, and endocrinologists ensures optimal perioperative care, enhancing the likelihood of successful outcomes and minimizing complications [45].

Postoperative Management

Close monitoring of calcium and PTH levels is essential following SPTX to detect and manage potential complications such as hypocalcemia or recurrent hyperparathyroidism. Early hypocalcemia, also known as hungry bone syndrome, necessitates aggressive calcium and vitamin D supplementation. Long-term follow-up includes periodic evaluation of biochemical markers,

bone health, and cardiovascular status to ensure sustained surgical benefits and early detection of disease recurrence [46].

Postoperative hypocalcemia remains a common complication, attributed to the sudden drop in circulating PTH levels. Early recognition and intervention are critical to avoid severe symptoms, such as tetany or cardiac arrhythmias [47].

Recurrent SHPT, though less common, can occur due to incomplete gland excision or regrowth of the residual tissue. Regular monitoring of PTH levels and imaging studies helps identify recurrence early, enabling timely intervention [48].

Lifestyle modifications, including dietary adjustments and physical activity, complement medical and surgical management of SHPT. These measures support overall health and reduce the risk of complications, such as bone fractures and vascular calcifications [49, 50].

References:

1. National Kidney Foundation. KDOQI clinical practice guideline for bone metabolism and disease in chronic kidney disease. Am J Kidney Dis. 2003;42(4):S1-S201.
2. Tominaga Y, Matsuoka S, Uno N. Surgical and medical management of secondary hyperparathyroidism in patients on chronic dialysis. World J Surg. 2009;33(11):2335-2342.
3. Pitt SC, Sippel RS, Chen H. Secondary and tertiary hyperparathyroidism, state of the art surgical management. Surg Clin North Am. 2009;89(5):1227-1239.
4. Malluche HH, Mawad HW, Monier-Faugere MC. Renal osteodystrophy in chronic kidney disease: pathogenesis, diagnosis, and treatment. Clin Nephrol. 2010;74(2):124-134.
5. Block GA, Martin KJ, de Francisco ALM, et al. Cinacalcet for secondary hyperparathyroidism in patients receiving hemodialysis. N Engl J Med. 2004;350(15):1516-1525.
6. Fukagawa M, Yokoyama K, Koiwa F. Meta-analysis of the clinical effects of parathyroidectomy in dialysis patients. Kidney Int. 2010;78(11):1041-1049.
7. Cheung K, Wang TS, Farrokhyar F, Roman SA, Sosa JA. A meta-analysis of preoperative localization techniques for patients with primary hyperparathyroidism. Ann Surg Oncol. 2012;19(2):577-583.
8. Bergenfelz A, Jansson S, Wallin G. Parathyroid surgery in renal hyperparathyroidism: evidence-based treatment. World J Surg. 2009;33(10):2307-2315.
9. Ruda JM, Hollenbeak CS, Stack BC Jr. A systematic review of the diagnosis and treatment of primary hyperparathyroidism from 1995 to 2003. Otolaryngol Head Neck Surg. 2005;132(3):359-372.
10. Schneider R, Slater EP, Karakas E, Bartsch DK, Schlosser K. Initial parathyroid surgery in 606 patients with renal hyperparathyroidism. World J Surg. 2012;36(2):318-326.
11. Isaksson E, Cramér K, Högström H, Smedby O, Prütz KG. Early parathyroidectomy for secondary hyperparathyroidism in patients on hemodialysis. Scand J Urol Nephrol. 2011;45(1):52-57.

12. Evenepoel P, Cooper K, Holdaas H. Vitamin D and outcomes in kidney failure. Clin J Am Soc Nephrol. 2010;5(3):512-518.

13. Moorthi RN, Moe SM. Recent advances in the noninvasive diagnosis of vascular calcification in chronic kidney disease. Curr Opin Nephrol Hypertens. 2013;22(6):629-635.

14. Sprague SM, Ross EA. Correcting the chronic kidney disease mineral bone disorder: is it possible? Curr Opin Nephrol Hypertens. 2009;18(6):464-471.

15. Rothmund M, Wagner PK. Late results following subtotal parathyroidectomy for renal hyperparathyroidism. World J Surg. 1982;6(6):772-779.

16. Mazokopakis EE, Fouka E, Kotsanis SD. Calciphylaxis (calcific uremic arteriolopathy) and its management. Am J Med Sci. 2011;341(6):451-457.

17. Tominaga Y, Katayama A, Sato T, Tominaga D, Tagawa Y. Current surgical management of secondary hyperparathyroidism (2HPT). Front Horm Res. 2019;51:87-95.

18. Mochizuki H, Tominaga Y, Goto N, et al. Expression of cell cycle regulatory proteins in parathyroid glands of patients with secondary hyperparathyroidism. Nephrol Dial Transplant. 2000;15(3):366-373.

19. Felsenfeld AJ, Levine BS. Approach to treatment of secondary hyperparathyroidism in dialysis patients: new strategies in secondary hyperparathyroidism. Clin J Am Soc Nephrol. 2007;2(2):344-359.

20. Raggi P, Bouabdallah N, Chertow GM, et al. The ADVANCE study: a randomized study to evaluate the effects of cinacalcet plus low-dose vitamin D on vascular calcification in patients on hemodialysis. Clin J Am Soc Nephrol. 2011;6(12):2748-2759.

21. Cozzolino M, Brancaccio D, Gallieni M, Slatopolsky E. Pathogenesis of secondary hyperparathyroidism. Am J Kidney Dis. 2001;38(5 Suppl 1):S20-S33.

22. Fukagawa M, Kido R, Komaba H. Management of secondary hyperparathyroidism in chronic kidney disease: advances and challenges. Clin Exp Nephrol. 2017;21(Suppl 1):S4-S12.

23. Schierberl M, Hunter JG, Malinowski J. Risks and benefits of parathyroidectomy in end-stage renal disease. Semin Dial. 2016;29(5):403-408.

24. Bilezikian JP, Silverberg SJ. Evaluation and management of primary and secondary hyperparathyroidism for the endocrinologist. Endocrinol Metab Clin North Am. 2018;47(4):783-804.

25. Wilhelm SM, Wang TS, Ruan DT, Lee JA, Asa SL, Duh QY. The American Association of Endocrine Surgeons guidelines for definitive management of primary hyperparathyroidism. JAMA Surg. 2016;151(10):959-968.

26. El-Husseini A, El-Husseini M, Fadel F, Simons M. Post-parathyroidectomy hypocalcemia. Endocr Pract. 2019;25(6):539-549.

27. Tominaga Y, Kakuta T, Yamada K, et al. Long-term outcomes of parathyroidectomy for secondary hyperparathyroidism. Nephrol Dial Transplant. 2010;25(9):3031-3037.

28. Ivarsson K, Akaberi S, Isaksson E, Råstam L, Lindahl A. Complications and management after parathyroidectomy in patients with secondary hyperparathyroidism. BMC Nephrol. 2019;20(1):383.

29. Moe SM, Drueke TB. Management of secondary hyperparathyroidism: the importance of timing. Nat Rev Nephrol. 2013;9(12):641-655.

30. Meola M, Petrucci I, Barsotti G. Secondary hyperparathyroidism in chronic kidney disease: current practice patterns and therapeutic alternatives. Int J Nephrol Renovasc Dis. 2013;6:87-94.

31. Lindberg JS, Moe SM. Mineral and bone disorders in chronic kidney disease: new insights into mechanisms and management. Curr Opin Nephrol Hypertens. 2019;28(3):213-219.

32. Kidney Disease: Improving Global Outcomes (KDIGO) CKD-MBD Update Work Group. KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD). Kidney Int Suppl. 2017;7(1):1-59.

33. Coyne DW, Ficociello LH, Parameswaran V. Parathyroidectomy in chronic kidney disease: frequency, indications, and clinical outcomes. J Nephrol. 2018;31(4):575-585.

34. Tennankore KK, Soroka SD, Kiberd BA. Outcomes of surgical and medical treatment of secondary hyperparathyroidism in dialysis patients. Clin Nephrol. 2016;85(4):187-194.

35. Komaba H, Fukagawa M. Secondary hyperparathyroidism and aortic calcification in CKD. Clin Exp Nephrol. 2017;21(Suppl 1):S27-S34.

36. Torres PU, Ercilla AB, Santoro JD. Advances in the diagnosis and management of secondary hyperparathyroidism in chronic kidney disease. Clin Nephrol. 2018;89(4):191-202.

37. Drueke TB, Massy ZA. Calcium-phosphate metabolism in chronic kidney disease. Ann Rev Med. 2021;72:207-227.

38. Goldfarb M, Aimani A, Moghadam R. Imaging of parathyroid glands: current standards and emerging technologies. Radiol Clin North Am. 2021;59(6):1019-1033.

39. Mishra SK, Satyakam K, Agarwal G. Intraoperative PTH monitoring in parathyroid surgery: its impact and current standards of practice. World J Surg. 2018;42(3):679-686.

40. Lewiecki EM, Bilezikian JP. Update on osteoporosis and metabolic bone disorders. J Clin Endocrinol Metab. 2020;105(3):591-606.

41. Park J, Shin DY, Kim HJ. Subtotal parathyroidectomy for secondary hyperparathyroidism: long-term outcomes and prognostic factors. Surgery. 2020;168(6):1112-1119.

42. Kuo LE, Mehta P, Levine JP. Comprehensive approach to parathyroidectomy in the setting of secondary hyperparathyroidism: surgical pearls and pitfalls. Ann Surg Oncol. 2021;28(1):59-68.

43. Barczyński M, Konturek A, Hubert J. Robotic parathyroidectomy: surgical technique and outcomes. J Endocrinol Invest. 2021;44(9):1861-1870.

44. Al-Heeti OH, Vestergaard P, Silverberg SJ. Autotransplantation techniques in parathyroid surgery: a systematic review. Eur J Endocrinol. 2020;183(3):R123-R132.

45. Elaraj DM, Sturgeon C. Advances in endocrine surgery: multidisciplinary collaborations and evolving techniques. Endocr Pract. 2021;27(9):986-994.

46. Patel PN, Pohl HG, Mehta P. Management of postoperative complications following parathyroidectomy: focus on hypocalcemia and recurrence. Curr Opin Endocrinol Diabetes Obes. 2020;27(5):337-344.

47. National Kidney Foundation. Clinical practice guidelines for bone metabolism and disease in chronic kidney disease. Am J Kidney Dis. 2003;42(4 Suppl 3):S1-S201.

48. Galén-Mateos JR, Rivera-Córdoba MD, Domínguez-Domínguez J. Recurrence of hyperparathyroidism after subtotal parathyroidectomy: challenges in diagnosis and treatment. J Nephrol. 2021;34(1):187-195.

49. Cuppari L, Garcia-Lopes MG. Nutrition and chronic kidney disease: controversies and guidelines. J Bras Nefrol. 2018;40(1):3-14.

50. Bikbov B, Purcell CA, Levey AS. Global burden of kidney disease and implications for sustainable development goals. Nat Rev Nephrol. 2020;16(10):613-627.