

Hyperparathyroidism, Causes and Treatment

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Introduction

Hyperparathyroidism is defined by excessive secretion of parathyroid hormone (PTH), not by deficiency of the hormone. That distinction is fundamental because low PTH with hypocalcemia describes hypoparathyroidism, a different disorder with different symptoms and treatment. The parathyroid glands normally maintain calcium and phosphate balance by sensing circulating ionized calcium and adjusting PTH secretion. When calcium falls, PTH promotes calcium release from bone, increases renal calcium reabsorption, reduces renal phosphate reabsorption, and stimulates production of active vitamin D, which supports intestinal calcium absorption. Hyperparathyroidism is classified as primary, secondary, or tertiary according to the source of the abnormal stimulation. In primary hyperparathyroidism, one or more glands secrete PTH inappropriately, commonly because of a benign adenoma or multigland hyperplasia; the National Institute of Diabetes and Digestive and Kidney Diseases reports that a single adenoma accounts for about eight of ten primary cases (NIDDK, 2026). Secondary disease is usually a compensatory response to chronic hypocalcemic or mineral imbalance, whereas tertiary disease reflects autonomous secretion after prolonged stimulation, often in advanced kidney disease.

Pathophysiology and Major Disease Types

Primary hyperparathyroidism begins within the glands themselves. Excessive PTH raises serum calcium by increasing bone turnover, renal calcium conservation, and vitamin-D-mediated intestinal absorption, while phosphate is often reduced because PTH promotes urinary phosphate loss. Most patients now have mild disease detected by routine laboratory testing rather than the severe skeletal manifestations described in older literature. Secondary hyperparathyroidism develops when an external disorder chronically stimulates otherwise responsive glands. Chronic kidney disease is a major cause because phosphate retention, reduced active vitamin D, and disturbed calcium balance drive sustained PTH secretion; vitamin D deficiency, malabsorption, bariatric surgery, or very low calcium intake can also contribute. In these conditions, serum calcium may be normal or low rather than high. Tertiary hyperparathyroidism may develop after prolonged secondary disease when enlarged glands become partly autonomous and continue secreting PTH despite correction of the original stimulus, including after kidney transplantation. These categories cannot be used interchangeably because the biochemical patterns, complications, and treatment priorities differ substantially (Bilezikian et al., 2022).

Clinical Manifestations and Diagnostic Pattern

Many people with primary hyperparathyroidism have no obvious symptoms and are identified after routine chemistry shows elevated calcium. When manifestations occur, they may include fatigue, muscle weakness, bone or joint discomfort, constipation, nausea, increased thirst and urination, kidney-stone pain, mood change, or difficulty concentrating. Severe hypercalcemia can cause dehydration, vomiting, confusion, cardiac rhythm disturbance, or acute kidney injury and requires urgent medical evaluation. NIDDK describes bone thinning and kidney stones as important complications of prolonged disease (NIDDK, 2026). Diagnosis is usually biochemical: repeated elevated albumin-adjusted or ionized calcium together with elevated or inappropriately normal PTH supports primary hyperparathyroidism because normal glands should suppress PTH when calcium is high. Kidney function, serum phosphate, and 25-hydroxyvitamin D help interpret the pattern. A 24-hour urine calcium assessment can assist in distinguishing primary disease from familial hypocalciuric hypercalcemia. Imaging with ultrasound, sestamibi scanning, or four-dimensional CT is used mainly to localize abnormal glands after a biochemical diagnosis and surgical decision, not to establish the diagnosis itself.

Complications and Evaluation of Target Organs

Excess PTH and hypercalcemia can affect the skeleton and kidneys even when a patient feels well, which is why evaluation extends beyond symptoms. Dual-energy X-ray absorptiometry is used to measure bone mineral density at clinically relevant sites, and vertebral imaging may be appropriate when fracture is suspected. Kidney assessment can include estimated glomerular filtration rate, urinary calcium, and imaging for nephrolithiasis or nephrocalcinosis. The physiological reason for these complications is straightforward: chronic PTH excess increases bone turnover and releases calcium, while the kidneys filter an increased calcium load that can contribute to stones. In secondary hyperparathyroidism caused by chronic kidney disease, the broader problem is chronic kidney disease-mineral and bone disorder, in which phosphate, calcium, vitamin D, PTH, bone health, and vascular calcification interact. Symptoms such as tingling around the mouth, carpopedal spasm, tetany, seizures, and laryngospasm point instead toward hypocalcemia and possible hypoparathyroidism. Keeping those opposite clinical patterns separate prevents diagnostic and therapeutic errors, especially after thyroid or neck surgery.

Primary Hyperparathyroidism: Surgery and Medical Management

Parathyroidectomy is the definitive treatment for primary hyperparathyroidism because it removes the abnormal gland tissue responsible for inappropriate PTH secretion. NIDDK notes that surgery is the only sure cure and is generally recommended for symptomatic patients or for asymptomatic

patients who meet guideline criteria involving calcium elevation, skeletal involvement, kidney complications, reduced renal function, or younger age (NIDDK, 2026). Experienced endocrine surgeons may perform focused surgery when a single adenoma is well localized or broader exploration when multigland disease is suspected. Patients with mild disease who do not undergo surgery require active monitoring rather than simple observation without follow-up; calcium, kidney function, bone density, and evidence of stones or fractures should be reassessed periodically. Cinacalcet can lower serum calcium by increasing sensitivity of the calcium-sensing receptor and may be used when surgery is inappropriate or declined. Bisphosphonates or other bone-directed therapies may improve bone density in selected patients but do not remove the source of excess PTH. Hydration and adequate, not severely restricted, calcium intake are also important.

Secondary and Tertiary Hyperparathyroidism

Treatment of secondary hyperparathyroidism focuses on the condition driving PTH elevation rather than removing a normal gland response automatically. Vitamin D deficiency or malabsorption may improve with correction of the underlying deficiency and adequate calcium intake, while chronic kidney disease requires coordinated management of phosphate, calcium, vitamin D status, kidney function, and bone-mineral risk. Depending on disease stage, treatment may include dietary phosphate control, phosphate binders, vitamin D analogues, calcimimetic therapy, and optimization of dialysis under nephrology guidance. Tertiary disease is different because prolonged stimulation has produced partly autonomous PTH secretion; persistent hypercalcemia after kidney transplantation or advanced renal disease may require calcimimetics or parathyroid surgery. The therapeutic objective therefore changes by disease type. Primary disease seeks to eliminate inappropriate gland secretion and protect bone and kidneys, secondary disease seeks to correct the stimulus and reduce excessive adaptive PTH activity, and tertiary disease seeks control of autonomous secretion. Treating every elevated PTH result as the same disorder risks worsening calcium or phosphate balance and overlooking the underlying disease process.

Conclusion

Hyperparathyroidism is a disorder of excessive PTH activity whose meaning depends on the biochemical and clinical context. Primary hyperparathyroidism usually arises from an adenoma or multigland hyperplasia and commonly produces high calcium with elevated or inappropriately normal PTH; secondary hyperparathyroidism is a compensatory response to conditions such as chronic kidney disease or vitamin D deficiency; tertiary disease reflects autonomous secretion after prolonged secondary stimulation. The distinction from hypoparathyroidism is essential because low PTH with hypocalcemia produces a different symptom pattern and often requires calcium and active vitamin D rather than parathyroid surgery. Diagnosis relies first on laboratory relationships among calcium, PTH, kidney function, phosphate, and vitamin D, followed by tests for skeletal and renal complications. Imaging helps localize abnormal glands before surgery rather than proving the disorder. Primary

disease can often be cured by parathyroidectomy, while medical therapy and monitoring have roles when surgery is not performed, and secondary or tertiary disease requires treatment directed at the underlying physiology (NIDDK, 2026; Bilezikian et al., 2022).

References

Bilezikian, J. P., Khan, A. A., Silverberg, S. J., et al. (2022). Evaluation and management of primary hyperparathyroidism: Summary statement and guidelines from the Fifth International Workshop. *Journal of Bone and Mineral Research*, 37(11), 2293–2314.

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