CONDITION

Parathyroid hyperplasia

Parathyroid hyperplasia is enlargement of several parathyroid glands that can cause excess parathyroid hormone.

WHAT IT IS
Enlargement and overactivity of several parathyroid glands

BODY SYSTEM
Hormones, bones, kidneys, and digestion

MAIN SYMPTOMS
Fatigue, muscle weakness, bone pain, kidney stones

WHAT CAUSES IT
Long-term stimulation, gland changes, or inherited syndromes

HOW IT IS DIAGNOSED
Blood tests and multiple enlarged glands found during surgery

MAIN TREATMENT
Cause-specific medicines, monitoring, or parathyroid surgery

CAN IT BE CURED?
Sometimes - surgery can remove overactive tissue

IS IT AN EMERGENCY?
Sometimes - severe high-calcium symptoms need urgent care

LONG-TERM OUTLOOK
Often manageable, but persistent causes can lead to recurrence

Overview

Parathyroid hyperplasia means that several parathyroid glands have become enlarged. Most people have four of these small glands in the neck, behind the thyroid gland. They make parathyroid hormone, often shortened to PTH.

PTH helps control calcium and phosphorus levels in your blood. It also affects your bones, kidneys, and vitamin D activity. When enlarged glands release too much PTH, the condition is called hyperparathyroidism.

Parathyroid hyperplasia usually affects all four glands, although they may not be enlarged equally. This is different from a parathyroid adenoma, which is a non-cancerous growth involving one gland. The distinction is not always clear before surgery.

There are three main settings:

Parathyroid hyperplasia matters because long-term hormone imbalance can weaken bones, contribute to kidney problems, and disturb calcium levels. Some people have no symptoms and are diagnosed after routine blood tests.

- Primary hyperparathyroidism: the glands become overactive without being driven by another medical problem
- Secondary hyperparathyroidism: another condition, most often chronic kidney disease, repeatedly stimulates the glands
- Tertiary hyperparathyroidism: after long-term stimulation, the glands continue releasing too much PTH even when the original trigger has improved

Symptoms

Symptoms come mainly from the effects of excess PTH, high or low calcium, and the condition causing the gland enlargement. Mild hyperparathyroidism may cause no noticeable symptoms.

When calcium is high, possible symptoms include:

Long-standing excess PTH can reduce bone strength, increasing the risk of fractures. Kidney stones are more typical of primary or tertiary hyperparathyroidism than secondary hyperparathyroidism.

In chronic kidney disease, calcium may be normal or low while phosphorus and PTH are high. Bone pain, muscle weakness, itching, and fractures can occur, but these symptoms may also have other causes related to kidney disease.

Very high calcium can cause dehydration, vomiting, marked weakness, confusion, or abnormal heart rhythms. This is uncommon, but it needs urgent assessment.

- Tiredness, low energy, or muscle weakness
- Poor concentration, low mood, or feeling mentally slowed
- Increased thirst and passing urine more often
- Constipation, nausea, reduced appetite, or abdominal discomfort
- Bone or joint pain
- Kidney stones

Causes and risk factors

Primary parathyroid hyperplasia can occur without a clear inherited cause. Changes within the glands allow several of them to produce too much PTH.

Less often, primary hyperplasia is linked to an inherited syndrome. Examples include multiple endocrine neoplasia type 1, called MEN1, and multiple endocrine neoplasia type 2A, called MEN2A. These syndromes can affect other hormone-producing organs as well. A family history of parathyroid, pituitary, pancreatic, thyroid, or adrenal tumours may be relevant.

Secondary parathyroid hyperplasia develops when the glands are repeatedly stimulated to maintain calcium balance. Chronic kidney disease is the most common setting. Damaged kidneys have difficulty removing phosphorus and activating vitamin D. These changes can lower calcium signals and raise PTH.

Other causes of prolonged stimulation include severe vitamin D deficiency, poor calcium absorption, and some digestive conditions or operations that interfere with nutrient absorption.

Tertiary hyperparathyroidism can develop after secondary hyperparathyroidism has been present for a long time. The enlarged glands begin working independently and may continue to release excess PTH. This can happen in advanced kidney disease or after a kidney transplant.

Risk depends on the underlying cause. Having an inherited syndrome, advanced chronic kidney disease, or long-lasting disturbances in calcium and vitamin D regulation raises the chance of multigland hyperplasia.

Diagnosis

Diagnosis begins with blood tests. These commonly measure calcium, PTH, kidney function, phosphorus, vitamin D, and sometimes magnesium. Because blood protein levels affect total calcium measurements, an adjusted calcium result or an ionised calcium test may be used.

The pattern of results helps identify the type of hyperparathyroidism. For example, high calcium with a PTH level that is high or not appropriately suppressed suggests primary hyperparathyroidism. In secondary hyperparathyroidism from kidney disease, PTH is raised while calcium may be low or normal.

Results are often repeated because hydration, medicines, kidney function, and laboratory differences can affect them. Urine calcium testing may help distinguish primary hyperparathyroidism from certain inherited causes of lifelong high blood calcium.

Bone-density scanning can check for bone loss. Kidney imaging may be used to look for stones or calcium deposits.

Ultrasound, sestamibi scanning, computed tomography, or other imaging may locate enlarged glands before surgery. These scans do not establish whether hyperparathyroidism is present, and they may miss multigland disease. Diagnosis is based mainly on biochemical tests.

It can be difficult to prove hyperplasia before an operation. Surgeons may identify several enlarged glands during surgery. The operative findings, blood-test pattern, and examination of removed tissue together support the diagnosis. Even under a microscope, the boundary between hyperplasia and some forms of multigland adenoma is not always exact.

Genetic counselling and testing may be offered when hyperparathyroidism occurs at a young age, affects several glands, recurs, runs in the family, or appears with other hormone-related tumours.

Treatment and management

Treatment is tailored to the type of hyperplasia, calcium level, symptoms, kidney function, bone health, age, and inherited risk. Not every person needs immediate surgery.

For primary hyperparathyroidism, follow-up may include calcium and kidney blood tests, bone-density scans, and checks for kidney stones. Surgery is the definitive treatment when the condition meets accepted surgical criteria or causes significant problems.

Because hyperplasia usually involves several glands, surgery is different from removing one adenoma. A surgeon may remove most parathyroid tissue while leaving a small amount, or remove all identified glands and place a small piece of parathyroid tissue elsewhere in the body. The approach depends on the cause and surgical findings.

Secondary hyperparathyroidism is managed by treating the ongoing mineral imbalance. In chronic kidney disease, this may involve controlling phosphorus, correcting vitamin D-related problems, using medicines that reduce PTH release, and adjusting dialysis care. These treatments require monitoring because calcium and phosphorus can move outside the desired range.

Surgery may be considered when severe secondary hyperparathyroidism does not respond adequately to medical treatment, especially when it causes persistent symptoms or mineral and bone complications.

Tertiary hyperparathyroidism may improve with medicines in some people, but surgery is often considered when high calcium and excess PTH continue.

After parathyroid surgery, calcium can fall quickly as bones take up calcium again. This is sometimes called hungry bone syndrome. Blood tests and temporary calcium or vitamin D treatment may be needed. Treatment should follow the surgical team's plan rather than being

started or changed without guidance.

Complications

Possible complications of untreated or poorly controlled hyperparathyroidism include:

In advanced kidney disease, long-term mineral imbalance can also contribute to abnormal calcium deposits in blood vessels and soft tissues.

Surgery can cause low calcium if too little working parathyroid tissue remains. Symptoms may include tingling around the mouth, numbness in the fingers, or muscle cramps. Injury to a nerve controlling the vocal cord is another recognised surgical risk, although specialist surgeons take steps to reduce it.

Hyperplasia can persist or return if enlarged tissue remains, an additional parathyroid gland is present, transplanted tissue becomes overactive, or the underlying stimulus continues.

- Reduced bone density, bone pain, or fractures
- Kidney stones or calcium deposits in the kidneys
- Worsening problems with calcium and phosphorus balance
- Muscle weakness and reduced physical function
- Severe high calcium, causing dehydration or confusion

Prevention

Many cases of primary parathyroid hyperplasia cannot be prevented. Inherited forms cannot be prevented through diet or lifestyle.

If you have an inherited syndrome, planned screening may find hormone changes before complications develop. Genetic counselling can explain what a test result may mean for you and your relatives.

Secondary hyperplasia may sometimes be limited by managing the underlying problem. In chronic kidney disease, regular monitoring of PTH, calcium, phosphorus, and vitamin D allows treatment to be adjusted over time. This does not guarantee prevention because kidney-related mineral changes can progress despite appropriate care.

Taking large amounts of calcium or vitamin D without medical advice is not a safe way to prevent parathyroid problems. These supplements can be helpful in selected situations but may be harmful when calcium is already high.

Living with it and what to expect

The course depends mainly on why the glands enlarged. Mild primary hyperparathyroidism may remain stable for years, but some people develop worsening calcium levels, bone loss, or kidney stones. Regular follow-up is used to detect these changes rather than relying only on symptoms.

After successful surgery, calcium and PTH often return toward the expected range. Energy, muscle symptoms, and bone health may improve gradually rather than immediately. Bone density can take months or years to change.

Multigland disease has a greater chance of persistence or recurrence than a straightforward single-gland adenoma. This is especially important with inherited syndromes, because remaining parathyroid tissue can enlarge later. Long-term blood-test follow-up may still be needed after surgery.

Secondary hyperparathyroidism often continues alongside chronic kidney disease. PTH levels may rise and fall as kidney function, dialysis, diet, and treatment change. The aim is ongoing control of mineral balance and protection of bones and other tissues, rather than a single permanent correction.

After a kidney transplant, secondary hyperparathyroidism may settle over time. In some people, the glands remain independently overactive and calcium stays high, which may require further treatment.

Most people can continue their usual daily activities. Follow-up may include blood tests, medication reviews, bone-density scans, kidney checks, or appointments with kidney, hormone, or surgical specialists.

When to get medical help

Arrange routine medical follow-up if you have known parathyroid hyperplasia and develop increasing thirst, frequent urination, constipation, fatigue, muscle weakness, bone pain, or possible kidney-stone symptoms. These changes may mean that calcium or PTH needs to be checked.

After parathyroid surgery, contact your care team promptly if you develop tingling around the mouth, new numbness, muscle cramps, or twitching. These can be signs of low calcium and may need a blood test and treatment adjustment.

Seek urgent medical care for severe or persistent side or back pain, especially with vomiting, fever, or blood in the urine. A kidney stone or infection may need prompt assessment.

Get emergency help for confusion, collapse, a seizure, severe weakness, repeated vomiting with dehydration, breathing difficulty, or sustained muscle spasms. These symptoms can occur with a serious calcium disturbance or another urgent condition.

Common questions

Is parathyroid hyperplasia cancer?

No. Hyperplasia is an increase in the amount of gland tissue, not cancer. Parathyroid cancer is rare and is a separate diagnosis.

Is it the same as a parathyroid adenoma?

Not usually. An adenoma is generally one non-cancerous overactive gland. Hyperplasia commonly affects several glands. Scans may not reliably tell the difference before surgery.

Can parathyroid hyperplasia go away on its own?

Primary hyperplasia usually does not disappear on its own. Secondary hyperplasia may improve if the condition stimulating the glands is corrected, although long-standing enlargement may not fully reverse.

Do all enlarged parathyroid glands need to be removed?

No. The need for surgery and the amount of tissue removed depend on the type of hyperparathyroidism, its effects, and the operative findings. Leaving some working tissue helps reduce the risk of permanent low calcium.

Can parathyroid hyperplasia come back after surgery?

Yes. Remaining or transplanted parathyroid tissue can enlarge, particularly when there is an inherited syndrome or an ongoing cause such as chronic kidney disease. Follow-up blood tests help detect recurrence.

Does diet cure parathyroid hyperplasia?

No specific diet cures it. Dietary advice depends on calcium, phosphorus, vitamin D, kidney function, and treatment. People with kidney disease may need individual guidance because general advice about calcium or phosphorus may not be suitable.

Sources

- Bilezikian JP, Khan AA, Silverberg SJ, and colleagues. "Evaluation and Management of Primary Hyperparathyroidism: Summary Statement and Guidelines from the Fifth International Workshop." Journal of Bone and Mineral Research . 2022;37(11):2293-2314.
- Wilhelm SM, Wang TS, Ruan DT, and colleagues. "The American Association of Endocrine Surgeons Guidelines for Definitive Management of Primary Hyperparathyroidism." JAMA Surgery . 2016;151(10):959-968.
- Kidney Disease: Improving Global Outcomes CKD-MBD Update Work Group. "KDIGO 2017 Clinical Practice Guideline Update for Chronic Kidney Disease-Mineral and Bone Disorder." Kidney International Supplements . 2017;7(1):1-59.
- Walker MD, Silverberg SJ. "Primary Hyperparathyroidism." Nature Reviews Endocrinology . 2018;14:115-125.
- National Institute of Diabetes and Digestive and Kidney Diseases. "Primary Hyperparathyroidism." Patient information from the US National Institutes of Health.
- Thakker RV, Newey PJ, Walls GV, and colleagues. "Clinical Practice Guidelines for Multiple Endocrine Neoplasia Type 1." Journal of Clinical Endocrinology and Metabolism . 2012;97(9):2990-3011.