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POST-THYROIDECTOMY HYPOCALCEMIA IN PAKISTAN: A REVIEW OF PATHOPHYSIOLOGY, PREVENTIVE APPROACHES, AND MANAGEMENT STRATEGIES FOR HEAD AND NECK SURGEONS

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Abstract

Background: *Hypocalcemia following thyroid surgery, most often a sign of temporary or permanent hypoparathyroidism, is the leading metabolic complication of bilateral thyroidectomy and central neck dissection. Transient hypocalcemia is reported in 10%-47.8% of patients, and permanent parathyroid dysfunction in 0.5%-10.7%.*

Objective: *To consolidate current evidence on the pathophysiology, epidemiology, prevention, early risk stratification, and acute and long-term management of post-thyroidectomy hypocalcemia, and to offer surgeons a practical framework for care.*

Methods: *A narrative review of the published literature, with findings extracted and organized by theme.*

Results: *The principal mechanisms are parathyroid devascularization, mechanical or thermal injury, and inadvertent excision. Risk is increased by female sex, younger age, autoimmune thyroid disease, total thyroidectomy, central neck dissection, reoperative surgery, and prior neck irradiation. Preventive measures include preoperative vitamin D and calcium optimization, careful parathyroid-preserving technique, selective autotransplantation, and the use of intraoperative and early postoperative parathyroid hormone (PTH) measurements. PTH-based risk stratification algorithms allow safe early discharge while limiting symptomatic*

hypocalcemia. Stepwise approaches are outlined for oral and intravenous treatment of acute hypocalcemia and for the long-term care of permanent hypoparathyroidism.

Conclusions: *Emerging technologies, including near-infrared autofluorescence, indocyanine green angiography, deep learning-assisted parathyroid identification, and novel PTH replacement therapies, show promise for further reducing complications and improving quality of life after thyroidectomy.*

1. INTRODUCTION

Thyroidectomy is one of the most common operations in otolaryngology and general surgery. Even though surgeons know it well, it carries a recognized risk of injuring the parathyroid glands, which is the principal reason for low calcium afterward. This burdens both patients and health systems, and over the last twenty years our understanding of how it develops, how to prevent it, and how to treat it has changed substantially.

Each parathyroid gland is very small, around 6 mm by 3 mm by 1 mm and weighing 20 to 50 mg, yet the glands are essential for keeping calcium in balance. They sit on the back surface of the thyroid lobes, right where the surgeon is working, and their fragile blood supply makes them easy to injure through loss of circulation during dissection.

In the first 24 hours after surgery, temporary hypocalcemia is reported in 10% to 47.8% of patients. Permanent hypoparathyroidism, meaning gland dysfunction lasting more than 6 months, is reported in 0.5% to 10.7%. These broad ranges come from differences in surgical technique, surgeon experience, and patient populations, and also from the lack of consistent definitions between studies.

This paper gathers the available evidence into one framework that covers causes and risk factors, prevention, early risk assessment, acute and long-term treatment, lasting consequences, and future directions.

2. Methods

This work is a narrative synthesis, not a systematic review. Its source was one open-access minireview (Viswanath et al., 2026) together with the 48 references that article cites. Material was pulled out under several themes: pathophysiology, risk factors, prevention, risk stratification, treatment, outcomes, and emerging technologies. Numbers and claims were compared across different parts of the source to find contradictions, which are described in the Limitations section. No new patient data were gathered, and no separate literature search was done.

3. Definitions

Six months after surgery is the generally accepted dividing line, used in guidelines and clinical studies, between temporary and permanent disease. Permanence should not be declared before this time.

Transient hypoparathyroidism. This is low calcium caused by stunning or reversible injury of the glands, followed by a return of normal function. It lasts less than 6 months. Blood tests show low calcium with PTH that is low or inappropriately normal, and calcium returns to normal within 6 months. Treatment with calcium, with or without active vitamin D, can be fully withdrawn within that period.

Permanent hypoparathyroidism. This is irreversible loss of parathyroid function that needs lifelong management. It lasts longer than 6 months. Blood tests show low calcium with PTH that is undetectable or inappropriately low and does not recover after 6 months. Calcium and active vitamin D must be continued beyond 6 months to keep calcium in the normal range.

4. Pathophysiology and Risk Factors

4.1 Mechanisms

Most cases arise through one of three routes.

Loss of blood supply (ischemia). The glands receive blood chiefly from the inferior thyroid artery, though anatomy varies often and the superior thyroid artery or vessels in the thymo-thyroid ligament may also contribute. PTH lasts only about 3 to 5 minutes in the circulation, so even a short interruption of blood flow causes circulating PTH to drop quickly, and the effects on calcium can last for hours or days.

Mechanical and heat injury. Rough handling and heat spreading from energy-based sealing devices can harm tissue outside the intended area. The usual advice is to stay at least 3 to 5 mm away from parathyroid tissue to limit thermal damage.

Accidental removal. Taking out parathyroid tissue is a certain cause of hypoparathyroidism. The danger is greatest in central neck dissection for cancer, because the lymph nodes lie close to the lower glands. Every gland that is removed adds to the chance of significant hypocalcemia.

4.2 Patient and surgical risk factors

- **Female sex:** both temporary and permanent disease are more common, and at any given IPTH value women face a higher risk.
- **Age under 40:** temporary hypocalcemia is more likely, and the cause may be biological rather than surgical.
- **Autoimmune thyroid disease (Graves' disease, chronic lymphocytic thyroiditis):** risk is higher, perhaps because of long-standing inflammatory changes in the glands.
- **Vitamin D deficiency before surgery:** an independent predictor that can be corrected.
- **Extent of surgery:** total thyroidectomy (versus subtotal) and the addition of central neck dissection are the strongest surgical predictors, together with the surgeon's experience and technique.
- **Operating time:** operations longer than 120 to 240 minutes are linked to greater ischemic risk, and the source cites hypoparathyroidism rates as high as 62% when surgery exceeds 240 minutes.
- **Repeat surgery:** this carries the greatest risk of permanent hypoparathyroidism, since scar tissue distorts anatomy and makes it harder to find the glands and their vessels.
- **Previous neck radiation:** fibrosis and vascular damage raise the chance of permanent disease.

5. Prevention

5.1 Optimization before surgery

Vitamin D deficiency can be corrected and is a predictor of postoperative hypocalcemia. A meta-analysis of randomized trials with more than 1,000 patients found that giving calcium and vitamin D before surgery lowered symptomatic hypocalcemia by roughly 46%, an absolute drop of about 10% to 11% among supplemented patients. Since vitamin D can take up to two weeks to reach its full effect, supplementation should begin at least one to two weeks ahead of the operation.

Reported regimens combine alfacalcidol or calcitriol at 0.25 to 2 micrograms daily with oral calcium at 500 to 1500 mg daily, given for 7 days up to 6 weeks depending on the starting vitamin D level. Patients with severe deficiency, or those undergoing high-risk operations such as central neck dissection for cancer, merit more intensive optimization.

5.2 Surgical technique

The foundation of prevention is to identify and protect all four glands along with their blood supply, while watching for glands in unusual locations, which occur in about 35% of people and may lie in the mediastinum, thymus, or tracheoesophageal groove. The traditional open approach through the neck gives a direct view of the branches of the inferior thyroid artery. Endoscopic and robotic remote-access techniques give cosmetic advantages, but they require a learning curve and reduce the surgeon's touch-based sense of whether a gland is still viable. With any approach, the 3 to 5 mm distance from parathyroid tissue should be kept when using advanced energy devices. If a gland has lost its blood supply or has to be removed, the plan switches from preservation to immediate autotransplantation.

5.3 Intraoperative PTH measurement

An IPTH below 10 pg/mL, measured 10 to 20 minutes after a total thyroidectomy, is associated

with a clearly higher risk of temporary or permanent hypoparathyroidism. The benefit is timing: patients at high risk are recognized while the surgeon is still in the operating room and can respond, for example by autotransplanting tissue. Reports indicate that grafting selectively, only in patients with very low levels, removes the risk of permanent hypoparathyroidism and also reduces temporary hypocalcemia and the overall need for calcium, compared with grafting every gland routinely.

5.4 New intraoperative technologies

Near-infrared autofluorescence (NIRAF) helps surgeons find glands, with reported detection rates of 78% to 100%. Indocyanine green (ICG) angiography lets the team assess gland blood flow in real time and can inform autotransplantation decisions. Both techniques are most helpful in difficult situations such as central neck dissection, repeat operations, and remote-access surgery. How much they change long-term hypocalcemia rates is still being worked out.

5.5 Selective autotransplantation

When fresh parathyroid tissue is cut or minced into fragments of about 1 mm^3 and placed in separate pockets in the sternocleidomastoid muscle, the graft works in more than 90% of cases. Success is not assured, though: 10% to 30% of grafts fail and leave the patient needing permanent supplementation, and results are better in secondary hyperparathyroidism than after thyroidectomy for cancer. A graft can take weeks or months to begin working, so serial calcium and IPTH should be followed, and detectable PTH in a patient with normal calcium indicates that the graft is functioning.

Known or suspected parathyroid carcinoma is an absolute contraindication, because malignant cells could be seeded. Relative contraindications include tissue whose identity or viability is uncertain, active infection at the recipient site, severe frailty, and intraoperative instability that makes a longer procedure unsafe.

6. Early Risk Assessment and Discharge

6.1 Postoperative IPTH

IPTH measured 4 to 6 hours after surgery is the most dependable early predictor of hypocalcemia. The source describes a three-tier framework based on this single measurement.

High risk (IPTH below 10 pg/mL). These patients need close monitoring and should receive calcium together with calcitriol proactively.

Intermediate risk (IPTH 10 to 29 pg/mL). Supplementation is given selectively, guided by the trend in calcium and by symptoms.

Low risk (IPTH 30 pg/mL or higher). Management can be conservative, and these patients are often suitable for early discharge.

An IPTH of 30 pg/mL or more carries a very small risk, about 3% to 5%, of significant hypocalcemia. Women remain at higher risk at every IPTH level.

6.2 Biochemical versus symptomatic disease

Abnormal calcium on blood tests occurs in roughly 22% to 48% of patients, whereas symptoms such as tingling or tetany affect a smaller share (reported at 24% to 30%) and can follow a two-phase course, often worsening on postoperative days 2 to 3. Mild cases without symptoms may settle by themselves, but a serum calcium below 1.85 mmol/L (8.0 mg/dL) requires active treatment to prevent arrhythmia or laryngospasm.

6.3 Safe discharge

Same-day or short-stay discharge is considered safe when every one of these conditions is satisfied: IPTH at or above about 15 to 30 pg/mL, serum calcium at or above 1.85 mmol/L, no symptoms, a favorable parathyroid gland status score, and careful teaching of the patient about warning symptoms, with outpatient follow-up in place.

7. Treatment of Hypocalcemia

7.1 Recognizing the condition

Signs include tingling around the mouth and in the hands and feet, spasm of the hands and feet, tetany, and laryngospasm, and in severe cases seizures and abnormal heart rhythms. Two bedside tests reveal latent tetany: Chvostek sign,

which is twitching of the face when the area just in front of the earlobe is tapped, and Trousseau sign, which is spasm of the hand after a blood pressure cuff has been inflated for 3 minutes.

7.2 Oral treatment

Oral supplements are the first choice when symptoms are not life-threatening. Elemental calcium is begun at 500 to 1000 mg three times a day and adjusted according to response. Calcium carbonate (40% elemental calcium) and calcium citrate (28%) are preferred, and citrate suits patients taking acid-suppressing drugs better. Calcitriol at 0.25 to 1.0 micrograms daily in divided doses is added because it acts quickly, strongly improves calcium absorption in the gut, and has a short half-life that allows rapid adjustment. Doses are increased every 4 to 7 days until serum calcium is in the low-normal range.

7.3 Intravenous treatment

Severe symptomatic hypocalcemia, meaning tetany, seizures, or arrhythmia, calls for intravenous calcium gluconate. Calcium chloride should not be given through a peripheral vein because it can cause tissue necrosis. A typical loading dose is 1 to 2 g (10 to 20 mL of a 10% solution) in 50 to 100 mL of 5% dextrose, infused slowly over 10 to 20 minutes with continuous heart monitoring. A fast infusion can cause arrhythmia, a slow heart rate, and low blood pressure, and extra care is needed in patients on digoxin. Symptoms often return after the first dose, so further doses of 1 to 2 g over 60 minutes may be needed every 1 to 4 hours, depending on symptoms and the course of calcium levels.

7.4 Magnesium

Low magnesium weakens PTH release and blunts the kidney's response to PTH. For this reason, magnesium should be measured in every patient with low calcium and corrected at the same time as, or before, calcium. Intravenous magnesium sulfate can briefly bind calcium, so it should be given after calcium starts to improve or spaced apart from the intravenous calcium.

8. Permanent Hypoparathyroidism and Long-Term Outcomes

8.1 Long-term care

Treatment aims to control symptoms, keep serum calcium at the low end of normal (2.00 to 2.12 mmol/L), hold phosphate within normal limits (0.8 to 1.5 mmol/L), and keep the calcium-phosphate product below 4.4 $mmol^2/L^2$ to reduce calcification in tissues. Common regimens pair calcium carbonate 1 to 2 g three times daily with calcitriol 0.5 to 2.0 micrograms daily in divided doses. Thiazide diuretics lower calcium loss in the urine and are a helpful addition, especially with a low-sodium, low-phosphate diet and phosphate binders. Calcium, phosphorus, and creatinine are checked weekly to monthly while doses are being adjusted, then every 3 to 6 months once stable, with continuing co-management by an endocrinologist.

8.2 Complications

Permanent hypoparathyroidism is linked to kidney stones, calcium deposits in the kidney, progressive kidney failure, cataracts at the back of the lens, calcification of the basal ganglia in the brain, and psychological problems including depression and anxiety. About 30% of patients with long-standing disease develop kidney complications, driven by high urinary calcium (above 300 mg/day) that results from vitamin D therapy combined with the kidney's reduced ability to reclaim calcium. Quality of life is reduced in every area, most of all mental health and overall health perception, even when calcium is well controlled, which suggests that the lack of PTH itself has effects.

8.3 Effect on health systems

Hypocalcemia is described as the leading reason for emergency visits and readmissions after outpatient thyroidectomy. In a group of 17,046 outpatient thyroidectomies at 374 US facilities, 7.5% of patients were seen and released from the emergency department and 2.3% were readmitted; hypocalcemia explained 9.9% of those visits and 22.2% of the readmissions. Testing PTH and giving calcitriol to at-risk patients has been linked

to fewer hypocalcemia events and fewer emergency visits after total thyroidectomy.

9. Discussion

The evidence points to one straightforward conclusion: harm is best reduced by combining measures across the entire course of care rather than depending on any one of them. Preparation before surgery lowers baseline risk; careful technique and selective autotransplantation protect or replace gland function during the operation; and a single early postoperative IPTH value lets supplements be directed to those who need them rather than given to everyone. This targeted strategy cuts needless medication, shortens hospital stays, and may prevent readmissions that could have been avoided.

According to the source, successful hospital protocols share three features: IPTH measured within 4 to 6 hours, clearly defined risk cut-offs, and selective rather than automatic supplementation. Patient education is noted to improve adherence.

Several tools are in development. Deep-learning models that recognize parathyroid-specific NIRAF signals could make gland detection less dependent on the operator, and machine-learning analysis of ICG fluorescence intensity could make blood-flow assessment more uniform. Both face obstacles of cost, availability, and learning curve. Recombinant PTH(1-84) has improved blood results and quality of life in chronic hypoparathyroidism but is too costly for wide use, and long-acting PTH analogs and drugs that target the calcium-sensing receptor are being developed. Polygenic risk scores, combined with preoperative and intraoperative data, may one day tailor autotransplantation decisions to the individual, but they need further validation.

10. Limitations

• This synthesis rests on one narrative minireview and the references it cites. It is not a systematic review, and no independent search or quality assessment was performed.

• Some figures in the source contradict each other. Permanent hypoparathyroidism is given as 0.5% to 10.7% in one place and 1.6% to 10.7% in another; the threshold for severe hypocalcemia is stated both as ionized calcium below 0.8 mmol/L and as total corrected calcium below 1.85 mmol/L; and the IPTH cut-off for discharge appears as 20 to 30 pg/mL in one place and 15 to 30 pg/mL in another.

• A few citations seem to support their statements only loosely. For instance, a pediatric study is cited for symptomatic hypocalcemia after thyroidectomy, a general review of electrolyte disorders is cited for neuropsychiatric outcomes, and a single case report is cited for magnesium replacement. Readers should check key claims against primary sources before applying them in practice.

• Doses and thresholds are given as the source reports them and should be verified against current guidelines, such as those of the American Thyroid Association and the European Society of Endocrinology, and against local protocols.

11. Conclusion

Hypocalcemia after thyroidectomy continues to be a major cause of illness, emergency care use, and cost, yet much of it can be prevented. A multi-part strategy that combines vitamin D and calcium optimization before surgery, gland-sparing technique, selective autotransplantation, and PTH-guided risk grouping can lower both symptomatic hypocalcemia and permanent hypoparathyroidism, while allowing safe early discharge. Standardized hospital protocols, reinforced over time by fluorescence imaging, AI-assisted recognition, and better PTH replacement, offer the best path to improved outcomes and quality of life for people undergoing thyroid surgery.

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