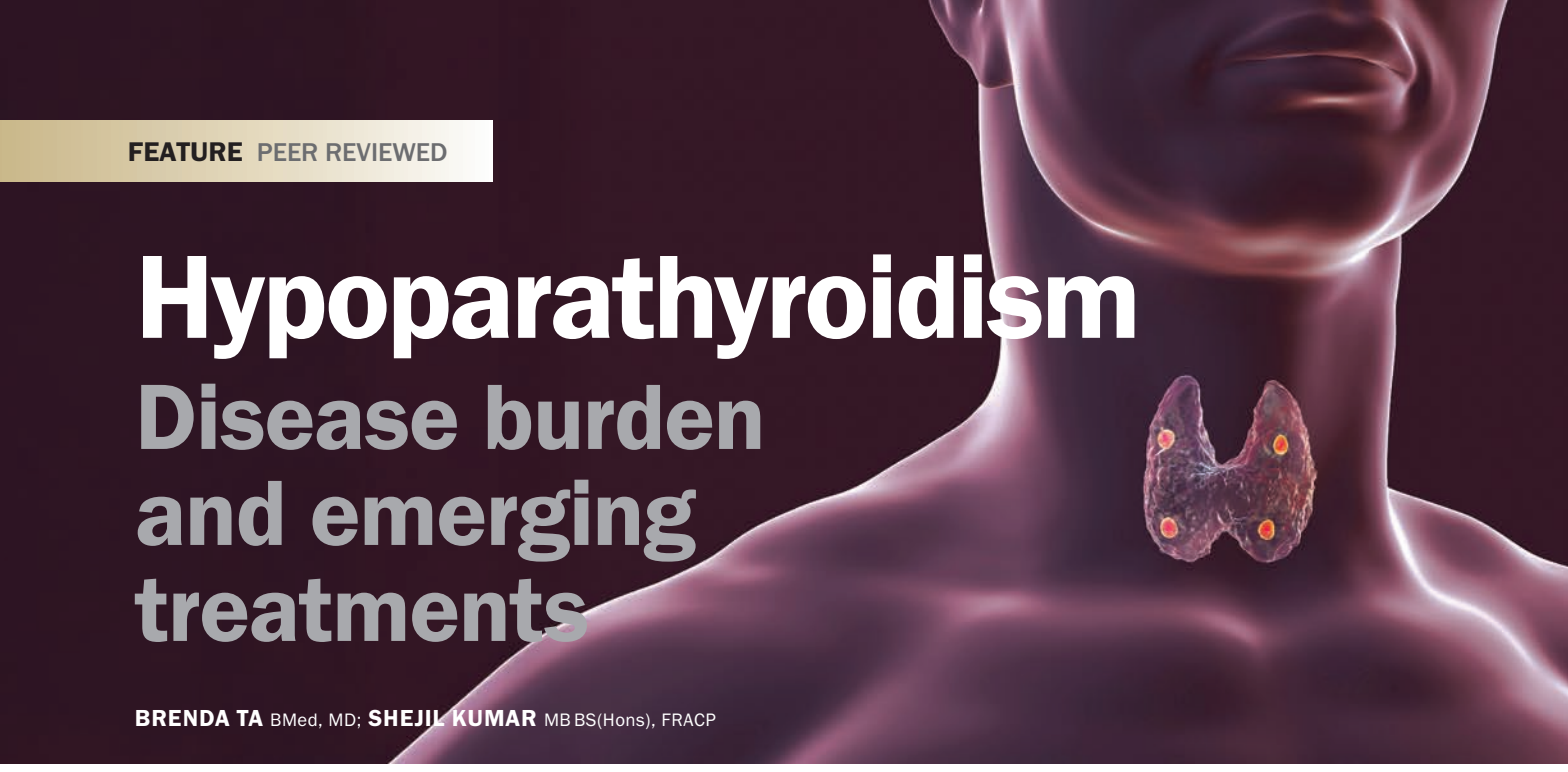

Hypoparathyroidism
Disease burden and emerging treatments

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Hypoparathyroidism is characterised by low or inappropriately normal parathyroid hormone (PTH) levels despite hypocalcaemia. Standard-of-care calcium and calcitriol supplementation normalises serum calcium but has several limitations. Palopegteriparatide, a novel PTH replacement therapy, improves quality of life and reduces reliance on conventional therapy but is not yet PBS subsidised in Australia.

Parathyroid hormone (PTH) is the key regulator of serum calcium homeostasis. Hypoparathyroidism, most commonly arising following anterior neck surgery, is characterised by low or inappropriately normal levels of PTH despite the presence of hypocalcaemia. It is increasingly recognised as a complex multisystem disease associated with significant morbidity and impaired quality of life.[1]

The current standard of care is conventional treatment involving calcium and active vitamin D (calcitriol) supplementation. Although this approach can help normalise calcium levels, it is associated with a high pill burden, has not been consistently shown to improve quality of life and may exacerbate complications such as hypercalciuria.[1] Palopegteriparatide, a long-acting PTH analogue, was approved by the TGA in early 2025 based on its efficacy and safety in a randomised, placebo-controlled trial. This article reviews current approaches to the diagnosis and management of hypoparathyroidism, with an emphasis on disease burden and emerging PTH-based therapies.

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Key points

- **Hypoparathyroidism is characterised by low or inappropriately normal levels of parathyroid hormone in the presence of hypocalcaemia.**
- **Post-surgical causes (e.g. thyroidectomy, cervical lymph node dissection, head and neck surgery) account for most cases, followed by nonsurgical causes (e.g. idiopathic, autoimmune, infiltrative, mineral deposition, hereditary).**
- **Hypoparathyroidism is a multisystem disorder associated with both disease- and treatment-related complications affecting the renal, cardiovascular, skeletal and neuropsychiatric systems.**
- **Conventional therapy with calcium and calcitriol is the standard of care; however, it carries significant pill burden, may exacerbate complications and has not been shown to improve quality of life.**
- **Parathyroid hormone replacement therapy with once-daily palopegteriparatide has demonstrated benefits in maintaining normocalcaemia, reducing reliance on conventional therapy and improving quality of life. It is TGA approved and currently under consideration for subsidised access via the PBS.**

Calcium homeostasis

Calcium is an intracellular secondary messenger that is critical in many physiological processes including muscle contraction, neurotransmitter release, and endocrine and exocrine secretion. Serum calcium levels are tightly regulated within a narrow physiological range through co-ordinated actions of PTH and vitamin D.[1,2]

PTH is the body's primary defence against low circulating calcium levels and acts to raise serum calcium concentrations through various mechanisms. Parathyroid chief cells sense circulating calcium concentrations via the calcium-sensing receptor (CaSR). High circulating ionised calcium levels increase stimulation of CaSRs and result in downregulation of PTH release. Low circulating ionised calcium levels reduce CaSR signalling, triggering release of preformed PTH. In the bone, PTH stimulates bone resorption and turnover to mobilise

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skeletal calcium into the circulation. In the kidneys, PTH increases tubular calcium reabsorption and stimulates activation of 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D (calcitriol). This in turn promotes intestinal calcium and phosphate absorption.[1]

PTH also plays a role in regulating phosphate and magnesium homeostasis.[1] Together with fibroblast growth factor 23 (FGF23), PTH inhibits renal phosphate reabsorption to decrease serum phosphate levels. PTH also enhances magnesium reabsorption in the distal convoluted tubule of the kidney. However, both hypermagnesaemia and hypomagnesaemia can result in hypocalcaemia. Hypermagnesaemia can inhibit PTH release, whereas severe, prolonged hypomagnesaemia can cause functional PTH resistance.[1]

In summary, hypoparathyroidism leads to low bone turnover, increased fractional urinary excretion of calcium, reduced levels of calcitriol (and hence intestinal calcium absorption) and, ultimately, hypocalcaemia.[1] Hypoparathyroidism is also associated with hyperphosphataemia and hypomagnesaemia.

Epidemiology

Hypoparathyroidism is a rare condition. Registry data from the USA and Denmark estimate a prevalence of about 20 to 40 cases per 100,000 person-years.[3] At least three-quarters of cases occur after anterior neck surgery, such as thyroidectomy, parathyroidectomy, cervical lymph node dissection or head and neck tumour resections. The remainder of nonsurgical cases may be idiopathic or due to other causes, including autoimmune, infiltrative or mineral deposition disease, as well as hereditary, congenital or drug-induced disorders (Box 1).[4] In contrast, pseudohypoparathyroidism is characterised by hypocalcaemia with elevated PTH levels due to target tissue resistance to PTH signalling. A detailed discussion of pseudohypoparathyroidism is beyond the scope of this article. Differential diagnoses for hypocalcaemia are presented in the Flowchart.

Post-surgical hypoparathyroidism

Although transient hypoparathyroidism is common after anterior neck surgery and occurs in about 25% of patients, most patients experience PTH recovery within one month. Permanent hypoparathyroidism occurs in less than 5% of patients, defined as hypocalcaemia persisting beyond 12 months post-surgery.[5,6]

Risk factors for permanent hypoparathyroidism include a postoperative day one PTH level of less than 0.5 pmol/L (sensitivity 100%, specificity 85%), as well as surgical factors such as greater extent of surgery (e.g. extensive neck dissection), inadvertent parathyroid removal and lower surgeon case volume.[6,7] In the absence of national or international guidelines, institutions should employ their own specific protocol for the screening, management and follow up of hypoparathyroidism post-neck surgery, which may incorporate pre-emptive calcium with or without calcitriol supplementation.

Clinical manifestations of hypoparathyroidism

Chronic hypoparathyroidism is a multisystem disease associated with significant morbidity and impaired quality of life.[6]

1. Aetiology of hypoparathyroidism

Surgical*
- Post-thyroidectomy (transient or permanent)
- Parathyroidectomy
- Cervical lymph node dissection
- Resection of head or neck cancer

Autoimmune
- Type 1 autoimmune polyglandular syndrome (associated with chronic mucocutaneous candidiasis and primary adrenal insufficiency)
- Isolated hypoparathyroidism (antibodies against CaSR)

Infiltrative
- Granulomatous
- Sarcoidosis
- Amyloidosis
- Metastatic disease

Mineral deposition
- Copper
- Iron

Drugs
- Cinacalcet
- Immune checkpoint inhibitor therapy

Genetic variants
- DiGeorge syndrome
- *CASR* gene mutation including autosomal dominant hypocalcaemia

Neck radiation therapy

Hypomagnesaemia or hypermagnesaemia

Idiopathic

Abbreviation: CaSR = calcium-sensing receptor.
* Secondary to parathyroid damage or disruption of parathyroid blood supply.

Complications can be disease-related but are also exacerbated by use of conventional treatment (calcium with or without calcitriol supplementation). Careful monitoring is therefore essential.

Renal complications

Hypoparathyroidism results in reduced renal calcium reabsorption and thus increased fractional urinary excretion of calcium. Conventional therapy can further exacerbate this by increasing the filtered calcium load. Hence, patients with hypoparathyroidism have about a twofold higher risk of developing renal impairment (estimated glomerular filtration rate <30 mL/min/1.73 m^2) and nearly a fivefold higher risk of renal stones.[1,6,8,9] Risk factors for renal complications include longer disease duration (>12 years), more prevalent episodes of hypercalcaemia (≥4) and higher serum calcium–phosphate product (>2.8 mmol/L).[10]

Cardiovascular complications

Chronic hypocalcaemia is associated with electrocardiographic abnormalities and a twofold higher risk of cardiovascular disease, cardiac arrhythmias and stroke, particularly in patients with nonsurgical hypoparathyroidism.[10,11] Similar to renal complications, risk increases with longer disease duration and increased number of hypercalcaemic episodes.[10]

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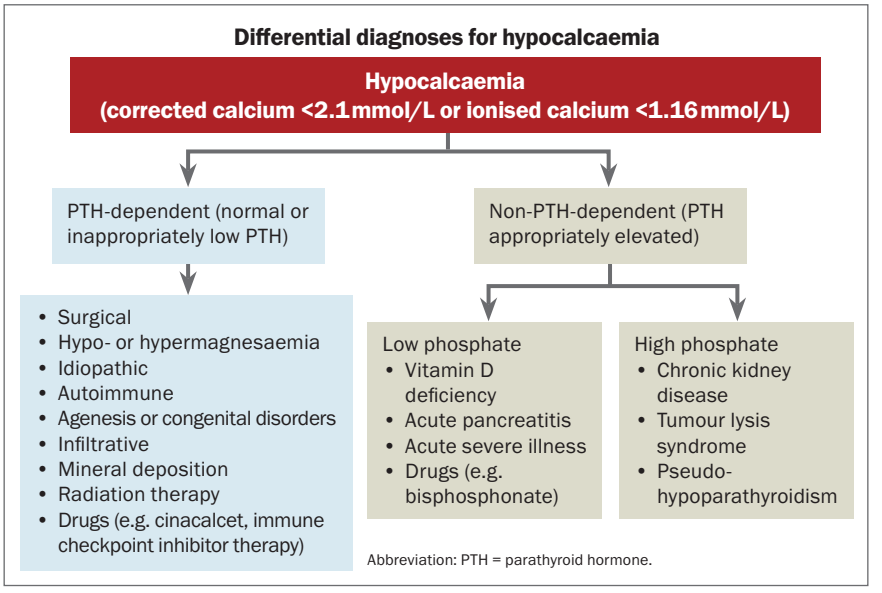

Skeletal manifestations

Hypoparathyroidism is associated with reduced bone turnover, leading to a positive bone balance and increased bone mineral density across all sites, as seen on dual energy x-ray absorptiometry and more specialised bone compartmental imaging.[1,4,6,10] However, the clinical significance of such skeletal manifestations remains uncertain due to limited and often conflicting data. It is unclear whether high bone mass confers resistance to fractures, or low bone turnover leads to compromised structural integrity. One study reported a twofold increase in upper limb fractures in patients with nonsurgical hypoparathyroidism compared with controls.[11] Individuals with hypoparathyroidism may also have an increased risk of vertebral fractures, particularly postmenopausal women or individuals receiving anticonvulsant therapy.[12,13]

Nonetheless, larger registry studies have not consistently confirmed an excess fracture risk in those with hypoparathyroidism.[14,15] A recent large Swedish outpatient registry study (n = 1915 patients with chronic hypoparathyroidism) did not show an increased risk of major osteoporotic fracture.[16] Vertebral fracture risk was higher (hazard ratio, 1.5), while hip fracture risk was lower (hazard ratio, 0.7) compared with controls, although analyses may have been limited by ascertainment and detection bias. Hence, the fracture risk associated with hypoparathyroidism remains controversial.

Neuropsychiatric complications and impaired quality of life

Neuropsychiatric and cognitive symptoms are common with hypoparathyroidism and include depression, fatigue, impaired memory and concentration, sleep disturbances and muscle weakness. Additionally, according to disease-specific quality of life assessments, patients report a high burden of paraesthesia, pain and cramps, and loss of vitality.[17,18] These symptoms often persist despite normalisation of calcium levels with conventional treatment and contribute substantially to reduced quality of life.[4,14,18]

High disease burden can also affect an individual's ability to work, with a Norwegian study reporting that up to 40% of patients with hypoparathyroidism receive permanent or temporary social security benefits, compared with 10% of the general population.[19] Patients often feel that their disease and the associated complications are poorly understood, leading to greater psychological distress.[20] Additionally, hospital admissions due to hypocalcaemia or hypercalcaemia are frequent and contribute to the overall disease burden on patients and the healthcare system.[21]

Other manifestations

Intracranial calcifications are observed in over 50% of patients with hypoparathyroidism.[1,22,23] The clinical significance of these findings and the association with parkinsonism is uncertain, so cerebral imaging is not routinely recommended.[1] Patients with nonsurgical hypoparathyroidism may also be at a two- to fourfold increased risk of cataracts.[10]

Management

The primary goals of management include maintaining serum calcium concentration in the low-normal range, preventing symptomatic or life-threatening hypocalcaemia, avoiding hypercalcaemia, hypercalciuria and hyperphosphataemia, and minimising the risk of disease- and treatment-related complications.[6]

Conventional treatment

The current standard of care for hypoparathyroidism is conventional treatment through supplementation with calcium (1 to 3 g/day in divided doses) with or without calcitriol (0.25 to 2.0 mcg/day, usually twice daily if total daily dose >0.5 mcg/day). The goal is to maintain serum calcium levels in the low-normal range using the lowest effective doses. In the absence of robust data to determine specific regimens, dosing is largely guided by clinician judgement, informed by serial serum and urinary calcium measurements, treatment tolerability and individual patient requirements.

Calcium carbonate is the most common and cost-effective over-the-counter formulation of elemental calcium, although it requires gastric acid for absorption and should be taken with food.[1] For patients taking proton pump inhibitors, calcium citrate may be more readily absorbed.[6] Calcitriol, due to its short half-life of three to six hours, is typically administered twice daily. Thiazide diuretics increase renal calcium reabsorption and can be used as an adjunct to reduce hypercalciuria.[6]

Dietary measures include a liberal intake of calcium, with restriction of phosphate and sodium to minimise hyperphosphataemia and hypercalciuria, respectively. Magnesium supplementation to maintain normomagnesaemia may also be used as tolerated.[6]

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Although conventional therapy helps maintain calcium levels, it does not address the underlying pathogenesis of PTH deficiency.[1] Calcium and calcitriol supplementation may also exacerbate the disease and its complications by further suppressing PTH secretion, increasing urinary calcium excretion and increasing renal phosphate reabsorption. Thus, conventional treatment may be limited by these adverse effects and a high pill burden, and has not been shown to improve quality of life.[24]

Monitoring

The required monitoring frequency for hypoparathyroidism depends on disease stability and clinician discretion. In stable patients, electrolytes and renal function may be monitored every six to 12 months.[6] However, unstable patients or those with recent dosage adjustments may need much closer monitoring until calcium levels stabilise. A 24-hour urinary calcium collection may be performed annually.[6] Renal tract imaging should be considered every five years or earlier in symptomatic patients or those with hypercalciuria to detect development of nephrocalcinosis and urinary calculi.[6]

PTH analogues

PTH replacement therapy is the optimal physiological treatment for hypoparathyroidism; however, consistent patient access has been limited by the short half-life of earlier formulations, the modest impact on clinically relevant outcomes and the prohibitive costs.

Teriparatide and recombinant PTH

Teriparatide (PTH 1-34), a once daily subcutaneous injection, is approved as an osteoanabolic agent for osteoporosis. Although, due to its short half-life of about one hour, it is poorly suited for hormone replacement and is therefore not indicated for chronic hypoparathyroidism.[25] Recombinant PTH (PTH 1-84) has a longer half-life of three hours, enabling once daily dosing. In a randomised controlled trial, PTH 1-84 was demonstrated to normalise calcium levels while achieving a 50% reduction in reliance on conventional treatment. Notably, it did not reduce urinary calcium excretion or show any significant improvement in quality of life parameters compared with placebo.[25,26] PTH 1-84 was approved by the FDA in 2015 as an adjunct to conventional therapy in hypoparathyroidism but was recalled in 2019 due to device-related safety concerns and is no longer available.[19,20]

Palopegteriparatide

Palopegteriparatide is a longer lasting form of PTH 1-34 with a half-life of 60 hours. The active PTH peptide is linked to a polyethylene glycol moiety that protects PTH from enzymatic degradation, reception activation and drug clearance and allows for sustained circulating PTH over 24 hours with daily subcutaneous dosing.[27] Palopegteriparatide is administered as an adjustable daily subcutaneous dose (prefilled pen) at the minimum dose required to maintain normocalcaemia. It is a specialised treatment for hypoparathyroidism, so commencement and monitoring should be supervised by a physician experienced in the management of patients with hypoparathyroidism.

2. Special considerations: managing hypoparathyroidism in pregnancy

- Physiological changes in pregnancy can increase or decrease requirements for calcium and calcitriol supplementation. Treatment should be individualised, with patient education and close monitoring
- Maternal hypocalcaemia is associated with an increased risk of miscarriage, preterm labour, fetal hypocalcaemia and fetal secondary hyperparathyroidism
- Maternal hypocalcaemia can cause fetal hypocalcaemia and seizures
- Pregnant women require frequent monitoring of serum calcium (every 3–4 weeks) and referral for multidisciplinary care
- Treatment targets include maintaining serum calcium in the low-normal range and ensuring normal magnesium, phosphate and vitamin D levels
- Thiazides and PTH replacement therapy should be avoided during pregnancy

Abbreviation: PTH = parathyroid hormone.

The efficacy and safety of palopegteriparatide was demonstrated in the PaTHway Trial, a 26-week international double-blind, randomised, placebo-controlled trial comparing palopegteriparatide with placebo in adults with stable chronic hypoparathyroidism on conventional therapy.[28] The study included 82 participants (85% post-surgical) who were randomised 3:1 to palopegteriparatide versus placebo. At the end of the 26-week placebo-controlled phase, 79% of patients receiving palopegteriparatide, compared with 5% in the placebo group, achieved the primary composite endpoint of normalisation of serum calcium and independence from conventional therapy.[28] Furthermore, participants receiving palopegteriparatide reported clinically meaningful improvements in quality of life, as well as physical and cognitive function.[22] Additional secondary safety endpoints included a reduction in urinary calcium excretion (by about 50%) and a 10 mL/min improvement in renal function.[29]

Palopegteriparatide was well tolerated; however, certain adverse events occurred more frequently than with placebo, including injection site reactions (26%), transient hypercalcaemia (14%, all in the first three months), nausea (9%) and headache (8%). No new safety signals have arisen in preliminary longer-term safety data with up to two to three years of use.[30,31]

Thus, once daily palopegteriparatide has been shown to maintain normal calcium concentrations without requiring conventional therapy. Unlike conventional treatment, it has demonstrated improvements in quality of life and reduction in urinary calcium excretion. Longer-term safety data are awaited. Given cost constraints, the role of palopegteriparatide in the management of chronic hypoparathyroidism in Australia remains uncertain, including whether it will receive approval for PBS subsidised access.

Special considerations

Box 2 outlines special considerations for managing hypoparathyroidism during pregnancy and Box 3 outlines considerations for autosomal dominant hypocalcaemia.

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3. Special considerations: autosomal dominant hypocalcaemia

- ADH type 1 and 2 are caused by activating mutations in *CASR* and *GNA11* signalling, respectively
- Patients with ADH have oversensitive CaSRs, requiring lower serum calcium levels to stimulate PTH release (i.e. a shifted setpoint)
- ADH causes inappropriate suppression of PTH, leading to hypocalcaemia
- Key features of ADH include hypocalcaemia, low-normal PTH, hypomagnesaemia and increased fractional urinary excretion of calcium
- Treatment with vitamin D with or without calcitriol may exacerbate hypercalciuria, nephrolithiasis, nephrocalcinosis and renal impairment
- Consider *CASR* genetic testing in patients with early-onset (<40 years of age) nonsurgical hypoparathyroidism, a positive family history of nonsurgical hypoparathyroidism or clinical features suggestive of a hereditary cause

Abbreviations: ADH = autosomal dominant hypocalcaemia; CaSR = calcium-sensing receptor; PTH = parathyroid hormone.

Conclusion

Hypoparathyroidism is characterised by insufficient circulating PTH to maintain normal serum calcium. Transient hypoparathyroidism is relatively common following anterior neck surgery; however, permanent hypoparathyroidism (>12 months) is rare. Chronic hypoparathyroidism is associated with multiple complications, including renal, cardiovascular, skeletal and neuropsychiatric effects, leading to considerable disease burden and impaired quality of life.

Although calcium and calcitriol supplementation is the standard of care for hypoparathyroidism and maintains serum calcium, it carries a high pill burden, can aggravate disease-related complications and does not target the underlying disease pathogenesis. Palopegteriparatide, a long-acting PTH analogue, represents a new PTH replacement therapy that is well tolerated, normalises serum calcium, reduces the need for conventional therapy and enhances quality of life. Palopegteriparatide is now TGA approved for chronic hypoparathyroidism, with PBS subsidisation currently under review to support patient access. **ET**

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A list of references is included in the online version of this article (www.endocrinologytoday.com.au).

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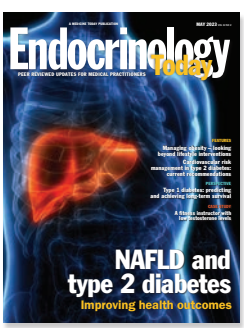

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