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Mini-Review

Pediatric primary hyperparathyroidism; historical milestones, evolving diagnosis, and future directions

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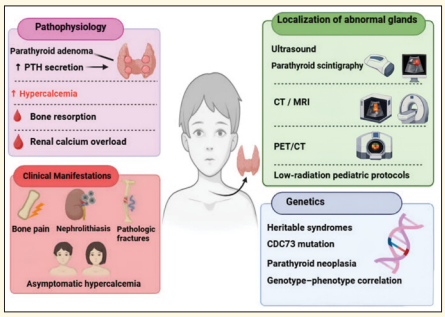
Abstract

Primary hyperparathyroidism (PHPT) in children, once regarded as exceedingly rare, is increasingly recognized following the introduction of sensitive parathyroid hormone (PTH) assays since the mid-1980s. This uncommon endocrine disorder, defined by autonomous PTH secretion and hypercalcemia, may manifest with bone pain, nephrolithiasis, or pathologic fractures, though asymptomatic hypercalcemia is also observed. Early cases accentuated on the importance of routine calcium screening to prevent under-diagnosis in pediatric populations. Advances in molecular genetics have elucidated heritable syndromes and mutations, such as CDC73, contributing to parathyroid neoplasia and hyperplasia, thereby refining risk stratification and management. Imaging plays a pivotal role in localizing abnormal glands, with ultrasound and parathyroid scintigraphy as first-line modalities; since, multiphase CT and MRI serve as adjuncts, though pediatric protocols must minimize radiation exposure. Emerging research explores normocalcemic PHPT variants and novel imaging techniques like PET/CT to enhance preoperative localization. Whilst understanding of the genetic architecture of PHPT evolves, future pediatric-focused studies aim to integrate molecular diagnostics with refined imaging strategies to optimize early detection, surgical planning, and long-term outcomes in affected children. Continued emphasis on genotype–phenotype correlations and low-radiation diagnostic protocols will be indispensable to improving care for these patients.



Keywords: Primary hyperparathyroidism, Children, Hypercalcemia, Bone pain, Urolithiasis, Nephrolithiasis, Parathyroidectomy

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Introduction

Pediatric primary hyperparathyroidism (PHPT) is a rare endocrine condition in childhood, a significant contrast to its prevalence in adults (1). The diagnosis of this disease in children was largely unknown until the mid-1980s, when the development of newer, more specific parathyroid hormone (PTH) assays allowed for a more confident diagnosis of this disorder during childhood (2). These advancements have been particularly valuable in identifying PHPT in pediatric patients. This disease is characterized by the overproduction of PTH, which leads to disrupted calcium metabolism and hypercalcemia. This hormonal imbalance can impact multiple organ systems, with the most significant effects observed in the skeleton and kidneys (3). Historically, PHPT was recognized as a symptomatic disease, presenting with severe manifestations affecting skeletal, renal, and neuromuscular systems (4). However, the routine screening of serum calcium levels has led to a shift in the clinical profile of PHPT towards milder, often asymptomatic presentations, especially in developed countries (5). This evolving highlights the importance of assessing serum calcium concentration in all uncertain disease states in children, as PHPT is likely underdiagnosed in this age group (6). The clinical features of pediatric PHPT can be diverse and often include bone pain, urolithiasis, or nephrolithiasis, alongside nonspecific symptoms like fatigue and weakness (7,8). Asymptomatic hypercalcemia may also be detected incidentally. In children with PHPT, bone pain is a frequent complaint in pediatric hyperparathyroidism, since orthopedic surgeons may be among the first healthcare providers to encounter such cases, even though presentation with a fracture is exceedingly rare (7,9). This narrative review considers the historical milestones that defined our

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Implication for health policy/practice/research/medical education

Pediatric primary hyperparathyroidism (PHPT) remains a rare endocrine disorder, historically recognized through symptomatic hypercalcemia and end-organ damage rather than asymptomatic biochemical detection common in adults. Early milestones included the development of parathyroid hormone (PTH) assays and recognition of its strong association with hereditary syndromes, particularly multiple endocrine neoplasia types 1 and 2A, and hyperparathyroidism-jaw tumor syndrome, where up to one-third of pediatric cases harbor germline mutations. Diagnosis has evolved from reliance on calcium measurements alone to comprehensive biochemical profiling, advanced neck imaging, and mandatory genetic testing at presentation to guide management and familial screening. Unlike adults, children typically present with overt symptoms including nephrolithiasis, bone demineralization, and neuropsychiatric manifestations, necessitating prompt parathyroidectomy.

comprehension of this condition, examines the evolution of diagnostic paradigms that have shifted from crude clinical observation to sophisticated molecular analysis, and explores emerging frontiers that promise more precise, less invasive, and potentially curative strategies for affected children.

Search strategy

For this narrative review, we conducted a literature search across multiple databases, including PubMed, Google Scholar, the Directory of Open Access Journals (DOAJ), Web of Science, EBSCO, Scopus, and Embase, using a variety of relevant keywords such as "pediatric primary hyperparathyroidism", "primary hyperparathyroidism", "parathyroid hormone", "hypercalcemia", "calcium metabolism", "pediatric endocrinology", "bone pain" and "nephrolithiasis".

A fresh look at the pediatric PHPT

As pediatric endocrinology and surgery developed as subspecialties, larger retrospective series helped define the clinical profile of PHPT in children more systematically, revealing important distinctions from the adult form of the disease (1). In contrast to adults, where asymptomatic hypercalcemia detected on routine biochemistry predominates most children present with overt symptoms and established end-organ damage at the time of diagnosis, including nephrolithiasis, nephrocalcinosis, bone pain, fractures, growth disturbances, and nonspecific complaints such as fatigue or abdominal pain (10). Cohort data spanning several decades indicate that roughly two-thirds of pediatric patients have solitary parathyroid adenomas, while a substantial minority display multi-gland hyperplasia or lesions associated with multiple endocrine neoplasia type 1 or other familial syndromes, making the etiologic spectrum more strongly influenced by genetic predisposition than in the typical adult population (11). The rarity of the condition and the predominance of symptomatic presentations mean that the diagnosis of this disease for many children remains protracted, with non-specific musculoskeletal or renal complaints often investigated in other directions before hypercalcemia and inappropriately elevated PTH levels are finally identified (12). This delay is clinically significant since prolonged exposure to hypercalcemia in childhood can produce lasting effects on bone mineral growth, renal function, and quality of life, emphasizing on the importance of earlier recognition and systematic evaluation of serum calcium in children with suggestive symptoms (10).

Biochemically, PHPT in pediatric patients is defined by elevated or inappropriately normal PTH levels in the setting of hypercalcemia, after secondary causes such as vitamin D deficiency and chronic kidney disease (3). Elevated serum calcium and unsuppressed PTH together remain the hallmark of diagnosis in children, and classic series have reported that this combination is diagnostic in virtually all affected patients when measured appropriately, affirming its role as the fundamental starting point for evaluation (11). Alongside these core markers, many children display associated biochemical abnormalities such as elevated alkaline phosphatase reflecting high bone turnover, hypercalciuria associated with nephrolithiasis or nephrocalcinosis, and variable phosphate levels, each of which can provide additional clues to disease severity and chronicity (13). The historical evolution of diagnostic practice has been marked by a shift from reliance on advanced bone disease and overt radiographic findings to a modern paradigm in which routine biochemical panels, targeted testing in high-risk groups, and greater awareness among pediatricians allow earlier identification of mild or atypical cases (13). Nonetheless, in contemporary cohorts, a substantial proportion of children continue to be diagnosed only after experiencing significant complications, underlining the need to integrate serum calcium measurement into the evaluation of persistent bone pain, recurrent renal stones, unexplained fractures, or neuromuscular symptoms in the pediatric setting (13).

Meanwhile, imaging strategies for localization of parathyroid lesions in children have evolved considerably and now largely parallel approaches in adults, although age-related anatomical and radiation-exposure considerations shape their application (14). High-resolution neck ultrasonography is frequently the first-line modality, while it is non-invasive, widely available, and free of ionizing radiation, making it particularly attractive in young patients (15). In fact, in experienced hands, it can identify enlarged parathyroid glands or adenomas in a majority of cases, while also assessing the thyroid gland for concomitant pathology (16). Radionuclide imaging with sestamibi scintigraphy, often combined with single-photon emission computed tomography, has long been a mainstay for preoperative localization, whilst recent series in children confirm its value, particularly when used in conjunction with ultrasound to guide minimally invasive or focused parathyroidectomy (17).

Cross-sectional imaging with computed tomography or magnetic resonance, including four-dimensional CT in selected settings, provides complementary information in cases of ectopic glands, re-operative surgery, or discordant findings, however must be weighed against concerns about radiation exposure (18). Overall, imaging has progressively shifted the surgical approach from traditional bilateral neck exploration towards more targeted operations where a single adenoma is confidently localized, although surgeons must remain prepared to convert to a comprehensive exploration when imaging is negative or when familial or multigland disease is suspected (19).

Focus on genetic evaluation

Germline mutations in genes such as MEN1, CDC73, and CASR underlie a spectrum of familial hyperparathyroid syndromes, including multiple endocrine neoplasia type 1, hyperparathyroidism-jaw tumor syndrome, familial isolated hyperparathyroidism, and neonatal severe hyperparathyroidism, since many of which can manifest in childhood or adolescence (20,21). In pediatric cohorts, the prevalence of identifiable germline mutations is substantially higher than in unselected adult populations (22). Hence, contemporary series advocate routine or at least liberal genetic testing for children with PHPT, particularly those with multigland disease, recurrent or persistent hypercalcemia, syndromic features, or a positive family history (23).

Management of children PHPT

Surgical management remains the definitive treatment for most children with PHPT, and long-term outcome data consistently demonstrate high cure rates with acceptable morbidity when procedures are performed in experienced centers (24). Retrospective series over several decades show that parathyroidectomy normalizes serum calcium in the vast majority of pediatric patients, with durable remission documented on follow-up extending into adolescence and adulthood, reflecting both the biological effectiveness of removing the source of hormone excess and improvements in perioperative care (25,26). The choice between focused or minimally invasive parathyroidectomy and traditional bilateral neck exploration is influenced by the pretest probability of solitary adenoma versus multigland disease, the results of localization imaging, and the presence of hereditary syndromes, with many centers favoring a tailored approach that balances operative efficiency against the risk of missed hyperfunctioning glands (27). Intraoperative PTH monitoring, when available, adds a dynamic biochemical endpoint to the surgeon's visual assessment by confirming adequate removal of hyperfunctioning tissue, which is especially helpful in the setting of inconclusive imaging or suspected multigland involvement (28). Postoperative complications such as transient hypocalcemia and hungry bone syndrome are relatively common in children with severe preoperative bone disease but can be anticipated and managed with intensive calcium and active vitamin D supplementation, while permanent hypoparathyroidism and recurrent laryngeal nerve injury remain infrequent in published pediatric cohorts (29, 30). Medical management plays a more limited but still important role, particularly in bridging children to surgery, managing high-risk neonates, or addressing residual or recurrent disease in complex familial syndromes (25). Hydration and loop diuretics can temporarily reduce serum calcium in acutely ill patients, while bisphosphonates may help stabilize the skeleton in cases of severe osteitis fibrosa cystica or multiple fractures prior to definitive parathyroidectomy (31). Calcimimetic agents such as cinacalcet, which allosterically activate the calcium-sensing receptor, have expanded the therapeutic repertoire in selected pediatric cases, including some with inoperable disease, residual hypercalcemia after surgery, or certain genetic disorders, though data remain more robust in adults and their use in children generally requires specialist oversight and careful monitoring (32). In neonatal severe hyperparathyroidism, aggressive medical therapy is sometimes attempted as an initial measure but often proves insufficient, and total or near-total parathyroidectomy remains the most reliable means of achieving long-term control, highlighting the ongoing primacy of surgery even in this extreme form of the disease (33). Looking ahead, advances in pharmacologic targeting of the calcium-sensing receptor and downstream signaling pathways may offer more durable non-surgical options for selected pediatric patients, though such approaches will need to be evaluated carefully for effects on growth plates, bone accrual, and neurodevelopment (1).

PHPT in older children

Primary hyperparathyroidism in older children tends to resemble the adult form of the disease. Sporadic cases are frequently caused by an adenoma, while familial forms, which can occur independently or as part of a polyendocrine syndrome, are often due to hyperplasia (34). Treatment for these cases typically involves parathyroidectomy, the extent of which is determined by the familial context, imaging findings, and frozen section histology during exploratory cervicotomy (25). Regardless of the child's age, family investigation is crucial to detect PHPT within the context of a hereditary disorder (35). In recent years, advancements in blood calcium concentration screening, diagnostic imaging for localization, and intact PTH checks have improved diagnosis and treatment outcomes. These improvements have made small incision parathyroidectomy, often guided by intraoperative positioning technology, as the primary surgical procedure (36).

Neonatal severe PHPT

Neonatal severe primary hyperparathyroidism (NSHPT)

represents a particularly rare and life-threatening autosomal recessive genetic disorder, resulting from inactivating mutations in calcium-sensing receptors. These cases often present rapidly with life-threatening symptoms and involve hyperplasia of the chief cells of the parathyroid glands (37). Treatment for NSHPT is always surgical and requires rapid intervention, usually a total parathyroidectomy with auto-transplantation (38). One reported case involved a three-day-old neonate with poor feeding, failure to thrive, and lethargy, who was successfully treated with total parathyroidectomy after laboratory results showed uncontrolled hypercalcemia with hyperparathyroidism (39).

Follow-up of children treated for PHPT

Long-term follow-up of children treated for PHPT is crucial to assess for recurrence, monitor bone and renal recovery, and address psychosocial consequences of a chronic endocrine condition diagnosed in formative years (40). Studies with more than a decade of follow-up demonstrate sustained normocalcemia in the vast majority of surgically treated patients, accompanied by progressive improvement in bone pain, radiographic healing of lytic lesions, and stabilization or partial reversal of nephrocalcinosis and nephrolithiasis, although residual structural damage may persist in some cases (6). Careful surveillance of bone mineral density, growth velocity, and pubertal progression is important after cure, given the possibility that years of preoperative hyperparathyroidism may have interfered with achieving optimal peak bone mass, which in turn carries implications for osteoporosis risk later in life (41). Similarly, serial assessment of renal function and urinary calcium excretion helps identify patients at risk of chronic kidney disease or recurrent stones, who may benefit from dietary counseling, hydration strategies, and, in some cases, pharmacologic interventions (42-44). Since many familial forms of the disease carry broader tumor risks, collaboration with endocrinologists, surgeons, geneticists, nephrologists, and when appropriate, oncologists ensures that surveillance protocols address the full spectrum of potential complications rather than focusing on the parathyroid glands in isolation (45, 46).

Conclusion

Pediatric PHPT remains a rare but clinically significant endocrine disorder distinct from its adult counterpart in etiology, presentation, and management. Historically, its recognition lagged behind adult PHPT, with early 20th-century descriptions limited to severe, symptomatic cases often linked to genetic syndromes like MEN syndromes. The identification of *MEN1*, *CDC73*, and *CASR* mutations revolutionized understanding of its hereditary basis, revealing that most of pediatric cases harbor germline mutations. Diagnostic evolution has shifted from reliance on overt symptoms toward biochemical screening, though challenges persist in interpreting age-adjusted calcium and PTH norms in children. Emerging technologies including 4D-CT, selective venous sampling, and molecular imaging have refined preoperative localization, while minimally invasive parathyroidectomy now offers cure rates in most of the cases with reduced morbidity. In the meantime, establishing pediatric-specific diagnostic criteria, detecting long-term skeletal and neurocognitive outcomes after cure, and developing targeted medical therapies for inoperable cases are necessary. Accordingly, gene-editing approaches and calcium-sensing receptor modulators represent promising frontiers. Notably, universal genetic testing at diagnosis is becoming standard to guide surveillance for associated tumors and enable family screening. As awareness grows among pediatric endocrinologists and surgeons, earlier detection of milder phenotypes is anticipated. Multicenter registries will be essential to accumulate sufficient data given the disease's rarity. Finally, optimizing outcomes in pediatric PHPT demands a precision medicine approach by integrating genetic profiling, advanced imaging, and tailored surgical strategies, while recognizing that children are not merely small adults but a unique population requiring specialized, lifelong multidisciplinary care.

Authors' contribution

Conceptualization: Elham Farasat, Mohsen Akhavan Sepahi.
Data curation: Mohsen Akhavan Sepahi
Investigation: Fatemeh Fallah Tafti
Resources: Elham Farasat.
Supervision: Fatemeh Fallah Tafti.
Validation: Fatemeh Fallah Tafti.
Visualization: Mohsen Akhavan Sepahi.
Writing–original draft: Mohsen Akhavan Sepahi.
Writing–review and editing: All authors.

Conflicts of interest

The authors declare that they have no competing interests.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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