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Review

Suppression and recovery dynamics of infant parathyroid function; clinical outcomes following maternal primary hyperparathyroidism

Barno Badridinova[1*], Dilrabo Nazarova[2], Dilafruz Turaeva[3], Mashkhura Safarova[4], Dildora Matrasulova[5], Dilobar Sevinchova[1], Nigora Khodjayeva[6], Ekaterina Anvarova[7], Azizjon Khaydarov[8], Makhliyo Abdusalomova[9]

Abstract

Maternal primary hyperparathyroidism (PHPT), though rare during pregnancy, poses significant and disproportionate risks to the fetus and newborn due to transplacental passage of maternal calcium and parathyroid hormone (PTH). This condition disrupts the fetal mineral regulatory milieu, establishing a critical pathophysiological sequence. Sustained maternal hypercalcemia directly suppresses the development and function of the fetal parathyroid glands in utero, inducing a state of functional dependence on the maternal hypercalcemic environment for calcium homeostasis. Consequently, at delivery, the abrupt termination of both the maternal hypercalcemic drive and placental calcium transfer precipitates a high-risk period for the neonate. The infant, born with profoundly suppressed parathyroid reserve, faces a substantial challenge in mounting an adequate PTH secretory response to the sudden drop in calcium supply. This pathophysiological lag universally triggers neonatal hypocalcemia, the severity and duration of which are intrinsically linked to the depth and chronicity of in utero parathyroid suppression, as well as the individual neonate's capacity for parathyroid functional recovery. Clinical manifestations can range from asymptomatic biochemical hypocalcemia to severe, life-threatening complications including seizures, tetany, apnea, and cardiomyopathy. Understanding this mechanistic pathway, from maternal hypercalcemia to fetal gland suppression and the predictable postnatal hypocalcemic crisis is paramount. It underscores the necessity for vigilant prenatal diagnosis of maternal PHPT, meticulous intrapartum planning, and intensive, protocol-driven neonatal monitoring and calcium/active vitamin D replacement strategies to prevent significant neonatal morbidity. Early recognition of this iatrogenic dependence is crucial for optimizing outcomes in affected offspring.

Keywords: Maternal, Infant, Neonatal, Parathyroid, Calcium homeostasis, Hypocalcemia, hypercalcemia, Hyperparathyroidism, Parathyroid hormone, Neonatal outcomes, Maternal

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Introduction

Pregnancy induces profound changes in mineral metabolism. There is a physiological increase in glomerular filtration rate, leading to increased renal calcium clearance (1). To compensate, intestinal calcium absorption significantly enhances under the influence of elevated calcitriol levels, driven by placental and renal 1-alpha-hydroxylase activity (2). Serum albumin decreases, causing a fall in total serum calcium, while ionized calcium is typically maintained within or slightly below the non-pregnant reference range (3). Parathyroid hormone (PTH) levels normally decrease during pregnancy, particularly in the second and third trimesters, due to the calcium-sparing effects of these adaptations (4). Diagnosis of primary hyperparathyroidism (PHPT) in this context is difficult because the biochemical hallmarks, like hypercalcemia and inappropriately normal or elevated parathormone, can be masked (5). Mild hypercalcemia might be dismissed, making interpretation tricky. Furthermore, routine calcium screening is not standard prenatal care, asymptomatic cases may go undetected until complications arise (6). The maternal risks of untreated PHPT in pregnancy are substantial and escalate with the degree of hypercalcemia (7). Severe hypercalcemia

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[1]Bukhara State Medical Institute, Bukhara, Uzbekistan. [2]Department of Obstetrics and Gynecology, Tashkent State Medical University, Tashkent, Uzbekistan. [3]Department of Pediatrics, Faculty of Medicine, Samarkand State Medical University, Samarkand, Uzbekistan. [4]Department of Surgical Dentistry, Bukhara State Medical Institute, Bukhara, Uzbekistan. [5]Urgench State Medical Institute, Urgench, Uzbekistan. [6]Department of Faculty Pediatrics, Tashkent State Medical University, Tashkent, Republic of Uzbekistan. [7]Ultrasonologist, Sh.Alimov Republican Specialized Medical Center of Phthisiology and Pulmonology, Tashkent State Medical University, Tashkent, Uzbekistan. [8]Department of Traumatology and Orthopedics, Fergana Medical Institute of Public Health, Fergana, Republic of Uzbekistan. [9]Department of Phthisiology and Pulmonology, Andijan State Medical Institute, Republic of Uzbekistan

***Corresponding author:** Barno Badridinova, Email: badridinova.barnoxon@bsmi.uz

Implication for health policy/practice/research/medical education

Primary hyperparathyroidism (PHPT) during pregnancy represents a significant clinical challenge due to its extreme rarity, profound diagnostic complexities, and potentially severe, life-threatening consequences for both mother and fetus. Its infrequency means clinicians often lack experience, and symptoms like fatigue, nausea, bone pain, or polyuria are readily misattributed to normal physiological adaptations of pregnancy, leading to dangerous delays in recognition. This diagnostic ambiguity is compounded by pregnancy-induced alterations in calcium metabolism and albumin levels, which can mask typical biochemical abnormalities. Undiagnosed or poorly managed disease significantly elevates maternal risks, including nephrolithiasis, pancreatitis, severe hypertension, and bone demineralization. Notably, the fetus faces substantial dangers such as intrauterine growth restriction, preterm delivery, neonatal hypocalcemia with seizures, and even intrauterine demise. Consequently, maintaining a high index of suspicion, utilizing careful biochemical interpretation adjusted for gestation, and prompt multidisciplinary management involving endocrinology and maternal-fetal medicine are paramount to mitigate these dual risks and optimize outcomes.

can lead to debilitating symptoms like profound fatigue, nausea, vomiting, constipation, polyuria, polydipsia, and muscle weakness (8). More critically, it significantly increases the risk of nephrolithiasis, which can cause severe pain, urinary tract infections, and potential renal impairment (9). Meanwhile, pancreatitis is a rare but serious complication of hypercalcemia (10). Hypertensive disorders of pregnancy, including preeclampsia, appear to be more common in women with PHPT, though causality is complex (11). Cardiac arrhythmias are a potential consequence of severe hypercalcemia (12). The most catastrophic maternal risk is the development of hypercalcemic crisis, a life-threatening emergency characterized by extreme hypercalcemia, severe dehydration, confusion, coma, and multi-organ failure, requiring immediate intensive intervention (13). However, the fetal and neonatal risks are often even more devastating and emphasize on the critical need for timely diagnosis and management (13). In this narrative review, we sought to consider suppression and recovery dynamics of infant parathyroid function; clinical outcomes following maternal PHPT.

Search strategy

A comprehensive literature search was conducted up to date to January 2026 to identify relevant studies for this narrative review. Multiple electronic databases were explored, including PubMed, Google Scholar, the Directory of Open Access Journals (DOAJ), Web of Science, EBSCO, Scopus, and Embase. The search strategy employed a combination of Medical Subject Headings (MeSH) terms and free-text keywords related to maternal and neonatal calcium-regulating disorders. The primary keywords comprised "maternal," "infant," "neonatal," "parathyroid," "calcium homeostasis," "hypocalcemia," "hypercalcemia," "hyperparathyroidism," "parathyroid hormone," and "neonatal outcomes," along with additional modifiers such as "endocrine" and "maternal." Boolean operators (AND, OR) were applied to optimize the search sensitivity and specificity. No temporal restrictions were applied, and the references of retrieved articles were manually screened to ensure inclusion of all pertinent studies.

Maternal primary hyperparathyroidism

Maternal PHPT arises from autonomous overproduction of PTH, most commonly due to a solitary parathyroid adenoma, leading to hypercalcemia (14). In pregnancy, physiological changes complicate the picture. The normal pregnancy-induced increase in glomerular filtration rate enhances calcium clearance, often partially counteracting the hypercalcemia (15). Additionally, the placenta produces PTH-related protein (PTHrP), which shares receptor homology with PTH and can contribute to mineral regulation, though its role in modulating the effects of maternal PHPT on the fetus is complex and not fully protective against suppression (16). Importantly, maternal calcium, being ionized and unbound to protein, readily crosses the placenta by active transport mechanisms (17). The fetus, therefore, is exposed to chronically elevated calcium levels throughout gestation (17). This persistent hypercalcemia acts directly on the developing fetal parathyroid glands (18). Fetal parathyroid development begins around the 5^{th}-6^{th} week of gestation, with functional PTH secretion detectable by 10-12 weeks (19). However, these glands are exquisitely sensitive to ambient calcium concentrations (20). Sustained hypercalcemia in- utero leads to down-regulation of the calcium-sensing receptor expression on parathyroid chief cells and suppresses PTH synthesis and secretion (16). Histologically, the parathyroid glands may appear smaller, with reduced cellularity and evidence of atrophy. This suppression is a physiological adaptation, since the fetus does not require high PTH output when bathed in high calcium (18). Consequently, the fetus often maintains normocalcemia or even mild hypercalcemia in -utero, despite the suppressed PTH levels, primarily due to the continuous influx of maternal calcium (21). The fetal skeleton, however, may be affected; maternal hypercalcemia can suppress fetal PTH needed for normal bone resorption and remodeling, potentially leading to under-mineralization or, paradoxically in severe cases, sub-periosteal bone resorption visible on prenatal ultrasound (22). However, the critical transition occurs at birth; since, following clamping of the umbilical cord, the exogenous source of high calcium is abruptly severed (23). The infant is now solely dependent on its own regulatory systems. However, the parathyroid glands, suppressed for months, are functionally immature and unable to respond adequately to the rapidly falling serum calcium levels (24). This condition results in neonatal hypocalcemia, as the most significant and immediate clinical consequence (24). The timing and severity of this hypocalcemia are

highly variable but typically manifest within the first 24 to 72 hours of life, though it can be delayed for some days in patients (25). Early onset, within first 48 hours is often more severe and symptomatic (25, 26). Symptomatic hypocalcemia presents with non-specific but potentially alarming signs like jitteriness, tremors, irritability, high-pitched cry, vomiting, poor feeding, lethargy, hypotonia, seizures, apnea, and in severe cases, cardiac arrhythmias or congestive heart failure due to prolonged QT interval (27). Asymptomatic hypocalcemia is also common but requires vigilant monitoring as symptoms can develop rapidly (28). The depth of in-utero suppression is the primary determinant of severity (26); infants born to mothers with poorly controlled, severe hypercalcemia throughout pregnancy—especially if undiagnosed or untreated until late gestation—are at highest risk (18). Prematurity further exacerbates the risk, as the parathyroid glands are inherently less mature (29). Conversely, infants whose mothers underwent successful parathyroidectomy before significant suppression occurred, or whose hypercalcemia was well-controlled medically, generally have milder or no neonatal hypocalcemia (30).

Management of neonatal hypocalcemia

Management of neonatal hypocalcemia in this setting is urgent and requires a complete approach involving neonatology, endocrinology, and often cardiology (24). The primary goal is to rapidly correct hypocalcemia to prevent seizures and cardiac complications, while carefully monitoring for rebound hypercalcemia or other electrolyte disturbances (24). Acute symptomatic hypocalcemia or severe asymptomatic hypocalcemia mandates intravenous calcium replacement (24). Calcium gluconate (10% solution) is preferred over calcium chloride due to less tissue necrosis risk if extravasation occurs (24). It must be administered slowly by a central line or a well-functioning peripheral line with careful cardiac monitoring (risk of bradycardia) and frequent ionized calcium checks to avoid hypercalcemia (31). Asymptomatic or mild hypocalcemia may be managed with enteral calcium supplementation and active vitamin D metabolites to enhance intestinal absorption (24). In fact, calcitriol is the preferred active metabolite, as it bypasses the need for renal 1-alpha hydroxylation, which can be immature in newborns (32). Magnesium levels must be checked and treated if low, as hypomagnesemia impairs PTH secretion and end-organ response (33). Phosphate binders are rarely needed acutely unless hyperphosphatemia is severe and contributing to hypocalcemia (34). The cornerstone of long-term management, however, is supporting the recovery of infant parathyroid function (24). In this regard, oral calcium and calcitriol supplementation should be continued, but doses are meticulously weaned based on frequent monitoring of ionized calcium, phosphorus, magnesium, creatinine, and urinary calcium excretion (to avoid nephrocalcinosis from overtreatment) (24). The key biomarker for recovery is the infant's PTH level. Serial measurements (initially weekly, then bi-weekly or monthly) showing a gradual rise in PTH, particularly in response to falling calcium levels, signal functional recovery (35). This recovery follows a characteristic pattern; since, parathormone levels begin to increase within days to weeks after birth, but normalization often lags behind the normalization of serum calcium (36). The stimulus for this recovery is the persistent hypocalcemia itself, acting as the natural trigger for parathyroid cell proliferation and PTH synthesis (24). The rate of recovery is influenced by the duration and severity of suppression, gestational age at birth, genetic factors influencing parathyroid reserve, and the adequacy of initial calcium replacement without causing hypercalcemia, which could theoretically prolong suppression (37).

Clinical outcomes

Clinical outcomes are generally favorable with prompt recognition and meticulous management; however, significant morbidity can occur if hypocalcemia is severe, prolonged, or undiagnosed (38). Neurological sequelae are the most concerning potential long-term outcome. Neonatal seizures due to hypocalcemia, if prolonged or recurrent, can lead to hypoxic-ischemic injury (39). More insidiously, chronic or recurrent hypocalcemia during critical periods of brain development has been linked, to subtle neurodevelopmental delays, including cognitive deficits, learning disabilities, and behavioral problems, though establishing direct causality is complex due to confounding factors like prematurity or genetic syndromes (40). Moreover, cardiac effects are usually acute and reversible with calcium correction, but severe, untreated hypocalcemia can rarely cause permanent myocardial damage (41). Previous authors demonstrated that, skeletal effects can persist beyond the neonatal period. In-utero suppression of PTH can lead to under-mineralization of the fetal skeleton, presenting as radiological evidence of osteopenia or even neonatal fractures (42). Rarely, severe in-utero hypercalcemia can cause perinatal asphyxia due to placental vascular calcification, intrauterine growth restriction, or preterm delivery, all of which carry their own independent risks for adverse outcomes (18). Importantly, transient hypoparathyroidism is the rule, not the exception. Permanent hypoparathyroidism is exceedingly rare following maternal PHPT and should prompt investigation for alternative diagnoses like DiGeorge syndrome or autoimmune polyglandular syndrome (43). However, subtle deficits in bone density or neurocognitive function may warrant periodic assessment (24).

Conclusion

In summary, the suppression and subsequent recovery of infant parathyroid function following exposure to maternal PHPT in utero represent a well-defined,

predictable physiological adaptation. This suppression, driven by chronic fetal hypercalcemia that suppresses the developing parathyroid glands, carries significant clinical consequences: unrecognized or inadequately managed neonatal hypocalcemia can lead to seizures, cardiac dysfunction, and respiratory distress, contributing substantially to avoidable neonatal morbidity. The severity and duration of in utero suppression directly correlate with the depth and persistence of postnatal hypocalcemia, dictating the intensity of required intervention. While parathyroid axis recovery typically unfolds over days to weeks, the timeline is highly individualized; protracted suppression necessitates vigilant monitoring and sustained therapeutic support, often involving intravenous or enteral calcium supplementation alongside active vitamin D metabolites to maintain normocalcemia and prevent complications. Timely maternal definitive management, typically parathyroidectomy during pregnancy, when indicated and feasible, significantly attenuates fetal exposure and subsequent neonatal risk. Irrespective of maternal intervention timing, smooth collaboration between obstetricians, endocrinologists, maternal-fetal medicine specialists, surgeons, and neonatologists is non-negotiable. This multi-approach ensures appropriate antenatal counseling, targeted fetal monitoring, immediate postnatal calcium surveillance protocols, and pre-arranged neonatal management strategies. Only through such coordinated vigilance and prompt, protocol-driven management of the neonate can the substantial but preventable morbidity associated with this condition be consistently avoided, safeguarding infant health during this critical transition period.

Authors' contribution

Conceptualization: Barno Badridinova and Dilrabo Nazarova.
Data curation: Abdusalomova Makhliyo, Nigora Khodjayeva, and Barno Badridinova.
Investigation: Dildora Matrasulova and Dilobar Sevinchova.
Supervision: All authors.
Validation: Dilafruz Turaeva and Mashkhura Safarova.
Visualization: Ekaterina Anvarova and Khaydarov.
Writing–original draft: All authors.
Writing–review and editing: All authors.

Ethical issues

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

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