



## *Primary Hyperparathyroidism and Pregnancy: A Case Report of a Favorable Outcome with Conservative Management*

**Dina Houjjaj<sup>1\*</sup>, Sara Mouhmouh<sup>2</sup>, Amal Benbella<sup>3</sup>, Bouchaib Allae Eddine<sup>3</sup>, Abdelhai Adibe Filali<sup>3</sup>, Mohammed Hassan Alami<sup>3</sup> and Rachid Beza<sup>3</sup>**

<sup>1</sup>Department of Obstetrics and Gynecology, Les Orangers Maternity Hospital, Mohammed V University, Rabat, Morocco

<sup>2</sup>Department of Obstetrics and Gynecology, Mohammed VI University, Agadir, Morocco

<sup>3</sup>Department of Obstetrics and Gynecology, Les Orangers Maternity Hospital, Mohammed V University, Rabat, Morocco

**Citation:** Dina Houjjaj, Sara Mouhmouh, Amal Benbella, Bouchaib Allae Eddine, Abdelhai Adibe Filali, et al. (2026) Primary Hyperparathyroidism and Pregnancy: A Case Report of a Favorable Outcome with Conservative Management. *J. of Inn Clin Trail Case Reports* 2(3), 1-8. WMJ/JCTC-141

### **Abstract**

**Introduction:** Primary hyperparathyroidism during pregnancy is a rare condition (incidence: 0.15%) yet potentially serious, associated with significant maternal, obstetrical, and neonatal complications. Optimal management remains controversial, with debate between conservative treatment and surgical intervention.

**Case Report:** We report the case of a 22-year-old patient, G2P1, with a 3-cm parathyroid adenoma diagnosed six months before pregnancy following the investigation of dysphagia. The patient had elevated PTH levels with normal serum calcium. Close clinical and biological monitoring throughout pregnancy was unremarkable. The patient was admitted at 39 weeks and 6 days of gestation for premature rupture of membranes. Vaginal delivery was uncomplicated, with the birth of a 3,850 g newborn with Apgar scores of 10/10.

**Discussion:** This case illustrates the potential for a favorable outcome with conservative management in asymptomatic forms of primary hyperparathyroidism during pregnancy. Comparative analysis of the literature reveals that second-trimester parathyroidectomy remains the gold-standard treatment in cases of symptomatic hypercalcemia (calcium > 110 mg/L), while a conservative approach can be considered in paucisymptomatic forms with normal or moderately elevated calcium levels. Neonatal follow-up is crucial, as neonatal hypocalcemia occurs in 50% of cases, typically between the 5th and 14th postpartum day.

**Conclusion:** Early diagnosis and multidisciplinary surveillance are essential to optimize maternal and neonatal prognosis. The therapeutic choice should be individualized according to the severity of hypercalcemia and gestational age.

**\*Corresponding author:** Dina Houjjaj, Department of Obstetrics and Gynecology, Les Orangers Maternity Hospital, Mohammed V University, Rabat, Morocco.

**Keywords:** Primary Hyperparathyroidism, Pregnancy, Parathyroid Adenoma, Hypercalcemia, Neonatal Complications, Conservative Management

## Introduction

Primary hyperparathyroidism (PHPT) is an endocrine disorder characterized by inappropriate hypersecretion of parathyroid hormone (PTH), most often secondary to a parathyroid adenoma (80–85% of cases), and more rarely to multiglandular hyperplasia (10–15%) or parathyroid carcinoma (< 1%) [1,2]. During pregnancy, this condition is exceptional, with an estimated incidence of 0.05 to 0.15% of gestations [3,4].

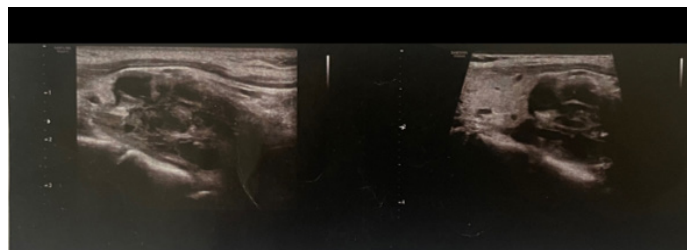
The physiological changes of pregnancy complicate the diagnosis and management of PHPT. The physiological increase in glomerular filtration rate and the placental production of PTH-related protein (PTHrP) alter maternal calcium homeostasis [5]. In addition, the symptoms of PHPT (nausea, vomiting, fatigue, constipation) may be confused with the physiological manifestations of pregnancy, thus delaying diagnosis [6].

Maternal complications include nephrolithiasis (24–36%), acute pancreatitis (7–13%), preeclampsia (25%), and cardiac arrhythmias [7,8]. Obstetrical complications include intrauterine growth restriction, threatened preterm labor, and spontaneous miscarriage. In the newborn, neonatal hypocalcemia is the most frequent complication (30–50% of cases) and may manifest as seizures, tetany, or even respiratory distress [9,10].

## Case Report

### Background and Discovery of the Condition

We report the case of a 22-year-old woman, gravida 2 para 1 (G2P1), with a history of one previous uncomplicated vaginal delivery. Six months prior to the current pregnancy, the patient consulted for progressive swallowing difficulties. Investigation revealed a 3-cm parathyroid adenoma on cervical imaging, associated with elevated plasma PTH, while serum calcium remained within normal limits.



**Figure 1:** Cervical Ultrasound of the Thyroid Region

**Legend:** Well-circumscribed hypoechoic mass measuring 30 mm, located in the right postero-inferior position of the thyroid bed, consistent with a parathyroid adenoma.

**Interpretation:** Cervical ultrasound shows a thyroid gland of normal morphology and echostructure. Exploration of the thyroid bed reveals a hypoechoic, well-circumscribed mass with regular contours, located in the right postero-inferior position relative to the right thyroid lobe, consistent with a parathyroid adenoma. Ultrasonographic measurements confirm the significant size of the lesion, estimated at approximately 30 mm along its long axis. The homogeneous hypoechoic appearance and the anatomical location are characteristic of a typical parathyroid adenoma.



**Figure 2:** Coronal Contrast-Enhanced Cervical Computed Tomography

**Legend:** Tissue mass measuring 30 × 21 mm in a right retro-thyroid inferior position, showing moderate and homogeneous contrast enhancement, with no invasion of adjacent structures. Appearance consistent with a hypervascularized parathyroid adenoma.

**Interpretation:** Contrast-enhanced cervical computed tomography confirms the presence of a tissue mass of intermediate density, measuring 30 × 21 mm, located in a right retro-thyroid inferior position. The lesion demonstrates moderate and homogeneous enhancement following injection, characteristic of a hypervascularized parathyroid adenoma. Anatomical relationships are well visualized, showing an extra-thyroid location with posterior displacement of the cervical neurovascular axis without signs of invasion of adjacent structures. The absence of intralesional calcifications and the regularity of the contours rule out malignancy. The other parathyroid glands are not visualized, which is a typical CT finding in the absence of pathological hypertrophy.

In view of this finding and the patient's desire for pregnancy, a multidisciplinary discussion involving an endocrinologist, a head-and-neck surgeon, and an obstetrician was organized. The decision was made to opt for close clinical and biological monitoring during pregnancy, given the asymptomatic nature of the hyperparathyroidism—apart from the initial mechanical dysphagia—and the normal serum calcium.

### Course of Pregnancy

The current pregnancy was dated at 39 weeks and 6 days of gestation based on the crown-rump length measured during the first-trimester ultrasound at 12 weeks of gestation. Prenatal surveillance included monthly consultations with systematic measurement of total and ionized calcium, as well as PTH levels. Biological parameters remained stable throughout pregnancy, with serum calcium ranging between 95 and 102 mg/L (normal range: 88–104 mg/L) and PTH between 85 and 110 pg/mL (normal range: 15–65 pg/mL).

Trimestral ultrasound monitoring did not reveal any fetal morphological abnormalities or intrauterine growth restriction. Repeated cervical examinations did not identify a palpable mass despite the known 3-cm adenoma.

### Delivery and Postpartum Course

At 39 weeks and 6 days of gestation, the patient was admitted on an emergency basis for premature rupture of membranes with clear amniotic fluid. Clinical examination on admission was unremarkable apart from the rupture of membranes. Cardiotocographic monitoring was reassuring.

Obstetrical ultrasound confirmed cephalic presentation, with biometry consistent with gestational age and an estimated fetal weight of 3,777 g. Labor induction was not required, as the patient spontaneously developed regular uterine activity.

Vaginal delivery ultimately proceeded without further complications. A male newborn weighing 3,850 g was born with an Apgar score of 10/10 at one and five minutes of life.

Maternal serum calcium measured in the immediate postpartum period was 98 mg/L, confirming the stability of calcium parameters. A close neonatal surveillance protocol was implemented with regular neonatal calcium measurements at day 1, 3, 5, 7, and 14, given the high risk of transient neonatal hypocalcemia.

The maternal postpartum course was uneventful. The patient attended an endocrinological consultation at 6 weeks postpartum for reassessment of the indication for parathyroidectomy, now feasible outside the obstetrical context.

### Discussion

#### Epidemiology and Diagnosis

Primary hyperparathyroidism during pregnancy is a rare but not exceptional clinical entity. The true incidence is probably underestimated due to the often asymptomatic nature of the disease and the absence of systematic calcium measurement during prenatal surveillance [11]. Retrospective cohort studies suggest an incidence ranging from 0.05 to 0.15% of pregnancies, with an upward trend in recent years, likely related to improved diagnostic techniques [3,4,12].

Diagnosis is often incidental, discovered during a biological workup performed for nonspecific symptoms or during the investigation of complications such as recurrent renal lithiasis [13]. In our observation, the diagnosis was established before pregnancy as part of the investigation of dysphagia, illustrating the

importance of a rigorous diagnostic approach in patients presenting with cervical symptoms.

Cervical ultrasound and computed tomography are the reference examinations for preoperative localization of parathyroid adenomas. Ultrasound enables a detailed analysis of the cervical region with a sensitivity of 70–80% for the detection of parathyroid adenomas [14]. Adenomas typically appear as hypoechoic, homogeneous, well-circumscribed formations located posterior or inferior to the thyroid gland. Contrast-enhanced cervical CT offers similar sensitivity (75–85%) and allows better evaluation of anatomical relationships with cervical neurovascular structures [15]. In our case, the concordance of ultrasonographic and CT findings enabled precise localization of the 3-cm parathyroid adenoma in a right retro-thyroid inferior position.

The clinical presentation of PHPT during pregnancy is polymorphic and nonspecific. Classic symptoms include nausea and vomiting (hyperemesis gravidarum), severe fatigue, excessive thirst (polydipsia), abdominal pain, constipation, and mood disorders that may progress to depression [16]. These manifestations are frequently attributed to the pregnancy itself, delaying diagnosis. Severe hypercalcemia ( $> 120$  mg/L) may present with neuropsychiatric disturbances (confusion, lethargy) and cardiac arrhythmias, and constitutes a therapeutic emergency [17].

### Maternal and Obstetrical Complications

The maternal complications of PHPT during pregnancy are multiple and potentially serious. Nephrolithiasis is the most frequent complication, reported in 24 to 36% of cases according to published series [7,18]. Acute pancreatitis, although rarer (7–13% of cases), is a feared complication that can endanger both maternal and fetal life [8]. Acute hypercalcemic crises, characterized by serum calcium  $> 140$  mg/L, are exceptional but represent an absolute endocrinological emergency [19].

From an obstetrical standpoint, PHPT is associated with an increased risk of preeclampsia (25% of cases), significantly higher than in the general population [20]. Intrauterine growth restriction, threatened preterm labor, and spontaneous miscarriage are also more frequent, with a risk of fetal loss estimated between 2 and 5% in untreated forms [21,22]. The cesarean

section rate is higher, reaching 30 to 40% in some series, due to maternal and fetal complications [23].

In our observation, the absence of significant maternal or obstetrical complications may be explained by several factors: the asymptomatic nature of the hyperparathyroidism, normal serum calcium despite elevated PTH, and the close surveillance implemented from the start of pregnancy. These elements highlight the importance of an individualized follow-up and a multidisciplinary approach.

### Neonatal Complications

Neonatal hypocalcemia is the most feared fetal complication of maternal PHPT, occurring in 30 to 50% of cases according to published series [9,10,24]. The pathophysiology is based on the suppression of fetal parathyroid gland activity by chronic maternal hypercalcemia. Maternal calcium freely crosses the placental barrier, maintaining elevated fetal calcium levels during intrauterine life and inhibiting fetal PTH secretion [25].

At birth, the abrupt interruption of this maternal calcium supply, combined with functional neonatal hypoparathyroidism, leads to often severe hypocalcemia. Clinical manifestations typically appear between the 5th and 14th day of life, the period during which residual calcium stores are depleted [26]. Clinical signs include neuromuscular hyperexcitability, tremors, hypertonia, seizures, lethargy, and—in severe forms—apnea and respiratory distress [27].

The prognosis of neonatal hypocalcemia is generally favorable with treatment. Management relies on calcium and vitamin D supplementation, adapted to the severity of hypocalcemia [28]. The average duration of treatment ranges from 3 to 5 months, corresponding to the time required for functional recovery of the neonatal parathyroid glands [29]. Cases of permanent neonatal hypoparathyroidism have been exceptionally reported, justifying prolonged endocrinological follow-up [30].

In our case, the close neonatal surveillance protocol implemented from birth allows early detection and optimal management of any potential hypocalcemia. This preventive approach is essential to minimize the risk of long-term neurological complications.

### Therapeutic Strategies: Comparative Analysis

The optimal management of PHPT during pregnancy remains controversial, opposing supporters of conservative treatment and those favoring surgical intervention. Comparative analysis of the literature allows recommendations to be drawn according to the severity of hypercalcemia and gestational age.

### Conservative Treatment

Conservative treatment relies on several pillars:

- Oral hyperhydration (2.5 to 3 liters per day) aimed at maintaining abundant diuresis and promoting renal calcium excretion [31].
- Moderate restriction of dietary calcium intake (< 1,000 mg/day) while avoiding deficiencies [32].
- Close clinical and biological monitoring with biweekly measurement of serum calcium and PTH during the first and third trimesters, and monthly during the second trimester [33].
- Pharmacological agents may be considered in selected situations:
- Cinacalcet, a calcimimetic agent that increases the sensitivity of the calcium-sensing receptor, has been used in 13 pregnant women according to a recent systematic review, with no reported fetal adverse effects [34]. However, its efficacy is limited in severe forms, and its use remains off-label during pregnancy.
- Calcitonin, which inhibits bone resorption, does not cross the placental barrier and therefore presents a favorable safety profile [35]. However, its efficacy remains modest and transient.

A conservative approach may be preferred in the following situations: asymptomatic PHPT, normal or moderately elevated serum calcium (< 110 mg/L), absence of maternal or obstetrical complications, and the possibility of rigorous close follow-up [36]. Our observation falls within this framework, justifying the choice of surveillance without surgical intervention.

### Surgical Treatment

Parathyroidectomy represents the definitive treatment for PHPT and is generally recommended in the second trimester of pregnancy (14–28 weeks of gestation), considered the optimal therapeutic window [37]. The first trimester carries a teratogenic risk related to anesthetic exposure during organogenesis, while the third trimester is associated with an increased risk of surgery-induced preterm delivery [38].

Indications for parathyroidectomy during pregnancy include:

- Symptomatic hypercalcemia with serum calcium > 110 mg/L [39]
- History of nephrolithiasis or nephrocalcinosis [40]
- Acute pancreatitis related to hypercalcemia [41]
- Progressive hypercalcemia despite optimal conservative treatment [42]
- Suspected parathyroid carcinoma [43]

Studies report a success rate of 95 to 98% for parathyroidectomy during pregnancy, with immediate normalization of serum calcium [44]. However, the risk of postoperative hypocalcemia is significantly higher than outside pregnancy, reaching 50 to 62% of cases in some series [45]. This complication is related to “hungry bone syndrome,” which is particularly marked during pregnancy due to increased calcium demands for the development of the fetal skeleton.

### Comparative Case Analysis

Comparative analysis of published cases illustrates the importance of timing and therapeutic strategy in maternal and fetal prognosis.

#### Case 1: Favorable Outcome with Second-Trimester Surgery

A 36-year-old patient, G2P1, with recurrent nephrolithiasis, was diagnosed with severe PHPT in the first trimester of pregnancy. The biological workup revealed major hypercalcemia at 111 mg/L (normal < 104 mg/L) and a markedly elevated PTH at 519.7 pg/mL (normal 15–65 pg/mL). Cervical ultrasound identified a large right inferior parathyroid adenoma measuring 54 mm.

Given the severity of hypercalcemia and the history of lithiasis, a parathyroidectomy was performed at 21 weeks of gestation under general anesthesia. The procedure was uncomplicated, with complete excision of the adenoma. Serum calcium normalized in the immediate postoperative period (95 mg/L on day 1). The pregnancy continued uneventfully until term, with vaginal delivery of a healthy newborn. The newborn did not develop significant neonatal hypocalcemia, probably owing to the early normalization of maternal calcium levels during pregnancy [46].

#### Case 2: Unfavorable Outcome with Delayed Management

A 28-year-old primigravida presented with severe

PHPT diagnosed in the second trimester of pregnancy during the investigation of persistent gastrointestinal symptoms (intractable nausea, abdominal pain). The workup revealed hypercalcemia at 112 mg/L and elevated PTH at 146 pg/mL. Imaging identified two supracentimetric parathyroid adenomas.

Despite recommendations for prompt surgical intervention, the patient declined parathyroidectomy due to concerns about anesthetic risks. Intensive conservative treatment was initiated, including intravenous hydration and cinacalcet. However, serum calcium remained elevated despite this treatment (108–115 mg/L). At 28 weeks of gestation, intrauterine fetal death was diagnosed during a routine consultation. Histopathological examination of the placenta revealed diffuse placental calcifications, suggesting a direct role of chronic hypercalcemia in the fatal outcome [47].

### Case 3: Our Observation – Favorable Outcome with Conservative Treatment

Our patient presents several features that distinguish her case from the preceding ones:

- Preconceptional diagnosis allowing planned surveillance
- Asymptomatic hyperparathyroidism with normal serum calcium despite elevated PTH
- 3-cm parathyroid adenoma precisely localized by ultrasound and CT
- Close multidisciplinary monitoring throughout pregnancy
- Absence of maternal, obstetrical, or fetal complications

These elements illustrate that the conservative approach can be successfully considered in specific circumstances, provided there is rigorous patient selection, precise localization of the adenoma by imaging, and optimal follow-up.

### Proposed Decision Algorithm

Based on the analysis of the literature and our experience, we propose the following decision algorithm:

#### Conservative Treatment if:

- Normal or moderately elevated serum calcium (< 110 mg/L)
- Absence of severe symptoms
- Absence of complications (lithiasis, pancreatitis)
- Possibility of close multidisciplinary follow-up

Second-Trimester Parathyroidectomy if:

- Persistently elevated serum calcium > 110 mg/L
- Severe or disabling symptoms
- Maternal complications (lithiasis, pancreatitis)
- Failure of well-conducted conservative treatment
- Suspected parathyroid carcinoma

#### Parathyroidectomy in the First or Third Trimester:

- Only in cases of life-threatening emergency (hypercalcemic crisis, severe pancreatitis)

### Conclusion

Primary hyperparathyroidism during pregnancy remains a rare but potentially serious condition, requiring coordinated multidisciplinary management. Our observation illustrates the potential for a favorable outcome with conservative treatment in asymptomatic forms with normal serum calcium, provided that optimal clinical and biological surveillance is maintained.

Preoperative imaging—particularly cervical ultrasound and computed tomography—plays an essential role in the precise localization of parathyroid adenomas and in therapeutic planning. In our case, the concordance of imaging findings allowed complete characterization of the 3-cm adenoma in a right retro-thyroid inferior position, contributing to the decision to pursue conservative surveillance.

Comparative analysis of the literature underscores the crucial importance of early diagnosis and risk stratification. Second-trimester parathyroidectomy remains the gold-standard treatment in symptomatic forms or those associated with hypercalcemia > 110 mg/L, with a high success rate and a significant reduction in maternal and neonatal complications. Conversely, diagnostic or therapeutic delay can lead to catastrophic outcomes, including intrauterine fetal death.

Close neonatal follow-up is imperative, as neonatal hypocalcemia occurs in nearly half of cases, typically between the 5th and 14th postpartum day. A systematic screening protocol with regular neonatal calcium measurements should be established for all newborns of mothers with PHPT during pregnancy.

Looking forward, the creation of multicenter registries would help better define predictive factors for unfavorable outcomes and refine therapeutic recommendations. The development of new pharmacological agents,

such as next-generation calcimimetics, could provide additional therapeutic options for patients with surgical contraindications or those refusing intervention.

This observation highlights the need for a personalized approach that takes into account the severity of hypercalcemia, gestational age, precise imaging localization of the adenoma, patient preferences, and the availability of an experienced multidisciplinary team. Close collaboration between endocrinologists, radiologists, obstetricians, surgeons, and pediatricians is the cornerstone of optimal management of this complex condition.

## References

- Fraser WD (2009) Hyperparathyroidism. *Lancet* 374: 145-158.
- Bilezikian JP, Bandeira L, Khan A, Cusano NE (2018) Hyperparathyroidism. *Lancet*. 391: 168-178.
- Norman J, Politz D, Politz L (2009) Hyperparathyroidism during pregnancy and the effect of rising calcium on pregnancy loss: a call for earlier intervention. *Clin Endocrinol (Oxf)* 71: 104-109.
- Schnatz PF, Curry SL (2002) Primary hyperparathyroidism in pregnancy: evidence-based management. *Obstet Gynecol Surv* 57: 365-376.
- Kovacs CS, Fuleihan GE (2006) Calcium and bone disorders during pregnancy and lactation. *Endocrinol Metab Clin North Am* 35: 21-51.
- Dania Hirsch I, Vered Kopel, Varda Nadler, Sigal Levy, Yoel Toledano, et al. (2015) Pregnancy outcomes in women with primary hyperparathyroidism. *J Clin Endocrinol Metab* 100: 2115-2122.
- Kelly TR (1991) Primary hyperparathyroidism during pregnancy. *Surgery* 110: 1028-1033.
- Truong MT, Lalakea ML, Robbins P, Friduss M (2008) Primary hyperparathyroidism in pregnancy: a case series and review. *Laryngoscope* 118: 1966-1969.
- Shangold MM, Dor N, Welt SI, Fleischman AR, Crenshaw MC Jr (1982) Hyperparathyroidism and pregnancy: a review. *Obstet Gynecol Surv* 37: 217-228.
- Kristoffersson A, Dahlgren S, Lithner F, Järhult J (1985) Primary hyperparathyroidism in pregnancy. *Surgery* 97: 326-330.
- Ip P (2003) Neonatal convulsion revealing maternal hyperparathyroidism: an unusual case of late neonatal hypoparathyroidism. *Arch Gynecol Obstet* 268: 227-229.
- Som M, Stroup JS, Chennat J, Hammoud MA (2010) Outpatient parathyroidectomy for hyperparathyroidism during pregnancy. *J Surg Res* 159: 707-709.
- Rigg LA, Zvirbulis EA, Yen SS (1979) Cushing's disease in pregnancy. *Obstet Gynecol* 53: 90-94.
- Eren Berber, Rikesh T Parikh, Naveen Ballem, Carolyn N Garner, Mira Milas, et al. (2008) Factors contributing to negative parathyroid localization: an analysis of 1000 patients. *Surgery* 144: 74-79.
- Steven E Rodgers, George J Hunter, Leena M Hamberg, Dawid Schellingerhout, David B Doherty, et al. (2006) Improved preoperative planning for directed parathyroidectomy with 4-dimensional computed tomography. *Surgery* 140: 932-940.
- Aisling McCarthy, Sophie Howarth, Serena Khoo, Julia Hale, Sue Oddy, et al. Management of primary hyperparathyroidism in pregnancy: a case series. *Endocr Pract* 15: 476-482.
- Abood A, Vestergaard P (2014) Pregnancy outcomes in women with primary hyperparathyroidism. *Eur J Endocrinol* 171: 69-76.
- J S Gelister I, J D Sanderson, C R Chapple, J L O'Riordan, A G Cowie, et al. (1989) Management of hyperparathyroidism in pregnancy. *Br J Surg* 76: 1207-1208.
- Kort KC, Schiller HJ, Numann PJ (1999) Hyperparathyroidism and pregnancy. *Am J Surg* 177: 66-68.
- Schnatz PF, Thaxton S (2005) Parathyroidectomy in the third trimester of pregnancy. *Obstet Gynecol Surv* 60: 672-682.
- Wagner G, Transbøl I, Melchior JC (1964) Hyperparathyroidism and pregnancy. *Acta Endocrinol (Copenh)* 47: 549-564.
- Ludwig GD (1962) Hyperparathyroidism in relation to pregnancy. *N Engl J Med* 267: 637-642.
- Carella MJ, Gossain VV (1992) Hyperparathyroidism and pregnancy: case report and review. *J Gen Intern Med* 7: 448-453.
- Bruce J, Strong JA (1955) Maternal hyperparathyroidism and parathyroid deficiency in the child. *Q J Med* 24: 307-319.
- Kovacs CS, Kronenberg HM (1997) Maternal-fetal calcium and bone metabolism during pregnancy,

- puerperium, and lactation. *Endocr Rev* 18: 832-872.
26. F Callies, W Arlt, H J Scholz, M Reincke, B Allolio (1998) Management of hypoparathyroidism during pregnancy – report of twelve cases. *Eur J Endocrinol* 139: 284-289.
  27. Thomas BR, Stacpoole PW (1983) Neonatal hypocalcemia after maternal hyperparathyroidism. *Arch Dis Child* 58: 233-235.
  28. B L Salle 1, E E Delvin, A Lapillonne, N J Bishop, F H Glorieux (2000) Perinatal metabolism of vitamin D. *Am J Clin Nutr* 71: 1317S-1324S.
  29. Sweeney LL, Malabanan AO, Rosen H (2010) Decreased calcitriol requirement during pregnancy and lactation with a window of increased requirement immediately post partum. *Endocr Pract* 16: 459-462.
  30. Landing BH, Kamoshita S (1970) Congenital hyperparathyroidism secondary to maternal hypoparathyroidism. *J Pediatr* 77: 842-847.
  31. Cetani F, Saponaro F, Banti C, Cianferotti L (2018) Cinacalcet efficacy in patients with mild primary hyperparathyroidism: a prospective, double-blind, randomized study. *J Clin Endocrinol Metab* 103: 4063-4071.
  32. Nilsson IL (2014) Primary hyperparathyroidism. *Lakartidningen* 111: 884-887.
  33. Mestman JH (1998) Parathyroid disorders of pregnancy. *Semin Perinatol* 22: 485-496.
  34. Carmen Horjus, Inge Groot, Darryl Telting, Petra van Setten, Adriaan van Sorge, et al. (2009) Cinacalcet for hyperparathyroidism in pregnancy and puerperium. *J Pediatr Endocrinol Metab* 22: 741-749.
  35. M N Montoro 1, R J Paler, T M Goodwin, J H Mestman (2000) Parathyroid carcinoma during pregnancy. *Obstet Gynecol* 96: 841-844.
  36. John P Bilezikian, Maria Luisa Brandi, Richard Eastell, Shonni J Silverberg, Robert Udelsman, et al. (2014) Guidelines for the management of asymptomatic primary hyperparathyroidism: summary statement from the Fourth International Workshop. *J Clin Endocrinol Metab*. 99: 3561-3569.
  37. Dochez V, Ducarme G (2015) Primary hyperparathyroidism during pregnancy. *Arch Gynecol Obstet* 291: 259-263.
  38. Kristoffersson A, Dahlgren S, Lithner F, Järhult J (1985) Primary hyperparathyroidism in pregnancy. *Surgery* 97: 326-330.
  39. Schnatz PF, Curry SL (2002) Primary hyperparathyroidism in pregnancy: evidence-based management. *Obstet Gynecol Surv* 57: 365-376.
  40. Nelson B Watts, John P Bilezikian, Pauline M Camacho, Susan L Greenspan, Steven T Harris, et al. (2016) American Association of Clinical Endocrinologists Medical Guidelines for Clinical Practice for the diagnosis and treatment of postmenopausal osteoporosis. *Endocr Pract* 16: 3: 1-37.
  41. Kreiger N, Paré P (1987) Parathyroid adenoma presenting with acute pancreatitis in pregnancy. *CMAJ* 137: 841-842.
  42. Matthias G, Diaz JH (2006) Parathyroidectomy in pregnancy. *Am Surg* 72: 654-657.
  43. Lee JB, Kim SW, Ra YS, Lee JW (2012) Parathyroid carcinoma during pregnancy. *J Korean Surg Soc* 83: 326-330.
  44. Leadbetter GW Jr, Cooper RA (1970) Hyperparathyroidism in pregnancy. *Obstet Gynecol* 36: 550-555.
  45. Schnatz PF, Thaxton S (2005) Parathyroidectomy in the third trimester of pregnancy. *Obstet Gynecol Surv* 60: 672-682.