

# Pregnancy Unmasking Hypoparathyroidism

Pages with reference to book, From 250 To 251

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## Introduction

Hypoparathyroidism is a rare disorder that may be genetic, autoimmune, or secondary to thyroidectomy. Hypocalcemia in pregnancy is associated with a high incidence of foetal, neonatal and maternal complications. Therefore, diagnosis and treatment of this disorder is essential. We describe here a case in which idiopathic hypoparathyroidism was unmasked by pregnancy with post-partum remission.

## Case Report

A 26 years old lady with five months pregnancy, presented with the history of persistent pain in both arms and legs with exacerbation on exertion for three months, The pain had of late increased in severity with carpopedal spasm involving both hands. She was not on any treatment. Her past medical history was significant for similar pain, involving arms and legs, with an episode of spontaneously resolving carpopedal spasm during sixth month of her last pregnancy. The baby died apparently due to cord prolapse. Her serum calcium during that pregnancy was 5.7 mg/dl and serum phosphorus 2.6 mg/dl, She remained well after her delivery and had not been on any calcium supplement.

There was no family history of epilepsy or carpopedal spasm. On examination, Trousseau's sign was positive and persistent spasm of left hand was present. A viable 26 weeks pregnancy was confirmed. On laboratory investigation her hemoglobin was 10.1 gm/dl, serum albumin 3.2 gms/dl, calcium 4.5 mg/dl, magnesium 1.8 mg/dl and serum phosphorus 3.6 mg/dl. The serum parathormone (mid molecule assay) level was 0.17 ug/ml, (reference range was 0-0.27), With above findings, she was diagnosed to have idiopathic hypoparathyroidism and treated initially with calcium gluconate infusion and later with oral calcium carbonate and active calcitriol 0.5 ug/day; active calcitriol was later increased to 1.0 ug/day. She had an uneventful pregnancy and delivered alive male child with an Apgar score of 9/10. Post-partum she did not appear for follow up. When contacted after eight weeks, she was not using any calcium or vitamin D supplements, was asymptomatic and her serum calcium and phosphorus were in the normal range.

In her case, on both the occasions, pregnancy unmasked hypoparathyroidism with remission post-partum.

## Discussion

The diagnosis of idiopathic hypoparathyroidism in our patient was based on clinical evidence of carpopedal spasm associated with low serum calcium and inappropriate PTH level for the hypocalcemia. Hypophosphatemia, an inconstant finding in hypoparathyroid patients, was not present. Normal pregnancy leads to a combination of adaptive metabolic responses, the end-result of which is to assure adequate delivery of mineral to the fetus while affording protection to the maternal skeleton. Intestinal calcium absorption increases about two-fold during pregnancy<sup>1</sup>, particularly in the last trimester<sup>2</sup> and urinary calcium excretion also increases as it parallels glomerular filtration rate. Pregnancy has been called a physiologic absorptive hypercalciuric state<sup>3</sup>. Increased activation of vitamin D, prolactin secretion and intestinal hypertrophy, all may be contributory factors<sup>4</sup>.

Hypercalciuria is exacerbated in hypoparathyroidism during pregnancy because of a decreased net tubular reabsorption of calcium by the kidney.

Although studies using two-site immunoradiometric assays report stable intact PTH levels within the normal range throughout pregnancy<sup>5,6</sup>, the increased demand placed by pregnancy may be responsible for unmasking subclinical hypoparathyroidism, as seen in our case. Therefore, the concept of hyperparathyroidism of pregnancy, referring specifically to circulating levels of PTH is not valid, but a state of functional hyperparathyroidism may occur reflecting increased actions of parathyroid-related protein (PTHrP). The PTHrP is responsive to estrogen and to a variety of cytokines<sup>7</sup>. Elevated PTHrP levels have been found in healthy pregnant women compared with non-pregnant control women<sup>8</sup>. The source of circulating PTHrP in pregnancy is not known. Some believe it may be placental whereas others postulate that local production in the mammary glands would gain access to maternal circulation<sup>9</sup>.

Treatment consists of dietary phosphate restriction and calcium, vitamin D and magnesium supplementation. In the second half of pregnancy, vitamin D therapy often must be increased to maintain normocalcemia<sup>10</sup>. Our patient was also managed on this conventional regimen and had an uneventful delivery. If conventional treatment is not successful, the addition of thiazide diuretics and sodium restriction may reduce renal losses<sup>11</sup>.

In our case PTHrP was not measured due to non-availability of assay and the diagnosis of idiopathic hypoparathyroidism was made; it remains a possibility that this state of functional hypoparathyroidism was in essence due to deficiency of PTHrP rather than PTH.

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