



Maternal hypoparathyroidism related transient normocalcemic hyperparathyroidism in newborns: case report

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Abstract

Maternal hypoparathyroidism causes enlargement in fetal parathyroid glands and hyperparathyroidism. This kind of neonatal hyperparathyroidism may lead to hypercalcemia. Normocalcemia may be observed in some cases. This temporary presentation of hyperparathyroidism generally recovers in a few days after birth as the infant takes enough amount of calcium and phosphorus. Especially significant intrauterine growth retardation and mortality rates are high in infants born from mothers with poorly controlled or untreated hypoparathyroidism. Contrary to the severe hyperparathyroidism of the newborn, these infants often have low birth weight, suppressed or normal serum calcium levels and normal or a little high serum phosphorus levels. The reasons for this difference between the two groups are unknown. In this article, literature was examined by presenting the case of an infant who had transient normocalcemic hyperparathyroidism and the mother who had thyroidectomy and hypoparathyroid symptoms and inadequate treatment during pregnancy.

Keywords: Maternal Hyperparathyroidism; Neonatal Hyperparathyroidism; Hypocalcemia.

INTRODUCTION

It may be fetal and newborn hyperparathyroidism secondary to different maternal hypocalcemia causes included poorly controlled maternal hypoparathyroidism (1,2), maternal pseudohypoparathyroidism, maternal or newborn renal tubular acidosis (3,4). The newborn with temporary normocalcemic hyperparathyroidism born from a mother who had insufficient treatment during pregnancy and had hypoparathyroidism presentation after thyroidectomy was presented in this case presentation to emphasize that the mothers who had thyroidectomy should be followed up appropriately during pregnancy and their treatment should not be hindered.

CASE REPORT

Our case was hospitalized on postnatal fifteen minutes. The mother of patient had thyroidectomy operation three years ago and hypoparathyroidism developed during pregnancy due to irregular medicine use. It was

learned that the case was born as the third child of a thirty-four year old mother on the 38th week of pregnancy through caesarean operation, the mother had total thyroidectomy three year ago, a treatment containing 150 microgram L-thyroxin, 3x0.5 microgram calcitriol and 3x2 gr calcium lactate but she couldn't take her medicine during pregnancy. Serum calcium was 6.1 (recovered calcium 7.22) mg/dl, phosphorus 4.8 mg/dl, magnesium 0.8 mmol/L, alkaline phosphatase 207 U/l, parathormone 6.4 pg/ml, total protein 5.1 g/dl, albumin 2.1 g/dl, FT4 0.937 ng/dl and TSH was 23.58 mU/L in the prenatal laboratory tests of the mother.

Among the physical measurements of the infant, height was 50 cm, weight was 3450 g, head circumference was 35.5 cm, chest circumference was 33 cm and other system examinations were evaluated as normal. In the laboratory tests made on postnatal fourth hour, serum calcium was found 9.1 mg/dl, phosphorus 6.2 mg/dl, magnesium 0.9 mmol/L, alkaline phosphatase 225 U/L, parathormone 510 pg/ml and calcitonin 12.3 pg/ml. In the thyroid function tests made on the second day, FT4 was measured 1.76 ng/dl, TSH 16.72 mU/L and thyroglobuline 16.72 ng/ml. In the tests of the infant made on the fourth day, calcium was measured 9.4 mg/dl, phosphorus 4.2 mg/dl, magnesium 1.1 mmol/L, alkaline phosphatase 480 U/L, albumin 3.2 g/dl and parathormone 218 pg/ml. When the infant was 30 days old, calcium and phosphorus levels were normal and parathormone level was 62 pg/ml.

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DISCUSSION

Hypoparathyroidism is a problem with a prevalence of 0.5-6.6% (5). Thyroidectomy and thyroid ablation treatment are generally present in the etiology (5,6). It may be idiopathic or may occur as a component of type 1 autoimmune polyglandular syndrome (autoimmune hypoparathyroiditis, Addison disease, mucocutaneous candidiasis, primary hypogonadism, alopecia, vitiligo, partial cellular atrophy, type 1 diabetes, hypothyroidism, autoimmune hepatitis) (5-7). Our case had thyroidectomy operation three years ago and hypoparathyroidism developed during pregnancy due to irregular medicine use. Fetal hypocalcemia occurring due to untreated maternal hypoparathyroidism causes hyperplasia and parathormone increasing in fetal parathyroid glands and neonatal transient hypercalcemia occurs (8,9).

Neonatal hyperparathyroidism presentation is observed in inactivated mutations of calcium sensitive receptors apart from maternal hypoparathyroidism and rarely in parathyroid adenoma (6,10). Diagnosis is provided by high calcium, low phosphorus and high alkaline phosphatase and parathormone levels in the cases and in inactivated calcium sensitive receptor mutations, the disease is diagnosed through genetic studies, hypocalciuria and high serum magnesium level (10). Maternal hypoparathyroidism related neonatal hyperparathyroidism cases are temporary (6). Calcium may not be high in some hyperparathyroidism cases and this is called normocalcemic primary hyperparathyroidism (10,11). High parathormone level is typical in cases when recovered and ionized calcium is at normal level (12). Most of the cases published were adults and the treatments were surgical.

While the albumin level was normal in our case, calcium level was found low, parathormone was high and magnesium level was normal and the phosphorus level was at the lower limit on the fourth day. So the infant was observed after normocalcemic hyperparathyroidism diagnosis, since calcium, phosphorus and parathormone levels were in normal limits on postnatal 30th day, transient normocalcemic hyperparathyroidism was the final diagnosis.

As a result, we would like to emphasize that maternal hypoparathyroidism may cause neonatal hyperparathyroidism and may occur as normocalcemic transient hyperparathyroidism as in our case and the mothers who had thyroidectomy should be observed well during pregnancy and treatments should be followed regularly.

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