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Neonatal Hypocalcemia Due to Asymptomatic Maternal Primary Hyperparathyroidism

Neonatal hypocalcemia resulting from maternal primary hyperparathyroidism (MPH) is usually detected clinically in the first 2 weeks of life. Occasionally, diagnosis of primary hyperparathyroidism in a young asymptomatic mother is made when the infant presents with hypocalcemia. We present an infant with late onset hypocalcemia resulting from a combination of transient hypoparathyroidism due to asymptomatic MPH and vitamin D deficiency.

A thirty-five-day-old infant was admitted to Pediatric Emergency Service because of recurrent tonic-clonic convulsions for 36 hours. He was a full term male baby born to a 22 year old G2, P2 woman by normal spontaneous vaginal delivery with a birth weight of 3500 g. He was only breast fed. The family history revealed that his brother was hospitalized and treated because of neonatal hypocalcemic convulsion but no further investigations were carried out.

On physical examination his anthropometric measurements were normal. He had no dysmorphic features. Initial laboratory evaluation showed hypocalcemia, hyperphosphatemia slightly increased alkaline phosphatase (ALP), inappropriately low PTH level for concurrent degree of hypocalcemia and sligthly low 25-OH vitamin D_3 (*Table I*). The remainder of the laboratory findings were unremarkable. Based on these findings, he was diagnosed as hypoparathyroidism and started on calcium boluses. After his calcium level reached 7mg /dL, oral calcium (75 mg/g/day) and vitamin D_3 supplements (800 IU/day) were given. He remained asymptomatic after second day on admission. Since he had no findings consistent with DiGeorge syndrome and he had a positive family history, maternal hyperparathyroidism was investigated. The mother had high levels of calcium and PTH and low level of phosphorus. She was diagnosed as hyperparathyroidism and referred to endocrinologist. A technetium scan showed an area of increased uptake on the right side of neck suggesting a parathyroid adenoma. It was removed by surgery.

The presentation of asymptomatic maternal hyperparathyroidism by convulsion in an infant is exceedingly rare. Hyperparathyroidism in asymptomatic mothers might easily have been missed if the maternal calcium status had not been investigated(1-4) like in our patient's mother. Suppression of the fetal parathyroid gland by maternal hypercalcemia often causes transient neonatal hypocalcemia(5). Low vitamin D levels of the patient might have exacerbated the hypocalcemia observed in this infant which may due to several causes : he has not received

TABLE I–*Laboratory Data and Treatment Details.*

	Normal ranges	Patient at admission	Follow up						
			2nd day	5th day	10th day	2nd wk	5th wk	16th wk	Mother
Ca(mg/dL)	8.5 -10.5	4.6	7.4	8.2	8.6	8.8	9	8.9	11.2
P(mg/dL)	2.7 - 6.7	8.6	5.9	5.1	4.7	4.7	4.2	4.1	2.2
ALP(IU/l)	60 - 250	438	437	464	464	460	–	420	519
Mg(mg/dL)	1.2 - 2.5	1.7	1.7	1.8	–	–	–	–	1.0
PTH(pg/mL)	15 - 65	19	32	–	64.1	–	–	–	1402
25 OH VD3 (ng/mL)	10 - 40	5	–	–	–	–	35	–	–
Treatment		Ca IV	Ca IV+PO D3(800 IU/d)	Ca IV+PO D3 (800) IU/d	D3 (800 IU/d	D3 (800 IU/d	D (800 IU/d	D3 (800 IU/d	Surgery*

*Parathyroid adenoma was removed.

vitamin D supplement , he has grown rapidly and there could have been low placental transfer of 25-OH D_3 from the mother who had increased conversion of 25-OH D_3 to 1,25-OH D_3 due to her hyperparathyroidism. We emphasize that all mothers with neonates showing tetany or hypocalcemic convulsions should have serum calcium concentrations measured to exclude primary hyperparathyroidism even if the mother is asymptomatic.

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