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Case Report

Asymptomatic parathyroid adenoma in mother as a cause of late-onset persistent hypocalcemia in a newborn

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ABSTRACT

Pregnant women with primary hyperparathyroidism may be asymptomatic or have mild symptoms such as fatigue, thirst, constipation, or transient depression. Transfer of calcium from mother to fetus leads to increased fetal calcium concentrations, suppressing fetal parathyroid hormone synthesis, and stimulating calcitonin secretion leading to neonatal hypocalcemia. Here, we present a report of a newborn admitted on day 10 of life with recurrent tonic convulsions. On investigation, it was found that the baby had severe persistent hypocalcemia which when further evaluated was due to asymptomatic maternal hyperparathyroidism due to parathyroid adenoma.

Keywords: Persistent late-onset hypocalcemia, Neonate, Maternal hyperparathyroidism, Seizures, Parathyroid adenoma

INTRODUCTION

Hypocalcemic seizures and tetany in newborn secondary to maternal hyperparathyroidism are rare. Intrauterine suppression of the fetal parathyroid glands is caused by the hyperfunction of the maternal parathyroid glands. Early neonatal hypocalcemia is more common within the first three days and is usually seen in preterm newborns. Delayed neonatal hypocalcemia often occurs toward the end of first week or during the second week and is often due to feeding with cow's milk or formula with high phosphate content. Here, we report a case of late-onset persistent hypocalcemia in a term newborn born to a mother with asymptomatic parathyroid adenoma.

CASE REPORT

A full-term male neonate (birth weight of 2601 g) born by normal vaginal delivery was admitted to the neonatal unit on day 10 of life with generalized tonic convulsions since day four of life. There was no history of birth asphyxia. The mother was para 1, gravida 1 with no risk factors for sepsis. The baby was mixed-fed with breast milk and infant formula. Physical examination was within normal limits with no dysmorphism. The initial blood glucose level was normal. The sepsis screen and cerebrospinal fluid examination were normal. Serum electrolytes were suggestive of severe hypocalcemia [Table 1]. Parenteral calcium was started at a dose of 8 mL/kg/day of calcium gluconate. Despite intravenous calcium, serum calcium remained low, so to evaluate the cause of persistent hypocalcemia, vitamin D levels [25(OH)D3], and serum magnesium

Table 1: Laboratory data and treatment details.

	Normal range	Patient at admission	Follow-up				
			2nd day	5th day	10th day	2nd week	Mother
Ca – Total (mg/dL)	8.5–10.5	5.07	5.58	6.96	7.05	9.53	11.65
Ca – Ionic (mg/dL)	4.5–5.5	1.52	2.52	3.16	2.92	4.68	5.88
Phosphorus (mg/dL)	2.7–6.7	8.73	9.37	9.25	7.90	3.27	2.80
ALP (IU/L)	60–250	634.38	511.75	-	-	756.10	126
Mg (mg/dL)	1.2–2.5	1.16	1.87	-	-	1.97	2.07
PTH (pg/mL)	15–65	32.10	40.2	-	-	44.4	201
25-hydroxyvitamin D3 (ng/mL)	10–40	8.60	-	-	-	22.3	55.61
Urine calcium (mg/dL)	0.50–35.70	-	0.96	-	-	-	19.10
Urine creatinine (mg/dL)	16.0–327.0	-	37.91		-	-	51.00
Treatment		Ca IV, Mg IM	Ca IV+PO, Mg PO	Ca IV+PO, Vit D 2000 IU	Ca PO, Vit D 2000 IU	Ca PO, Calcitriol 75 ng/kg	

Ca: calcium, ALP: Alkaline phosphatase, PTH: Parathyroid hormone, Mg magnesium, IM: Intramuscular

(Mg) were sent. Vitamin D levels were deficient (<10 ng/mL; hence, oral vitamin D in a dose of 2000 international units/day was started. Serum magnesium was borderline low and parenteral magnesium sulfate was supplemented at a dose of 0.2 mL/kg/day for three days followed by an oral supplement. Serum phosphorus level was high and serum parathyroid hormone (PTH) levels were inappropriately low. The kidney function tests were normal. Urine calcium to creatinine ratio was within normal limits with no hypercalciuria. Since high phosphate levels could not be explained by vitamin D deficiency, maternal samples were sent which showed hypercalcemia and hypercalciuria along with an increased level of PTH of 201 pg/mL. The diagnosis of maternal primary hyperparathyroidism was made which was further supported by a sestamibi parathyroid scan [Figure 1] which revealed a left inferior parathyroid adenoma. The bone mineral density evaluation showed osteoporosis. The fine needle aspiration cytology was suggestive of an adenomatoid goiter. The baby was started on oral calcitriol at a dose of 60 ng/kg/day with oral calcium of 50 mg/kg/day. Over the next one week, the neonate had no convulsions. Two weeks later, both calcium and magnesium concentrations were within the normal ranges. Vitamin D_3 supplementation and oral calcium with calcitriol supplementation were planned for at least six weeks.

As the mother was asymptomatic and her physical examination was normal, she was advised evaluation for surgery. She underwent a total thyroidectomy with a left inferior parathyroidectomy. Gradually, her serum PTH level became normal after surgery from post-operative day 1. She was discharged on oral calcium carbonate 500 mg twice a day with danger signs of hypocalcemia explained.

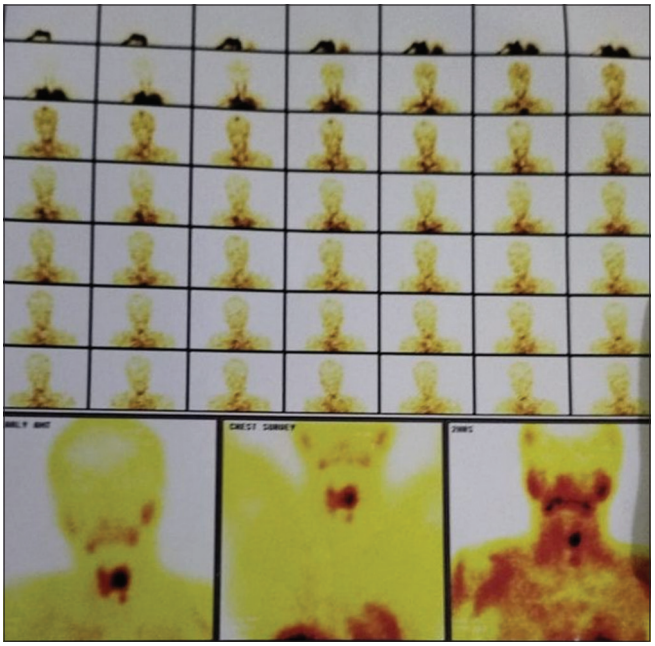

Figure 1: Tc-99 m sestamibi scan of patient's mother showing left inferior parathyroid adenoma.

DISCUSSION

Our patient presented with late-onset neonatal seizures due to hypocalcemia secondary to maternal hyperparathyroidism since day four of life. There are reports of similar interesting presentations in which newborns presented with recurrent tonic convulsions as the first manifestation of transient hypoparathyroidism due to asymptomatic maternal hyperparathyroidism and vitamin D deficiency. On further evaluation, parathyroid adenoma in the mothers was detected.[1,2]

Treatment for neonatal hypocalcemia consists of calcium administration and the use of vitamin D when deficiency/insufficiency is present. Thomas *et al.* and Jain *et al.* have recommended a duration of one-two months of treatment.[3,4] Hypomagnesemia sometimes may be an associated finding, which may also lengthen the recovery time.[3,5] In our case

too, hypomagnesemia and vitamin D deficiency were present. In our patient, 48 h of intravenous 10% calcium gluconate followed by oral calcium gluconate along with magnesium and vitamin D_3 supplementation was given with a plan to continue calcium with calcitriol supplementation for at least 6 weeks. The patient responded satisfactorily to the treatment [Table 1].

Fetal complications of maternal hyperparathyroidism include intrauterine growth restriction, preterm delivery, and a three- to five-fold increased risk of miscarriage. Neonatal complications include low birth weight and features of symptomatic hypocalcemia. In our case, baby was appropriate for gestational age with normal birth weight.[6,7]

During pregnancy, up to 80% of women with primary hyperparathyroidism are asymptomatic, so the diagnosis is often missed as other minor complications of pregnancy mimic similar symptoms. Therefore, maternal serum PTH levels in addition to calcium and phosphorus levels must be included while investigating for neonatal hypocalcemia.[1,8] In our case, the mother had a history of painless swelling in the neck for the past 4 years with no other symptoms during pregnancy. McCarthy *et al.* suggested the risk of developing maternal complications such as hyperemesis gravidarum, pre-eclampsia, hypercalcemic crises, nephrolithiasis, and pancreatitis in pregnant women with hyperparathyroidism and hypercalcemia.[8]

The presentation and findings in the newborn and the mother emphasize that undiagnosed maternal hyperparathyroidism may cause severe hypocalcemia requiring treatment and hospitalization and the pediatricians should keep this possibility in mind while looking for the cause of hypocalcemic seizures. While treating newborns with hypercalcemic seizures, maternal hyperparathyroidism should be excluded by doing an appropriate workup.[1]

CONCLUSION

Cases of neonatal hypocalcemia should be vigilantly investigated and followed.

Learning Points

- Apart from evaluating common causes of hypocalcemia in a neonate, the pediatricians should always keep in mind the possibility of maternal hyperparathyroidism.
- Proper evaluation and management will lead to a better outcome in the newborn and the mother.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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