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ARF with Nephrocalcinos is-Rare Presentation of Vitamin D Deficiency

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I. Introduction

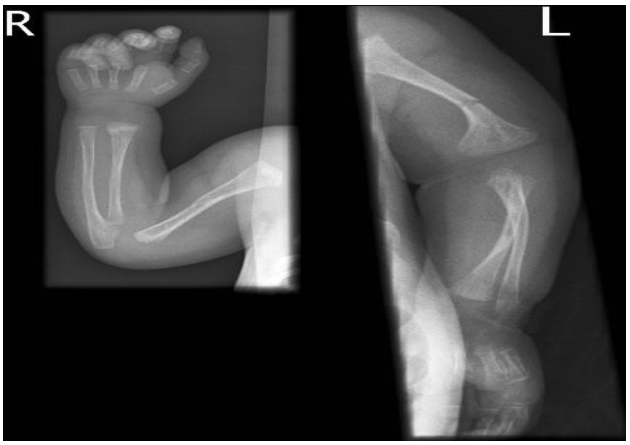
Vit D deficiency most commonly occurs in infancy due to combination of poor intake, inadequate cutaneous synthesis1. Decreased maternal vitamin D also contribute both by leading reduced vitamin D content in breast milk and by lessening trans placental delivery of vitamin D. Vitamin D deficiency causes an increase of serum PTH leading to bone resorption , osteoporosis and fractures. A negative relationship exists between serum 25(OH)D and serum PTH5.High PTH in turn causes hypercalcemia and significant hyperphosphaturia2. Hypercalcemia usually defined as serum calcium concentration greater than 11mg/dl and ionized calcium greater than 5mg/dl(1.4mmol/l) and associated with several clinical entities2. It may due to iatrogenic (calcium salt),parathyroid hyperfuntion, Williams syndrome, idiopathic infantile hypercalcemia, hypervitaminosis D, phosphate depletion and bartter's syndrome. Hypercalcemia may occur in a breast-fed pre-term infant due to feeding with low phosphate milk3. Here we are reporting a case of neonatal hypercalcemia, hypercalciuria and nephrocalcinosis who devolped ARF in a full term baby fed on low-phosphate breast milk as her mother had hypophosphatemia and severe vitamin D deficiency.

II. Case Report

A 12 day old baby girl born to primigravida mother with apparently normal antenatal period, full term, by normal vaginal delivery with Birth weight 3.5kg, cried immediately after birth, was on breast feeding till day 9 of life. Parents complaints of dullness, not accepting feed for last 2 days, not passed urine for 1 day for which baby was admitted at one of the Govt. Medical College in northern India where baby was treated in the line of ARF(acute renal failure) with peritoneal dialysis and was ventilated.

Baby shifted to our hospital (private tertiary care centre in same city) on day 12 of life with endotracheal tube in situ, PD catheter in situ, IV line with Foley's catheter in situ. On admission baby's Wt. was 3.1 kg, HC 34cm ,length 50cm,no dysmorphic features , dull, had one brief episode of seizure, AF at level, CFT<3secs,HR 134/min, mean BP 57mmHg(75/48) ,SPO2 100% with minimal ventilatory support, B/L air entry was equal, in CVS S1S2 normal with urinary output 3.8ml/kg/min. Immediate blood sugar was 254mg%,sepsis screen was negative, ABG showed alkalosis. Other significant lab parameters were ionic Calcium 1.48 (hypercalcemia) with total calcium 11mg%, s .creatinine-4.1, BUN 114,urinary Ca/Cr 0.64(s/o hypercalciuria) serum sodium 155,potassium 4.8,magnesium 1.1mg%, Parathyroid 281pg/ml (hyperparathyroidism) with low phosphorus 6.1,ALP129 with Vitamin D<8ng/ml. USG done outside on day 11 of life showed B/L Nephrocalcinosis with right renal cyst. Repeat USG on day 14 of life at our institute reviles calcium deposition in both kidneys with right renal cyst with minimal ascities. 2D ECHO was normal and skeletal survey showed osteopenia and osteolytic lesions on long bones.USG Cranium was normal. Maternal serum calcium was 9mg %(normal), serum phosphorus was 3.5mg% (low), Parathyroid high with vitamin D level is<8ng/l (severe deficiency) with phosphorus level in mother's milk significantly low 3.5.(normally breast milk contains 27-32mg/dl calcium and 14-15mg/dl phosphorus) Baby's dehydration part corrected and managed by IVF n/3saline (1and ½ times of maintenance fluid) and frusemide infusion for hypercalcaemia, and baby was extubated .Formula feeding was started and breast milk stopped as mother's hypophosphatemic milk is implicated possibility of hypercalcemia. Baby and mother both were treated with vitamin D 3 IM injection. Subsequently serum creatinine and BUN and serum sodium came down to normal.

On d3 of admission (d16 of life) Na133, K+3.7, Ionic Ca++1.1, serum creatinine 1.0, BUN 32. On D7 of admission serum creatinine was 0.3. Baby was passing urine normally, accepting feed well and gaining weight as expected. At one month of life baby's (6-2-15) PTH 103pg/ml, vitamin D 37.4ng/ml, serum phosphorus 3.01mg/dl. After 1 wk of vitamin D injection mother's vitamin D level 20.7ng/ml and subsequently (day10 of vitamin D inj) mother's milk phosphorus become 14.3 mg/dl, which is normal. Breast feeding is reestablished.



III. Discussion

Based on the physiology of vitamin D, its content in human milk is related to the lactating mother's vitamin D status, infants who are exclusively breastfed but who do not receive supplemental vitamin D or adequate sunlight exposure have a high prevalence of vitamin D deficiency based on serum 25(OHD)levels5. In our case both mother and baby found to be vitamin D deficient and increased PTH level.

Hypercalcemia may be asymptomatic and discovered incidentally or may manifest dramatically and may be life threatening, requiring immediate intervention. The clinical findings may include poor feeding, vomiting, constipation, polyuria, hypertention, tachypnea, dyspnea, hypotonia, lethargy and seizures2. Nephrocalcinosis, nephrolithiasis, diffuse bone underminerilisation are well recognized hypercalcemic complications2. . In a hypercalcemic infant baseline total and ionic calcium,phosphorus,urinary Ca/Cr,serum PTH level should be performed.

A low urinary calcium suggests familial hypocalciuric hypercalcemia. Whereas high urinary calcium with high PTH level implicates primary hyperparathyroidism 6. Neonatal severe hyperparathyroidism is a rare cause of hypercalcemia caused by homozygous mutation of calcium sensing receptor with feeding difficulties ,respiratory distress ,hypotonia ,failure to thrive and unexplained bone fractures7. Primary hyperthyroidism is the most common cause asymptomatic hypercalcemia and patients may have a family history of multiple endocrine neoplasia(type 1 and 2).The diagnosis is made by demonstrating persistent hypercalcemia in presence of inappropriately normal or elevated PTH9.In our case, this cause was excluded as there was prompt decline of calcium in response to formula feed and other supportive measures. Williams syndrome, characterized by supravulvular aortic stenosis, elfin-like faces and hypercalcemia6,was excluded by normal echocardiogram. Clinical adjustment of TPN (total prenatal nutrition) by removing the phosphorus can rapidly lead to hypercalcemia, especially in VLBW infants9.

Neonatal hypercalcemia due to phosphate depletion usually occurs in low birth weight babies who are fed unsuplimented human milk that lead to hypo phosphatemia, increased renal vitamin D3 and increased calcium absorption and suppressed PTH2. But in our case PTH was elevated due to severe vitamin D deficiency. Nephrocalcinosis implies deposition of calcium in the substance of kidney. In infants it may cause renal failure without stone formation4. In our case ARF partly caused by nephrocalcinosis(due to hypercalcemia) and partly due to dehydration by hypercalcemia/hypercalciuria(prerenal component).Hypophosphatemic mother milk, increased PTH ,Vitamin D deficient status were the correct explanation of hypercalcemia in our term baby.

Transient neonatal hyperparathyroidism is described in a phenotype of mucolipidosis type 2 which was resolved with vitamin D treatment10. In our case when infant was treated with vitamin D and top feed (containing adequate phosphorus), hyperperathyroidism was resolved, biochemically and radiologically. Similarly Hazza et al reported a case of hypercalcemia,hypercalciuria and nephrocalcinosis(in a breast fed baby) secondary to maternal hypophosphatemia and maternal vitamin D deficiency11 .

IV. Conclusion

Although hypercalcemia in preterm baby who is fed with hypophosphatemic breast milk is a well known problem, it may occur in a term baby with transient neonatal hyperparathyroidism due to severe vitamin D deficiency both mother and baby.

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