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Dyspnea as an Unusual Presentation of Rickets in A 3-Month-Old Infant: A Case Report

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ABSTRACT

Rickets, a condition caused by a deficiency of vitamin D, has a myriad of presentations and affects children all over the world. Infants who are exclusively breastfed by mothers who are vitamin D deficient are affected, particularly in the absence of vitamin D supplementation during pregnancy. While breast feeding is encouraged, breast milk is deficient in vitamin D thus; exclusively breastfed infants require vitamin D supplementation. This case describes a three-week-old male infant with a continuum of rickets, which went undiagnosed from the initial presentation of noisy, difficult breathing and snoring at the pediatric emergency department. This case emphasizes that rickets may not always have a classical presentation.

Keywords: Rickets, Nigerian, Vitamin D deficiency, Immigrant mothers.

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1. INTRODUCTION

Rickets, a disease described by Whistler and Gleason in 1645 and 1650 respectively occurs when there is poor mineralization of osteoid. It is prevalent in developed and developing countries. It may or not be symptomatic and can occur from infancy up until childhood [1-3]. Rickets is caused by Vitamin D deficiency, which leads to a reduction in intestinal absorption as well as anomalies in bone formation and mineralization [4]. In immigrant populations, childbearing women have vitamin D deficiency, a risk factor for developing rickets. During pregnancy, maternal vitamin D requirements are increased because of the requirements of the developing fetus. If vitamin D is deficient, infantile rickets may ensue. Exclusive breastfeeding of infants without vitamin D supplementation may complicate the situation [5, 6].

2. CASE REPORT

A 3-week-old American born male infant of Nigerian parents was delivered at 37 weeks gestation. Birth weight was 2.58kg. Immediate post delivery period showed transient tachypnea of the newborn; the child required continuous positive airway pressure in the special care nursery for 20 minutes of life which was weaned off after 4 hours. Successful breastfeeding started the following morning.

He initially presented after an elective bilateral inguinal hernia repair with noisy breathing i.e. occasional stridor and snoring worse at night. Feeding and weight gain not affected. Congenital laryngeal stridor was considered; ENT performed endoscopy which was determined to be normal. On examination, apart from noisy breathing, vital signs were normal, there was bilateral inguinal hernia also noticed at birth. On observation, complications were sought and these included choanal atresia (NG tube passed successfully down both nostrils), adenoids were normal, mild tracheomalacia and absent subglottic stenosis. Before the inguinal hernia repair, the patient was given Loratadine daily, however, no investigations were done since feeding and weight were normal.

Considering the weather (winter) at the time, the patient was determined to have common cold which was not expected to impact surgery, stridor however did not resolve prior to surgery. At 3 months of age, the patient presented for an elective bilateral hernia repair. Surgery was uneventful until extubation. The patient's noisy breathing resumed and vital signs deteriorated. There was respiratory distress, increased work of breathing and hypoxia Re-intubation was required with transfer to the intensive care unit. Considering the background history of noisy breathing, the patient remained in the intensive care unit. A working diagnosis of mechanical obstruction probably caused by edema was made, however on review by the otolaryngologist, no finding suggested this. Reviews by the cardiologist and pulmonologist added no new finding. The infant had clinical stigmata of nutritional rickets which included subtle rickety rosary, osteopenic long bones and mild cupping and fraying of metaphyses on x-ray. Blood investigations showed severe hypocalcemia, undetectable Vitamin D levels and extremely high Parathyroid hormone [calcium: 1.17 mmol/l, corrected calcium: 1.31mmol/l, PTH: 53 pmol/l, others include: Phosphorus normal: 2mmol/l; ALP very high: 1511; GGT: high 420. Features suggestive of rickets were found on chest and extremity x-rays (as discussed above). Emergent treatment began with calcium replacement and supranormal doses of vitamin D (10, 000 IU daily). It was concluded that the patient had laryngospasm, which was caused by severe hypocalcemia in the patient which was not to be confused with Laryngismus stridulus. It was deemed safe to extubate the patient five days later. The patient's mother had her vitamin D levels checked and was deficient of vitamin D. She had migrated to the US from Nigeria when she was five months pregnant. She received her prenatal care in Nigeria but did not receive vitamin D supplementation during pregnancy. The mother's blood investigations showed that she did not have a disorder of calcium metabolism. She exclusively breast fed her baby. Rickets was not apparent in the patient from the well-educated middle class parents' point of view. The mother began to receive very high vitamin D supplements. The patient continued to receive vitamin D and calcium supplements. For follow up, the pediatric endocrinologist recommended alternate day vitamin D supplement for the patient and the patient continues to be monitored on an outpatient basis yearly, he has vitamin D levels checked yearly. The patient is however well developed and nourished. at the time of writing this report. Developmental milestones at 1yr: Weight – 10.5kg (75^{th} percentile); Length – 75cm (50^{th} percentile); head circumference – 47cm (80^{th} percentile) and was Walking with support.

3. DISCUSSION

Difficult extubation after surgery uncovered vitamin D deficiency so it is understandable therefore why rickets is rarely an anesthetist's diagnosis.

In early pregnancy, twice the amount of calcium usually required is absorbed from the intestine as a result of increased calcitriol synthesis; this reaches a peak in the third trimester [3, 7, 8]. The placenta transports calcium, phosphorus and magnesium to the fetus such that there are increased levels in the fetus, which implies that there is low fetal calcitriol and parathyroid hormone (PTH) [9]. In fetal life, transplacental transfer of Vitamin D is not required for calcium to be maintained in the fetus however, Vitamin D and 1, 25OHD move through the placenta prior to delivery and vitamin D stores in the fetus are based on maternal supply. Calcium levels decrease sharply after delivery. The newborn now depends on calcitriol for the absorption of calcium and adequate bone mineralization. 25 hydroxyvitamin D (25-(OH) D) has a short half-life, the quantities reduce significantly and this makes requirement for vitamin D essential [10].

Breast milk contains between 12-60 IU/L even with adequately ingested amounts of vitamin D, which is not sufficient for infants' unexposed to sufficient sunlight. Breast milk may not be in itself low in vitamin D, however it may be related to vitamin D levels in the mother. Lactating women lose calcium significantly through breast milk several times over than the fetus requires [11].

Rickets has a plethora of manifestations, which includes dyspnea like in this patient. Biochemical findings are

dependent on the duration of the vitamin D deficiency and PTH feedback and response; these findings include: low serum total calcium, serum phosphate could be normal, high or low, serum alkaline phosphatase, abnormally low serum 25- OH (D), normal or elevated PTH levels [1, 2]. Associations of infantile rickets with iron deficiency exist in the infants for which mothers are also deficient in serum 25-OH (D). Vitamin D supplementation is important for deficient mothers [3]. Exposure of the infant to sunlight about 2-3 hours per week would be beneficial to prevent rickets, however estimates are not available for dark pigmented infants. Infants should receive foods fortified with vitamin D, fortified formula provides 400IU/day of vitamin D, and routine supplementation of vitamin D of 400IU/day for breastfed infants is encouraged [1, 3, 9]. The recommended daily intake of vitamin D for infants is 200IU vitamin D per day according to the American Association of Pediatrics, which should begin from 2 months of age till weaning has occurred. Infants with rickets may require calcium and oral doses of vitamin D [between 1,000-10,000 IU] with monitoring of biochemical and radiological parameters [11].

4. CONCLUSION

Vitamin D for mothers and infants prevents infant morbidity and mortality. It should be given the same consideration as vaccinations for infants. Rickets is preventable. Physicians should educate mothers about vitamin D supplementation in prenatal care and for infants and vitamin D deficiency breast milk and the different sources of vitamin D including fortified formula and sunlight even in countries that have many sunlight hours.

Physicians should have a high index of suspicion of rickets and its protean and unusual manifestations especially in children born of immigrant parents.

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