Case study

Neonatal rickets, due to maternal vitamin D deficiency, complicated by convulsion and dilated cardiomyopathy: Case report

Comment [LKYVM1]: Format for case Report
1. Title
2. Structured abstract
3. Introduction
4. Case report (methods and results)
5. Discussion
6. Conclusion
7. Acknowledgements (if applicable)
8. References
9. Tables
10. Figures and captions

Abstract

Deficiency rickets due to maternal hypovitaminosis D ~~is a cause of~~cause hypocalcaemia in infants, which may be complicated by dilated cardiomyopathy (DCM) with myocardial dysfunction. Calcium ~~has a central role~~is central in myocardial contraction coupling, and hypocalcemia decreases myocardial contractility. However, dilated cardiomyopathy (DCM) due to hypocalcemia in infants has been rarely reported. Correction of hypocalcemia was associated with resolution of congestive heart failure and the left ventricular (LV) geometry and systolic function.

We report the case of an infant who presented deficiency rickets due to maternal hypovitaminosis D, complicated by convulsion and dilated cardiomyopathy, with good improvement under treatment with calcium and vitamin D.

Keywords~~-~~: Rickets~~-~~; hypovitaminosis D; hypocalcemia; dilated cardiomyopathy; DCM

Introduction

Deficiency rickets due to maternal hypovitaminosis D ~~is a cause of hypocalcemia in infants which may be complicated by dilated cardiomyopathy (DCM) with reversible myocardial dysfunction after oral treatment with~~causes hypocalcemia in infants, which may be complicated by dilated cardiomyopathy (DCM) with reversible myocardial dysfunction after oral treatment with oral treatment calcium and vitamin D.[1, 2]

We report the case of an infant who presented deficiency rickets due to maternal hypovitaminosis D, complicated by convulsion and dilated cardiomyopathy, with good improvement under treatment with calcium and vitamin D.

Case report

The patient is a 4-month-old infant~~,~~ from consanguineous parents, the third of three siblings. The patient was born vaginally, with good adaptation to extrauterine life, birth weight of 3300g, exclusively breastfed, good psychomotor development, and current vaccination

Comment [LKYVM2]: He or she

according to the national immunization program. Since birth, the patient presented fatigue during feedings with the notion of gaze fixity complicated by respiratory discomfort with convulsion. On admission to our department, the clinical examination found a conscious, toned and reactive infant~~, ;~~his weight was 6 kg, head circumferencewas 42 cm. Height54cm, temperature 37C,and normal blood sugar. The patient is in respiratory distress with signs of heart failure.During the clinical examination, the patient had a convulsive seizur~~e, and he~~andwas put in condition with an intra-rectal valium injection.After the convulsion, we ~~carried out an assessment~~assessed: the brain imaging ~~is~~ was normal;the chest X-ray showed cardiomegaly (Figure 1) with bone demineralization.Echocardiography revealed a dilated cardiomyopathy with an ejection fraction ~~at~~ of30% (Figure 2), and an electrocardiogram showed repolarization disorders in V4 and V6. The complete blood count was normal. The infection profile is normal~~.~~.

T~~t~~he dosage of electrolytes revealed ~~a~~ hypocalcemia at Ca+=46mg/l with a corrected Calcium=62 mg/l~~,~~and normal phosphorus at 62 mg/l. ~~before~~ Before this clinical and paraclinical picture.We thought of rickets by a deficit in Vit D, the corrected calciuria decreased, and the 25 OH Vit Dest decreased ~~of 7 ng/ml, parathormone is increased of~~by seven ng/ml, parathormone increased by 120 pg. Increased alkaline phosphatase of 821U/l. The wrist x-ray showed bony signs of rickets (Figure3). The vitamin D dosage in the mother is less than ~~8~~ eight ng/ml. The diagnosis of deficiency rickets secondary to maternal vitamin D hypovitaminosis was retained. The patient was treated with calcitherapy in combination with the treatment of heart failure. Once the serum calcium normalized, the patient was put on vitamin D3. The evolution was favourable, and marked by the improvement of the size of the heart chambers, the normalization of the left ventricular ejection fraction(Figure 4), and the improvement of the radiological signs (Figure 5).The control PAL dosage showed 256ui/l.

Discussion

Maternal vitamin D deficiency during pregnancy can cause infantile rickets. Prevention involves providing vitamin D during pregnancy and breastfeeding.Vitamin D has ~~for some time been experiencing a spectacular resurgence of interest,~~been experiencing a spectacular resurgence of interest for some time due to its ~~“~~"classic~~”~~ "bone effects~~,~~ but also extra-osseous [3,4]. 80–90% of vitamin D comes from skin biosynthesis under the effect of ultraviolet radiation. Only 10–20% of vitamin D comes from an exogenous source~~,~~ via the

absorption of vitamin D-rich foods. Exogenous intake depends on the type of diet but also on regional habits [5]. Until recently, the minimum satisfactory concentration of vitamin D was defined as that which would prevent the onset of deficiency rickets in children and osteomalacia in adults, i.e., approximately 8 ng/ml (20 nmol/ l) [6]. I~~t was in 2010 that~~n 2010, most international experts agreed to set the limit values for adults. A level between 20 and 30 ng/ml (50 and 75 nmol/ml) is considered an ~~“"~~insufficiency~~”;";~~ a level between 10 and 20 ng/ml (25 and 50 nmol/l) a ~~“"~~deficiency~~”;";~~ and less than or equal to 10 ng/ml (≤ 25 nmol/ml) in vitamin D as the "deficiency" threshold below which the risk of short-term bone pathological consequences is significant. For children, there is no consensus, and it is considered that a minimum serum concentration of 20 ng/ml is necessary [7]. In the newborn, the vitamin status depends entirely on th~~at of the mother~~e mother's. Maternal vitamin D stores can sustain infant requirements for the first 6 weeks of life~~,~~ only if maternal vitamin D status ~~was~~ is sufficient at the end of pregnancy, which is often not the case. A vitamin D deficiency has been well demonstrated in pregnant women at the end of their pregnancy. Studies have shown a relationship between this poor vitamin D status and the frequency of late or even early neonatal hycalcemia accidents [8,9]. In a 2001 study,Bassir et al. found in a population of Iranian pregnant women very low or zero circulating levels of plasma 25(OH)D in 80% of the population studied (57 women). Newborns had low or undetectable levels with biological signs of osteomalacia (raised circulating PTH and alkaline phosphatase), as is the case of our patient.Moreover, the neonatal adaptation of calcium metabolism is disturbed with severe and lasting neonatal hypocalcemia [10].

Hypocalcemia caused by vitamin D deficiency leads to extra-osseous complications, ~~in particular~~particularly dilated cardiomyopathy and congestive heart failure.~~In fact, c~~Calcium ~~has a direct effect on the force of contraction of the myocardium~~directly affects the myocardium's force of contraction by excitation-contraction coupling. Hypocalcemia reduces myocardial contractility, but the incidence of congestive heart failure and cardiomyopathy due to hypocalcemia is very rare[11].Hypocalcemic cardiomyopathy is generally refractory to conventional treatments for heart failure but responds favorably to restoration of normocalcemia. Our patient's case had severe hypocalcemia due to vitamin D deficiency and responded dramatically to calcium and vitamin D correction. Maiya, et al. reported 16 cases of cardiomyopathy due to hypocalcemia in children associated with vitamin D deficiency[12]. Another study found four exclusively breastfed African American infants with congestive

heart failure and dilated cardiomyopathy due to hypocalcemia, whose heart function returned to normal within months of treatment with vitamin D and calcium[13].An Indian study found hypocalcemia in 16% of infants with severe left ventricular dysfunction~~,~~ ;vitamin D deficiency was identified as the main cause of hypocalcemia. These children improved after vitamin D and calcium supplementation[14].

Vitamin D deficiency in developing countries is primarily nutritional, particularly in exclusively breastfed infants whose mothers have low vitamin D stores[15].

Conclusion:

Neonatal and maternal hypovitaminosis D should be considered in the event of neonatal hypocalcemia. Systematic supplementation should be implemented in the third trimester of pregnancy to maintain adequate maternal vitamin ~~status~~ and optimal fetal status.

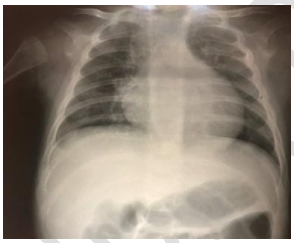

Figure 1~~-~~: Chest~~X~~X-ray

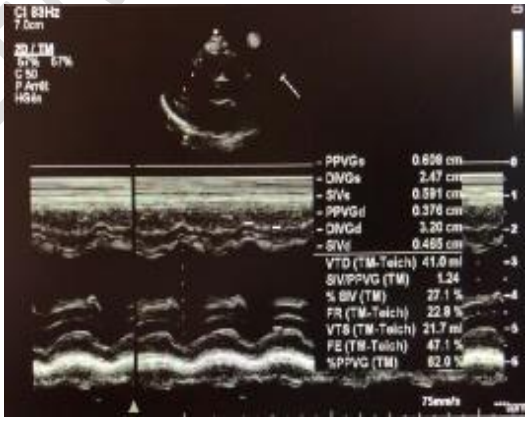

Figure 2~~-~~: Echocardiographybeforetreatment

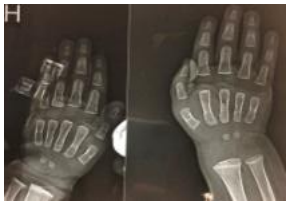

Figure 3 : Radiographybeforetreatment

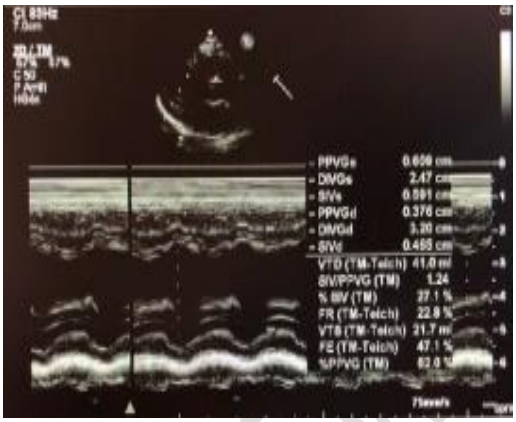

Figure 4 : Echocardiographyaftertreatment

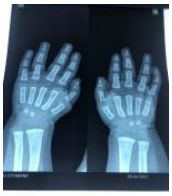

Figure 5 :Radiographyaftertreatment

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