A case of dilated cardiomyopathy due to nutritional vitamin D deficiency rickets

Haşim Olgun, Naci Ceviz, Behzat Özkan
Department of Pediatrics, Atatürk University Faculty of Medicine, Erzurum, Turkey

SUMMARY: Olgun H, Ceviz N, Özkan B. A case of dilated cardiomyopathy due to nutritional vitamin D deficiency rickets. Turk J Pediatr 2003; 45: 152-154.

Vitamin D deficiency rickets was detected as the cause in a nine-month-old girl with dilated cardiomyopathy and signs of congestive heart failure. The patient responded to calcium and vitamin D supplementation promptly and left ventricular systolic functions normalized at the 3rd month of treatment. Nutritional rickets must be remembered in etiological assessment of dilated cardiomyopathy among infants living in regions in which nutritional rickets is still common.

Rickets due to vitamin D deficiency is an important and common problem in developing countries[1,2]. Although it has been reported that asymptomatic left ventricular dysfunction may develop in patients with vitamin D deficiency rickets (VDDR) and that it improves with treatment[2], dilated cardiomyopathy and congestive heart failure are rare[3-5].

In this paper we report a case of dilated cardiomyopathy in which severe VDDR was diagnosed during the etiological assessment and which improved with subsequent vitamin D supplementation.

Case Report

The patient was a nine-month-old girl who had been treated for the diagnosis of bronchopneumonia in the state hospital. She was referred to our clinic because of the cardiomegaly on chest X-ray that has been obtained at the 20th day of treatment.

Her body weight was 6,200 g (<3rd p) and length 67 cm (10th-20th p). Physical examination revealed the presence of signs of rickets (rachitic rosary, enlarged wrist, enlarged anterior fontanel) in addition to signs of congestive heart failure. Chest X-ray showed apparent cardiomealy. In electrocardiography cardiac rhythm was normal sinus rhythm, QRS axis was measured as 0°, PR interval 0.12 seconds and QTc 0.35. Echocardiography study revealed an enlarged left ventricle and hypokinetic left ventricular wall motion. Left ventricular end diastolic diameter (LVDd) was measured as 35.2 mm, left ventricular end systolic diameter (LVDs) as 28.1 mm, ejection fraction (EF) as 49%, shortening fraction (SF) as 20% and distance between mitral E point and septum as 17.1 mm (Fig. 1). Wrist X-ray revealed signs concordant with severe rickets (Fig. 2). Total serum Ca^{++} level was detected as 6.8 mg/dl (8.8-10.8 mg/dl), phosphorus as

(a) (b)

Fig. 1. Echocardiographic recording of heart on two dimensional apical four-chamber view (a) depicts the enlarged left ventricle, and decreased left ventricular contractility is apparent on M-mode recording (b).

(a)

(b)

Fig. 2. Left wrist X-ray. Panel A shows the pre-treatment changes due to rickets, and two healing fractures on ulna (arrows). Panel B includes the 15th day radiography of left wrist. Calcification zone is

2 mg/dl, alkaline phosphatase as 1990 IU/L, intact-parathyroid hormone as 506.7 pg/ml (12-72 pg/ml) and 25-hidroxy vitamin D as <5 ng/ml (10-30 ng/ml). Multiple fractures on left ulna, fibula and radius were detected on bone survey graphies.

Liver and renal functions, blood gas values, serum ammonia level and qualitative measurement of blood and urine amino acids were normal. Serological tests against hepatitis A, B, C and Epstein-Barr viruses were negative. Serological tests against other viruses could not be performed. Total serum carnitine level was low normal (15 μmol/L).

The patient was diagnosed as having dilated cardiomyopathy due to severe VDDR. Treatment included anti-congestive drugs (digitalis, diuretics and ACE inhibitors) in addition to vitamin D and calcium supplementation. Signs related to heart failure disappeared in a few days and the wrist X-ray on 15th day of vitamin D supplementation showed calcification zone on the distal end of radius and ulna (Fig. 2). Total serum Ca^{++} level was measured as 9.1 mg/dl and 25-hidroxy vitamin D level as 20 ng/ml.

The patient was visited at the 3rd month of treatment and the echocardiographic study revealed LVDd as 30.2 mm, LVDs 22.1 mm, EF 60.8%, SF 26.8% SF and the distance between mitral E point and septum as 9.3 mm.

Discussion

Calcium ions have a key role in the excitation as well as the contraction of the cardiac muscle fibers[6], and reduction in serum calcium level may affect ventricular contraction[2]. Although hypocalcemia is a common problem in children, congestive heart failure due to hypocalcemia is a relatively uncommon but reversible problem[7]. Rickets is a metabolic disorder of the bone that develops due to insufficient mineralization of the bone tissue and presents its more striking findings on the skeletal system[8]. Hypocalcemia is present in most of patients with VDDR and may be severe causing convulsions. However, congestive heart failure and cardiomyopathy are rare, having been reported as case reports[3-5]. The only prospective study investigating cardiac functions in patients with rickets belongs to Uysal et al.[2]. Although none of their patients had cardiac symptoms, they reported some echocardiographic changes indicating that the heart may have been affected in rickets.

In patients with cardiomyopathy and rickets, long-standing hypocalcemia has been considered the leading cause of cardiomyopathy. Nevert-heless, the exact mechanism of cardiomyopathy in these cases has not been completely understood[9]. Dursun et al.[9] reported a decreased serum carnitine level and an

increased urinary carnitine excretion in patients with VDDR compared to controls. Although the levels of carnitine excreted in the urine had decreased significantly after treatment, increase in serum carnitine level was not found to be significant. The authors concluded that the carnitine metabolism is disturbed in nutritional rickets, but they failed to establish a "cause and result" relationship between cardiomyopathy and disturbed carnitine metabolism in nutritional rickets since eight of their patients evaluated with echocardiography did not have any cardiomyopathic change. We could not measure the urinary carnitine excretion in our case; however, the serum carnitine level was normal.

One of the most common causes of dilated cardiomyopathy in infancy is myocarditis. Its definite diagnosis can be made only by endomyocardial biopsy[10]. Although that may have been the case in our patient, presence of severe VDDR and prompt response to vitamin D treatment suggest that in our case the reason for dilated cardiomyopathy was rickets.

In conclusion, nutritional rickets must be remembered in the etiological assessment of dilated cardiomyopathy among infants living in regions in which nutritional rickets is still common. These patients promptly respond to calcium and vitamin D supplementation.

REFERENCES

1. Özkan B, Büyükavcı M, Aksoy H, Tan H, Akdağ R. Erzurum'da çocuklarda nütrisyonel rikets sıklığı. Çocuk Sağlığı ve Hastalıkları Dergisi 1999; 42: 389-396.
2. Uysal S, Kalaycı AG, Baysal K. Cardiac functions in children with vitamin D deficiency rickets. Pediatr Cardiol 1999; 20: 283-286.
3. Gilor A, Groneck P, Kaiser J, Schmiztz-Stolbrink A. Congestive heart failure in rickets caused by vitamin D deficiency (Abstract). Monatsschr Kinderheilkd 1989; 137: 108-110.
4. Yaseen H, Maragnes P, Gandon-Laloum S, et al. A severe from of vitamin D deficiency with hypocalcemic cardiomyopathy (Abstract). Pediatrie 1993; 48: 547-549.
5. Abdullah M, Bigras JL, McCrindle BW, Mustafa A. Dilated cardiomyopathy as a first sign of nutritional vitamin D deficiency rickets in infancy. Can J Cardiol 1999; 15: 699-701.
6. Connor TB, Rosen BL, Blaustein MP, Applefeld MM., Doyle LA. Hypocalcemia precipitating congestive heart failure. N Engl J Med 1982; 307: 869-972.
7. Levine SN, Rheams CN. Hypocalcemic heart failure. Am J Med 1985; 78: 1033-1035.
8. Root AW, Diamond FB, Mimouni FB. Parathyroid and vitamin D-reated disorders in children and adolescents. In: Sperling MA (ed). Pediatric Endocrinology (1st ed). Philadelphia: WB Saunders; 1996: 427-509.
9. Dursun A, Aliefendioğlu D, Özkan B, Coşkun T. Carnitinuria in rickets due to vitamin D deficiency. Turk J Pediatr 2000; 42: 278-280.