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CASE REPORT

Dual etiology of dilated cardiomyopathy: The synergistic role of vitamin D deficiency

Sérgio Laranjo*, **Conceição Trigo**, **Fátima F. Pinto**

Serviço de Cardiologia Pediátrica, Hospital de Santa Marta, CHLC, EPE, Lisboa, Portugal

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KEYWORDS
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Abstract Dilated cardiomyopathy is the commonest form of cardiomyopathy in pediatric patients. Various causal factors have been identified, including ionic imbalance. Calcium ions play an essential role in regulating myocardial contractile function, and the harmful role of hypocalcemia as a coadjuvant or even precipitating factor of worsening heart failure has been described in rare case reports. Multiple causative factors may occasionally be present. We describe the first case, to our knowledge, of dilated cardiomyopathy in an infant with severe hypocalcemia and viral myocarditis.

PALAVRAS-CHAVE
Cardiomiopatia dilatada;
Hipocalcemia;
Hipovitaminose D

Hipovitaminose D na cardiomiopatia dilatada: efeito sinergístico numa dupla etiologia

Resumo A cardiomiopatia dilatada é a forma mais comum de cardiomiopatia em idades pediátricas. Várias causas podem estar envolvidas, incluindo desiquilíbrios iónicos. O cálcio desempenha um papel essencial na regulação da função contrátil do miocárdio. O efeito nocivo da hipocalcemia como coadjuvante, ou mesmo, fator precipitante do agravamento da insuficiência cardíaca tem sido episodicamente relatado. Raramente, múltiplas causas estão presentes no mesmo doente; descrevemos o primeiro caso, tanto quanto é do nosso conhecimento, de cardiomiopatia dilatada numa criança com hipocalcémia grave associada a miocardite viral.

Introduction

Dilated cardiomyopathy (DCM), the commonest form of cardiomyopathy, has an estimated incidence of 1.13 cases per 100 000 children. It may be idiopathic, the product of familial genetic mutations, or the outcome of aggression to the

* Corresponding author.
E-mail address: sergiolaranjo@gmail.com (S. Laranjo).

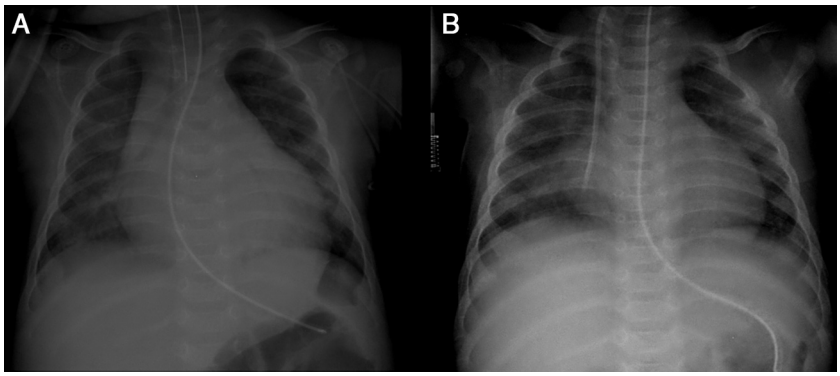

Figure 1 (A) Chest X-ray at admission showing cardiomegaly (cardiothoracic ratio 80%); (B) chest X-ray on day 25, showing reversal of cardiomegaly with treatment (cardiothoracic ratio 52%).

myocardium caused by various factors, including metabolic or endocrine disturbances, electrolyte imbalance, inflammation, infection, immune system impairment and toxins. The primary etiology is diagnosed in fewer than half of these children, but this significantly improves their outcome. In isolated case reports hypocalcemia is a rare and reversible cause of heart failure or DCM.[1–5] Multiple causative factors may occasionally be present. We describe the first case, to our knowledge, of DCM in an infant with severe hypocalcemia and viral myocarditis.

Case report

We report the case of a three-month-old African boy, born in Europe, presenting with poor feeding, respiratory distress and irritability. These symptoms had progressively worsened over a period of three days. The patient was born at 40 weeks of gestational age with a birth weight of 3380 g. He had been exclusively breast-fed, with no vitamin D supplementation. Medical and family histories were irrelevant except for a maternal upper respiratory infection seven days before admission.

On admission he was apyretic, with pulse 190 bpm, respiratory rate 70–80 breaths per minute and oxygen saturation 95% in room air. His body weight was 6 kg (50th percentile). Physical examination revealed an infant of normal appearance but irritable and in respiratory distress. He was tachycardic, with an S3/S4 gallop rhythm. There were moist rales in the lung fields and chest wall retraction. The abdominal exam revealed hepatomegaly. The extremities displayed cyanosis and poor perfusion, with no edema. The chest X-ray showed cardiomegaly (cardiothoracic ratio 0.8) with pulmonary congestion (Figure 1A). The echocardiogram revealed a markedly enlarged left ventricular (LV) cavity with hypokinetic ventricular wall motion. LV end-diastolic diameter was 45 mm, LV end-systolic diameter was 40 mm, and fractional shortening (FS) was 5%, with no structural abnormalities (Figure 2). Electrocardiography showed sinus tachycardia (heart rate 190 bpm), LV hypertrophy and normal QTc.

Due to the extent of congestive heart failure, with poor systolic function, the patient was admitted to the cardiac intensive care unit, sedated and intubated. Arterial blood gas analysis revealed pH 7.27, pCO_2 45.7 mmHg and HCO_3 19 mmol/l under 50% FiO_2 mechanical ventilation. Laboratory tests showed Ca^{2++} 0.76, lactate 11, total serum calcium 5.6 mg/dl (reference value: 8.8–10.8 mg/dl), inorganic magnesium 1.4 mg/dl (reference value: 1.8–2.6 mg/dl) and elevated alkaline phosphatase (422 IU/l, reference value: 82–383 IU/l), creatine kinase 497 IU/l (reference value <171 IU/l) and troponin I 0.43 ng/ml (reference value <0.06 ng/ml). Other biochemical parameters and electrolytes studied were within normal ranges. Enterovirus antigen was documented in feces.

Intravenous hyperimmune gamma globulin was given at admission and the patient was placed on milrinone and dopamine (titrated to a maximum dose of 0.48 μg/kg/min and 4 μg/kg/min, respectively) to improve systolic function, and furosemide for congestive heart failure. Two

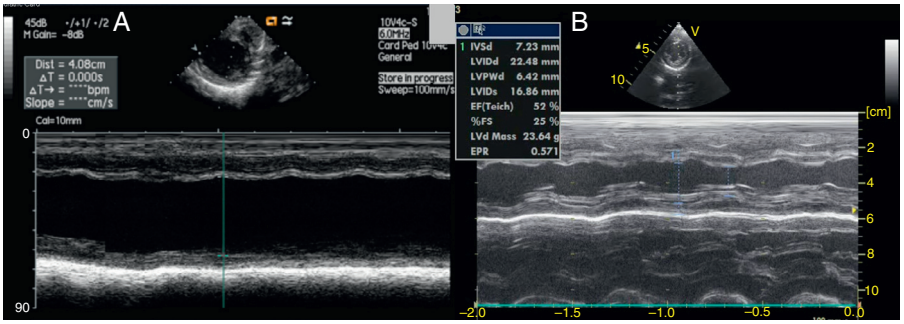

Figure 2 M-mode parasternal short-axis echocardiographic views: (A) enlarged left ventricle with hypokinesia on admission; (B) after 25 days of treatment, left ventricular diameters and contractile function are completely normal.