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Hypocalcemic Heart Failure: A Reversible Form of Heart Muscle Disease

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Summary: This paper reports the case of a 53-year-old woman with hypocalcemia-induced reversible cardiomyopathy. Laboratory tests showed hypocalcemia caused by idiopathic hypoparathyroidism. Her left ventricular dysfunction persisted for a long period even after normalization of the serum calcium level. Observations suggest that physicians should be aware that hypocalcemia can be a reversible cause of cardiomyopathy and congestive heart failure.

Key words: hypoparathyroidism, heart failure, calcium

Introduction

The central role of calcium in the sequence of myocardial excitation-contraction coupling and myocardial relaxation is well established; however, observations of congestive heart failure in hypocalcemic patients are rather rare.[1] Here, we report a patient with hypocalcemia-induced reversible cardiomyopathy and no underlying myocardial disease, in whom left ventricular (LV) dysfunction persisted for a long period after normalization of the serum calcium level.

Case Report

A 53-year-old woman was admitted to our hospital in January 1995 with increasing shortness of breath and orthopnea. She had a history of muscle cramps for more than 10 years but never had a medical examination. There were no symptoms of hyperthyroidism or hypothyroidism. Her initial blood pressure was 110/88 mmHg, and the pulse was 114 beats/min. Her jugular veins were distended. Third and fourth heart sound gallop was audible. Mild hepatomegaly and leg edema were observed. Both Chvostek's and Trousseau's signs were present.

Admission laboratory data included normal hemoglobin, serum sodium, potassium, chloride, bicarbonate, glucose, blood urea nitrogen, creatinine, triiodothyronine, and thyroxine. Serum albumin level was 3.1 g/dl. Serum glutamic oxaloacetic transaminase and glutamic pyruvic transaminase activities were elevated to 126 and 122 IU/l, respectively, due to congestion of the liver. The total serum calcium level was 3.1 mg/dl, phosphorus level 10.2 mg/dl, magnesium level 1.4 mg/dl, and intact parathyroid hormone level < 3.0 pg/ml (normal range 15–50 pg/ml), a value indicating parathyroid hormone deficiency.

Chest radiography demonstrated cardiac enlargement (cardiothoracic ratio of 0.78) with pulmonary congestion and pleural effusion. Electrocardiography showed sinus tachycardia with a rate of 100 beats/min. The corrected Q-T interval was 0.59 s, with T-wave inversions in leads V_1 to V_6. M-mode and two-dimensional echocardiography revealed enlargement (end-diastolic diameter 67.8 mm, end-systolic diameter 59.0 mm) and severe generalized hypokinesis of the left ventricle with an ejection fraction of 23% (Fig. 1A). No pericardial effusions were identified. Color Doppler echocardiography revealed severe mitral regurgitation.

The patient was diagnosed as having idiopathic hypoparathyroidism, and treatment was initiated with calcium gluconate, alfacalcidol, digoxin, furosemide, spironolactone, and catecholamines. Eighteen days after admission, the serum calcium level rose to 8.3 mg/dl and all symptoms of heart failure had resolved. The corrected Q-T interval returned to 0.44 s, but LV ejection fraction still remained < 30%. Cardiac catheterization was performed; coronary arteriogram was normal and the right ventricular biopsy samples showed no abnormal findings.

The patient was discharged 1 month after admission with a regimen of alfacalcidol 2 μg/day and spironolactone 50 mg/day. Three months after discharge, her serum calcium levels remained normal; no symptoms of heart failure were present but her cardiac function was still impaired with an ejection fraction of 47% on echocardiography (Fig. 1B). One year after discharge, cardiothoracic ratio decreased to 0.48, but LV ejection fraction remained reduced (52%). Results of repeated electrocardiography were normal without prolongation of the Q-T interval. Two years after discharge, her cardiac function was almost completely normal; LV ejection fraction increased to 64%, associated with reduced LV dimension (end-

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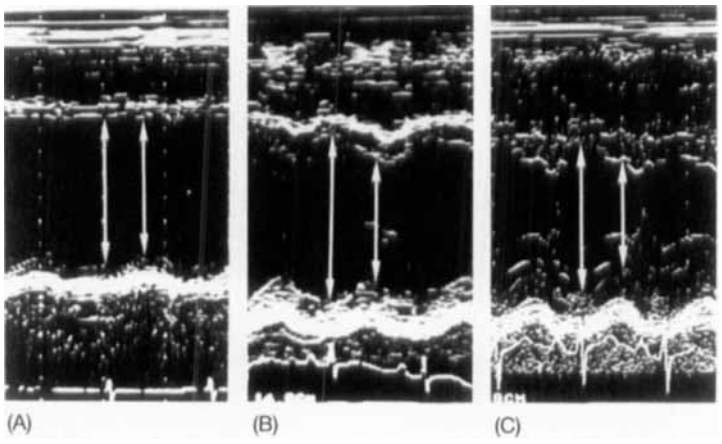

FIG. 1 Echocardiography on admission (A), 3 months after discharge (B), and 2 years after discharge (C). Arrows indicate end-diastolic and end-systolic diameters.

diastolic diameter 54.1 mm, end-systolic diameter 35.0 mm) (Fig. 1C). Color Doppler echocardiography revealed trivial mitral regurgitation.

Discussion

The clinical description of our patient is similar to that of patients previously reported to have reversible congestive heart failure associated with hypoparathyroidism,[2-8] although it took almost 2 years for complete improvement of LV function in this case. The delayed but excellent improvement in the cardiac state of our patient that occurred with treatment of hypoparathyroidism highlights the importance of this endocrinopathy in the pathogenesis of cardiomyopathy and congestive heart failure.

An increase in the intracellular concentration of calcium is required to initiate excitation-contraction coupling. Contraction of cardiac muscle requires availability of calcium from extracellular fluid, as well as release from the sarcoplasmic reticulum.[9] Although a decrease in the concentration of calcium in the plasma reduces cardiac contractility, heart failure has rarely been observed to be a complication of hypocalcemia.[10] This is in all likelihood due to the development of symptomatic neuromuscular irritability, causing the majority of hypocalcemic patients to seek early medical attention. Our patient had symptoms of muscle cramps and spontaneous muscular contractions for more than 10 years, but without medical intervention. It is likely that her lack of attention to symptoms of hypoparathyroidism allowed her hypocalcemia to go unrecognized for many years, ultimately precipitating decompensation of her cardiovascular status and also prolongation of the improvement of cardiac function after the normalization of serum calcium levels.

The patient was diagnosed as having idiopathic hypoparathyroidism. No other cardiac diseases including viral myocarditis and coronary or hypertensive heart disease were identified. Hypoparathyroidism occasionally accompanies thyroid disease, which could also cause reversible or irreversible cardiomyopathy.[11] However, our patient had no history of thyroid disease, and her thyroid function was normal. In addition to hypocalcemia, the mechanism of the development of cardiomyopathy in hypoparathyroidism might include hypomagnesium,[2] but serum magnesium levels were only slightly below the lower normal limit in our patient.

Hypocalcemia as a cause of cardiac decompensation is often overlooked in the ordinary clinical setting. As reported here, cardiac contractility could be seriously impaired by prolonged hypocalcemia. Physicians should be aware that hypocalcemia can be a reversible cause of cardiomyopathy and congestive heart failure.

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