

A rare cause of reversible dilated cardiomyopathy: hypocalcemia

Geri dönüşümlü dilate kardiyomyopatinin nadir bir nedeni: Hipokalsemi

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Hypocalcemia is a rare cause of reversible heart failure. We present a 27-year-old man who had severe heart failure unresponsive to medical therapy. He had vitamin D-resistant rickets for which he had received replacement therapy with vitamin D and calcium until age 20, but he discontinued treatment for the past seven years. Severe hypocalcemia was detected. Echocardiography showed left ventricular dilatation, global hypokinesia (ejection fraction 25%), and mitral and tricuspid regurgitation of grades 3 and 2, respectively. After calcium and vitamin D supplementation, his symptoms showed rapid improvement. At nine months, myocardial dysfunction improved fully. Hypocalcemia should be considered among the causes of heart failure unresponsive to medical treatment.

Key words: Cardiomyopathy, dilated/etiology; heart failure/etiology; hypocalcemia/complications; rickets.

Hipokalsemi geri dönüşümlü kalp yetersizliğinin nadir bir nedenidir. Bu yazıda, tedaviye dirençli ileri derecede kalp yetersizliği olan 27 yaşındaki bir hasta sunuldu. D vitaminine dirençli raşitizm nedeniyle 20 yaşına kadar D vitamini ve kalsiyum replasman tedavisi gören hastanın son yedi yıldır kalsiyum ve D vitamini tedavisini bıraktığı öğrenildi. Hastada ileri derecede hipokalsemi vardı. Ekokardiyografide sol ventrikül genişlemesi, global hipokinezi (ejeksiyon fraksiyonu %25), üçüncü derece mitral yetersizliği ve ikinci derece triküspit yetersizliği saptandı. Kalsiyum ve D vitamini replasmanı sonrası hastanın semptomları hızla geriledi. Dokuzuncu ayda miyokart disfonksiyonu tamamen düzeldi. Tedaviye dirençli kalp yetersizliğinin nedenleri arasında hipokalsemi de düşünülmelidir.

Anahtar sözcükler: Kardiyomyopati, dilate/etyoloji; kalp yetersizliği/etyoloji; hipokalsemi/komplikasyon; raşitizm.

Dilated cardiomyopathy is a condition characterized by dilatation of single or both ventricles and systolic dysfunction. Hypocalcemia is a very rare cause of reversible heart failure and dilated cardiomyopathy.^[1-7] Dilated cardiomyopathy due to hypocalcemia in children with rickets has been reported in many cases; however the incidence of the condition is lower in adults. We presented a case with dilated cardiomyopathy due to hypocalcemia unresponsive to medical therapy for heart failure.

CASE REPORT

A 27-year-old man who had dyspnea on effort for nearly 6 months was given treatment for heart failure (furosemide 80 mg/day, digoxin 0.25 mg/day, spironolacton 25 mg/day, ramipril 2.5 mg/day) following fatigue, dizziness and giddiness with increased complaints last week. The patient whose symptoms did not recover visited our clinic. Medical history of the patient revealed that he used vitamin D due to rickets until he was 20

years old but he discontinued treatment. The patient who visited our clinic with the symptoms of orthopnea, tachypnea and tachycardia (pulse rate 112/min) had blood pressure of 85/55 mmHg. Physical examination revealed crepitan rales in the mid and upper zones of both lungs. In addition, cardiac examination revealed normal S₁ and S₂ and S₃ (+). A 3-4/6 apical pansystolic murmur which radiates into the axilla was also heard. Pretibial edema (1+) was detected in both legs. Carpopedal spasm in hands and Chvostek's sign in jaw were observed. Electrocardiography also showed sinus rhythm with normal findings. A negative T wave and prolongation of the QT interval (QTc 0.58 sec) was found in lead I, lead III, aVF, V4-V6 derivations (Figure 1). In addition, chest x-ray revealed cardiomegaly and a stasis-like view. Electrocardiography showed left ventricular dilatation (diastolic diameter of 7.29 cm, systolic diameter of 6.39 cm), global hypokinesia (EF of %20-25) (Figure 2a), and grade 3 mitral regurgitation (jet length of mitral regurgitation measured by color

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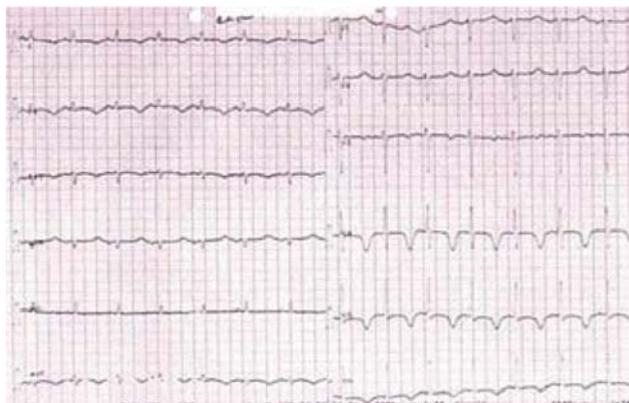


Figure 1. Electrocardiography showing sinus rhythm at baseline, a negative T wave and prolongation of the QT interval in lead I, lead III, aVF, V4-V6 (0.58 sec)

Doppler was 3.5 cm, jet area was 6.7 cm²) and grade 2 tricuspid regurgitation (Figure 2b). Pulmonary artery pressure was 50 mmHg. Laboratory test results also showed Ca 2.9 mg/dL (8.5-10.5 mg/dL), ionized Ca 0.4 mmol/L (0.85-1.05 mmol/L), albumin 3.5 gr/dL, Na 139 mmol/L, (135-145 mmol/L), K 3.5 mmol/L (3.5-5.5 mmol/L), P 4.4 mg/dL (3-4.5 mg/dL), ALP 367 U/l (25-100 U/l), PHT 109 pg/dL (15-67 pg/dL), 25 (OH) D vit 12.7 ng/dL (10-40 ng/dL). These findings were found to be consistent with the diagnosis of vitamin D-resistant rickets type I.

The patient was given dopamine and dobutamine infusion, and treatment with diuretics and intravenous calcium and oral vitamin D₃ was instituted to reach serum calcium level of 7.5 mg/dL over 24 hours. After inotropic and calcium treatment, his symptoms showed rapid improvement. Oral calcium and vitamin D₃ treatment was maintained following intravenous calcium. Inotropic treatment was discontinued on 3. days and an ACE inhibitor was given. In addition, low dose of beta-

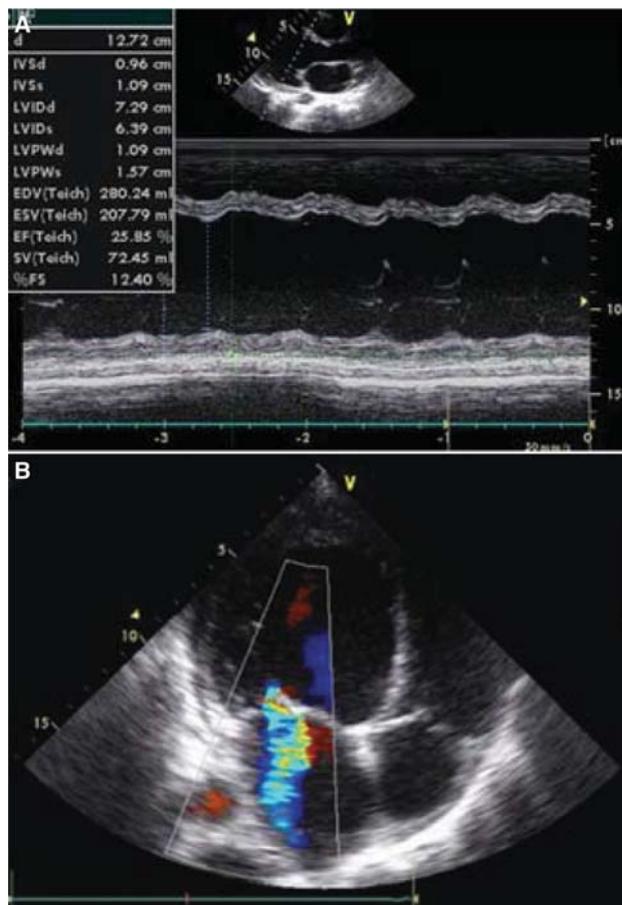


Figure 2. Baseline echocardiography showing (A) diameter of dilated left ventricle in the parasternal long axis (end-diastolic diameter 7.29 cm, end-systolic diameter 6.39 cm, ejection fraction of %25) and (B) grad 3 mitral regurgitation in the four chamber view performed by color Doppler.

blocker (6.25 mgr carvedilol) was added to the treatment and the patient was discharged from the hospital. Effort capacity was found to be increased gradually du-

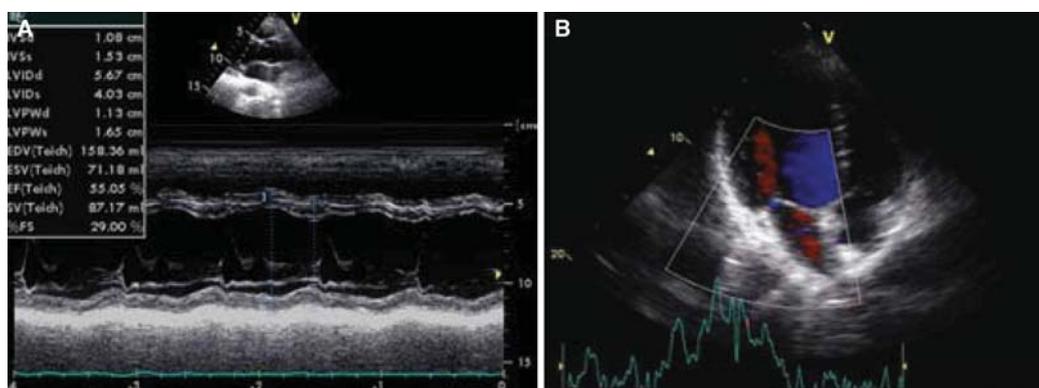


Figure 3. Echocardiography repeated at 9. months following treatment showing (A) end-diastolic diameter of 5.67 cm, end-systolic diameter of 4.03 cm and ejection fraction of 55% and (B) improved mitral regurgitation in the four chamber view performed by color Doppler.

ring follow-up. Repeated echocardiography also demonstrated reduced diameters of the left ventricle and improved EF. At nine months, normalized diameters of the left ventricle and EF normalized. Mitral regurgitation also recovered completely (Figure 3). The treatment with calcium, vitamin D₃ and ACE inhibitor was maintained.

DISCUSSION

Ionized calcium plays a key role in the contraction of myocardium; therefore hypocalcemia impairs the contraction function of the heart.^[8]

Reasons of hypocalcemia should be carefully investigated in patients with heart failure. There may be patients who do not receive treatment for many years despite the diagnosis of rickets, as in our case, more common causes of hypocalcemia such as total thyroidectomy and end-stage renal failure should also be considered. Cardiomyopathy due to hypocalcemia has been reported rarely. However, it can be suggested that heart failure unresponsive to medical treatment may also lead to hypocalcemia within the consideration of loop-diuretic treatment which is benefited mostly. In particular, susceptible patients for hypocalcemia (who performed total thyroidectomy and those with end-stage renal failure) should be followed closely.^[9]

Patients whose symptoms of heart failure did not regress despite satisfactory medical treatment and those with QT prolongation according to ECG results should be evaluated in respect to hypocalcemia.

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