A Rare Cause Of Dilated Cardiomyopathy: Hypocalcemia

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Summary

A 35-year-old female patient suffering from dyspnea was hospitalized with the diagnosis of severe heart failure which was refractory to digitalis and diuretics. The diagnosis was confirmed by radiography and echocardiography showing ventricular enlargement, global hypokinesia and mitral regurgitation. Clinical status was severe, blood pressure (BP) 80/50 mm Hg. There were Schovestec and Trouseaou signs without tetani. BP could not be increased despite using higher doses of dopamine. Serum calcium was 3.5 mg/dL, phosphatemia 5.7 mg/dL and level of parathormone (PTH) 10 pg/ml. After serum calcium had been intravenously raised, she had progressive hemodynamic improvement. She was put on oral calcium and vitamin D3 as well as digoxin, diuretic. Echocardiography revealed improved motion of ventricular walls and increase in ejection fraction (from 32% to 50%). As a result; hypocalcemia should be kept in mind for differential diagnosis of dilated cardiomyopathy (DCM), especially in the case of refractory DCM which develops after thyroidectomy.

Key Words:

Hypocalcemia, heart failure, dilated cardiomyopathy.

Introduction

Dilated cardiomyopathy (DCM) is a syndrome characterized by cardiac enlargement and impaired systolic function of one or both ventricles. Hypocalcemia is a rare and potentially reversible cause of secondary DCM[1]. PTH has a direct effect on the heart, resulting in an increased beating rate of isolated heart cells and a positive inotropic action. These effects are mediated by PTH, leading to increased entry of calcium into myocardial cells[2].

In this case report we studied a patient with refractory heart failure due to hypocalcemia secondary to hypoparathyroidism developed after subtotal thyroidectomy.

Case

A 35-year-old female patient suffering from dyspnea, palpitation and fatigue was admitted to cardiology clinic. Her complaints were getting worse during last ten days though she was using digitalis and diuretic. In 1990 she was submitted to subtotal thyroidectomy. She didn't have the history of acute rheumatic fever or consumption of alcohol. Clinical status was severe and she had orthopnea. BP and heart rate were respectively 80/50 mm Hg and 112 beats/minute, dysrhythmic, urine output <30ml/24 hour. Body temperature was 37°C. The neck veins were distended. Rales were auscultated up to the middle fields of both lungs. There were low first heart sound, audible protodiastolic gallop. On the apical region, 2/6 pansystolic murmur that radiated to axilla was heard. The liver was tender and enlarged 5 cm. Examination

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also revealed pretibial edema and bilateral costa-vertebral tenderness. Urine analysis showed pyuria. Erythrocyte sedimentation rate and white blood cell count with polymorpho nuclear cell dominance were respectively 78 mm/h and 10900/mm^3. Serum calcium was 3.5 mg/dL with calciurea of 20 mg/day, phosphatemia was 5.7 mg/dL and phosphaturia of 550 mg/day. The plasma level of parathormone was 10 pg/ml. Measurement of serum digoxin levels revealed the concentration of 1.7 ng/ml. Non-paroxysmal junctional tachycardia was recognized by initial electrocardiogram. The chest radiograph showed cardiomegaly, bilateral enlarged hilar shadows, congestion of lower and middle zones of both lungs. In blood and throat cultures patogen microorganism couldn't be isolated and the result of urine culture was interpreted as contamination. Echocardiographic study defined biventricular dilation, second degree mitral regurgitation, low ejection fraction and general hypokinesia (Figure-1). The patient was hospitalized

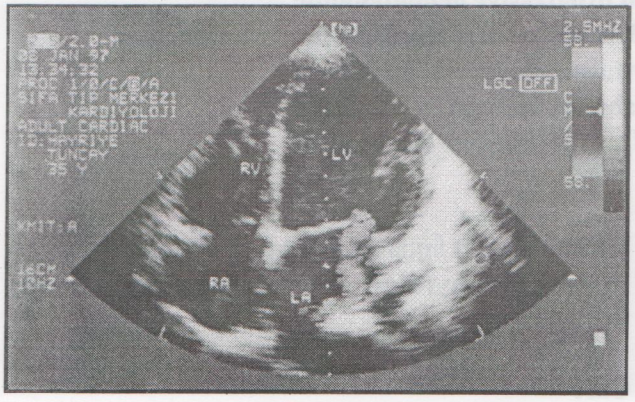

Figure-1.
View of apical 4-chamber echocardiography of patient.
It shows both ventricular and atrial enlargement.

with the diagnosis of congestive heart failure and uriner tract infection. She was first treated with dopamine, furosemide and oral amoxicillin+clavulanate. BP and urine output couldn't be increased despite using higher doses of dopamine. After serum calcium had been rised intravenously, she had progressive hemodynamic improvement. In the following three days, her signs dramatically disappeared and the rhythm came back to sinus. She was put on oral calcium and vitamin in addition to digoxin, diuretic. On the 10th day of hospitalization, she was discharged with serum calcium 8.8 mg/dL, phosphate 3.2 mg/dL and normal biochemical blood and urine values. Echocardiographical control revealed improvement of motion of ventricular walls and increase in ejection fraction (from 32% to 50%). She is now on follow up program by cardiology and endocrinology departments.

Discussion

Myocardial contractile function requires calcium. Hypocalcemia secondary to hypoparathyroidism may cause a DCM and heart failure. Additionally PTH has a positive inotropic action on myocardial cells[1,2].

Musse[3] reported a 39-year-old female patient with refractory heart failure, which is caused by hypoparathyroidism developed following subtotal thyroidectomy. Seven months after surgery, she had acute pulmonary edema confirmed by both telecardiogram and echocardiogram. The calcium was 5.0 mg/dL with calciuria of 28 mg/day, phosphatemia was 4.8 mg/dL and phosphaturia of 214 mg/day. She was first treated with digitalis, diuretic, vasodilator drugs and oral calcium. She had progressive hemodynamic improvement when higher doses of calcium were given with D3 vitamin. The most significant result of this treatment was reduction of the heart size that come back to normal.

Weise studied a 48-year-old woman developed a hypocalcaemic DCM secondary to hypoparathyroidism. She had signs of severe cardiac failure, but no tetany. She was put on calcium and vitamin D3 which raised calcium concentration. The cardiac status improved, as did the radiological and echocardiographic findings, without the patient having received any diuretics, digitalis or after load lowering drugs.

In an other case report in the literature[5]; a 41-year-old woman presented with mild congestive heart failure, initially controlled with a small dose of diuretic, subsequently there was a marked deterioration in her condition with severe cardiac failure resistant to treatment. She had a hyperphophataemic variety of osteomalacia. Hypocalcemia was recognized as cause of heart failure. Cardiac failure improved promptly when hypocalcemia was corrected.

Palazzuoli reported the clinical history of 56-year-old man with DCM due to undiagnosed hypocalcemia. Heart dilation and hypokinesia were assessed by echocardiography. Cardiac failure was refractory to

digitalis preparations and to diuretics. The restoration of serum calcium to normal levels obtained with adequate therapy dramatically improved the motion of ventricular walls and decreased the size of the left ventricle[6].

In our case, hypocalcemia that is secondary to hypoparathyroidism caused heart failure. There was no adequate sign supporting rheumatic heart disease or infective endocarditis. Adding calcium and vitamin D3 to inotropic and diuretic therapy improved clinical status of the patient dramatically. Improvement of left ventricular function was also defined by echocardiography. At the present time patient is being treated with digitalis, diuretic, oral calcium and vitamin D3. She is well on her fourth month of follow up program.

As a result; hypocalcemia should be kept in mind for differential diagnosis of DCM, especially in the case of refractory DCM which develops after thyroidectomy.

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