

Case Report

Long QT interval revealing severe hypocalcemic dilated cardiomyopathy : A case report

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Abstract:

Hypocalcemia is a rare cause of dilated cardiomyopathy, but should be suspected in the presence of an obvious long QT interval on the surface ECG; we report a case of 46 years-old-woman, with surgical history of thyroidectomy 6 years ago, admitted in our cardiology department for management of congestive heart failure secondary to dilated cardiomyopathy, her surface ECG showed sinus rhythm with long QT interval, his blood tests showed severe hypocalcemia and low serum concentration of parathyroid hormone (PTH), cardiac magnetic resonance imaging was in favor of non ischemic dilated cardiomyopathy.

Concomitant with conventional heart failure treatment, our patient had received parenteral calcium supplementation, vitamin D, levothyrox; after four weeks, heart failure symptoms were relieved, the interval QT has shortened, but persistent severe left ventricular systolic dysfunction.

Hypoparathyroidism is frequent after thyroidectomy, and could be responsible of severe hypocalcemia which in turn may induce irreversible dilated cardiomyopathy.

Keywords: Hypocalcaemia, dilated cardiomyopathy, thyroidectomy, hypoparathyroidism.

Introduction

Severe hypocalcemia may be caused by hypoparathyroidism which may be secondary to thyroidectomy, several complications related to hypocalcemia have been reported such as osteoporosis, cataract, seizures, and congestive heart failure but this latter is rare and may be irreversible, the diagnosis of hypocalcemic dilated cardiomyopathy may be suspected in the presence of a long QT interval on the surface ECG and confirmed with the blood tests

Case report

A 46 years-old woman, with a past history of thyroidectomy in 2018 for multinodular goiter under Levothyrox treatment; her physical cardiac examination and left ventricular function were normal before surgical intervention: she was admitted in cardiology department in the January 28th 2024, for management of congestive heart failure.

Three weeks before her admission, she had experienced dyspnea, intermittent chest pain and abdominal pain, a general practitioner had recommended thoracoabdominal computed tomography scan (CT scan) which showed cardiomegaly, and ascites, so she was admitted in one of peripheral hospitals, who was received intravenous diuretic treatment for three days without clinical improvement, so she was referred to our department.

At admission, she presented asthenia, dyspnea, cough and painful cramps in legs muscles; physical examination showed hypotension, the systolic blood pressure was about 75 mmHg and the diastolic about 50 mmHg, with peripheral signs of

congestive heart failure, moderate mitral systolic murmur and bibasilar crackles of the lungs.

She also presented the symptoms of early cataracts of both eyes, Chvostek and Trousseau's sign was positive, in favor of severe hypocalcemia.

Her surface ECG showed sinus rhythm; diffuse repolarization abnormalities represented essentially by negative T-waves, and obvious long QT interval which measured 608 msec after its correction with Bazett's formula (Figure 1); this prolongation is caused by long segment ST which suggested severe hypocalcemia.

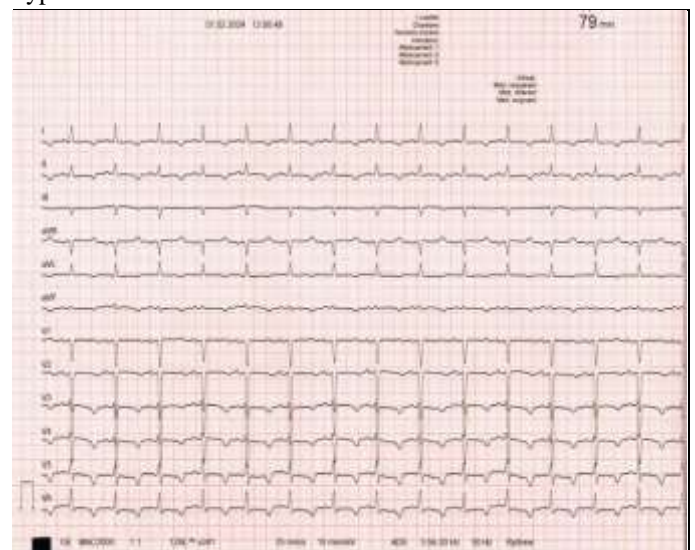


Figure 1 : Surface ECG showed sinus rhythm, diffuse negative T waves and QT interval = 530 msec, corrected QT interval = 608 msec using Bazett's formula.

During hospitalization, the chest X ray showed increased cardiothoracic ratio, which is in favor of cardiomegaly and dilation of ventricles (Figure 2)

the echocardiography doppler showed severe systolic left ventricular function with ejection fraction about 23 %, dilated diastolic left ventricle \approx 67 mm, normal diameter of ascending aorta, moderate mitral regurgitation, pulmonary artery systolic pressure about 41 mmHg, with minime pericardial effusion. (Figure 3 and Figure 4)

Cardiac magnetic resonance imaging was in favor of non-ischemic dilated cardiomyopathy. [5]

The biological assessment showed hemoglobin level : 10 g/dl, hypokalemia (3.2 mmol/l), low serum albumin level \approx 28 g/l, hypomagnesemia : 0.65 mmol/l, severe hypocalcemia: 0.975 mmol/l (normal range 2.20 to 2.60 mmol/l), with albumin adjusted calcium about 1.27 mmol/l, low Vitamin D level \approx 14.58 ng/ml (normal range 20 to 40 ng/ml), high phosphorus level: 101.22 mg/l (normal range 26 to 45 mg/l), low serum concentration of parathyroid hormone (PTH) \approx 7.233 pg/ml (normal range 15 to 65 pg/ml), high level of thyroid stimulating hormone (TSH) \approx 20.5 μ UI/ml (normal range 0.30 to 4.30 μ UI/ml), blood urea : 0.50 g/l, blood creatinine level : 9 mg/l.

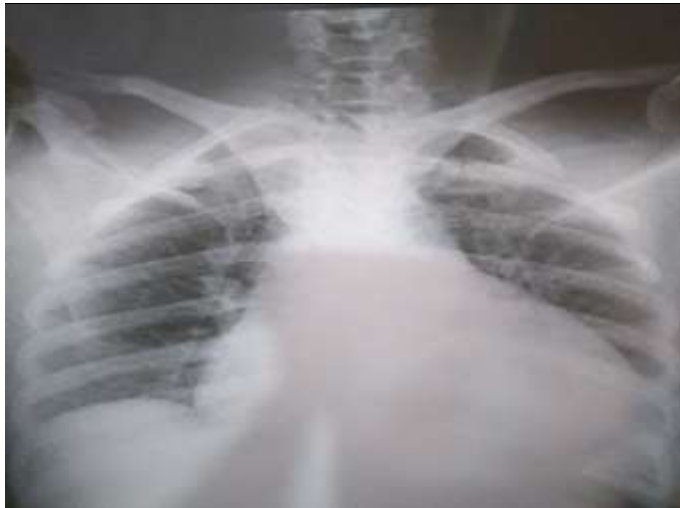


Figure 2 : Chest X ray showed increased cardiothoracic ratio

The patient had received conventional treatment of congestive heart failure such as oral Spironolactone as a potassium-sparing diuretic, hydrochlorothiazide, anticoagulation, correction of hypokalemia with potassium supplements, Vitamin D supplementation, Levothyrox 100 μ g, the patient had also received 1500 mg of Calcium Chloride per day, the 10 % Calcium Chloride Injection was administered via a central venous catheter.

After two weeks, we replaced Calcium Chloride Injection, with oral calcium supplementation (Calperos 500 mg tablets, 2 tablets /day).

The hypocalcemia has gradually increased; four weeks after supplementation treatment, heart failure symptoms were relieved, serum calcium level was 2.2 mmol/l, and albumin adjusted calcium was 2.15 mmol/l, the interval QT has shortened (Corrected QT interval = 491 msec) (Figure 6), but without improvement of severe left ventricular systolic dysfunction.

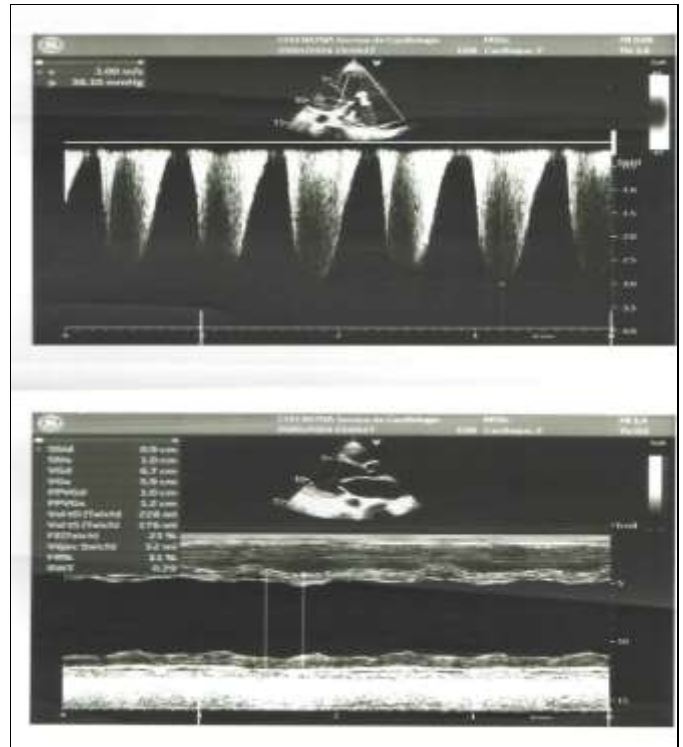


Figure 3 : Echocardiography doppler showed left ventricular ejection fraction \approx 23 %, dilated diastolic left ventricle \approx 67 mm, pulmonary artery systolic pressure \approx 41 mmHg

We have also seen improvement of other biological parameters such as hemoglobin level: 13.1 g/dl, serum Potassium level: 3.87 mmol/l, serum albumin level: 42 g/l, serum Phosphorus level: 69 mg/l, serum concentration of parathyroid hormone (PTH): 14.31 pg/ml, level of thyroid stimulating hormone (TSH) \approx 18.85 μ UI/ml, blood urea: 0.41 g/l, blood creatinine level: 7.88 mg/l.

COVID-19 Serology tests were negative with normal inflammatory parameters.

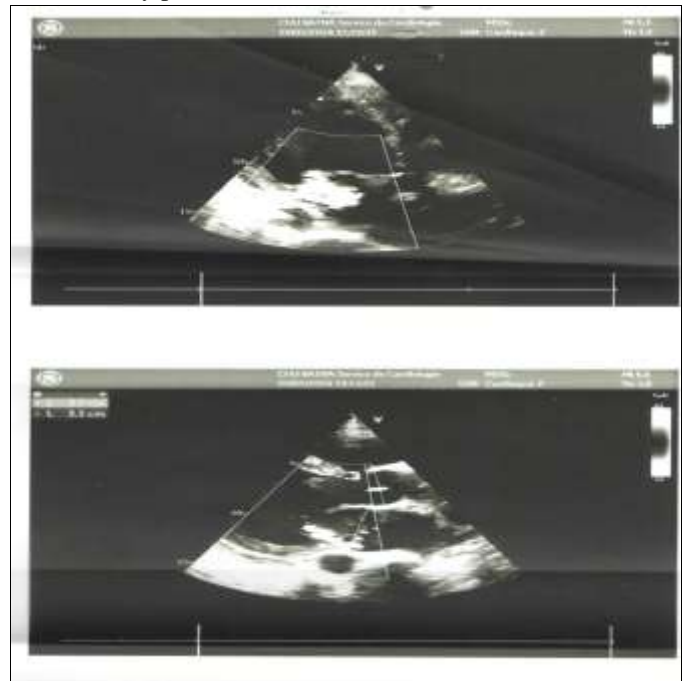


Figure 4 : Echocardiography doppler showed , normal diameter of ascending aorta, moderate mitral regurgitation, minime pericardial effusion

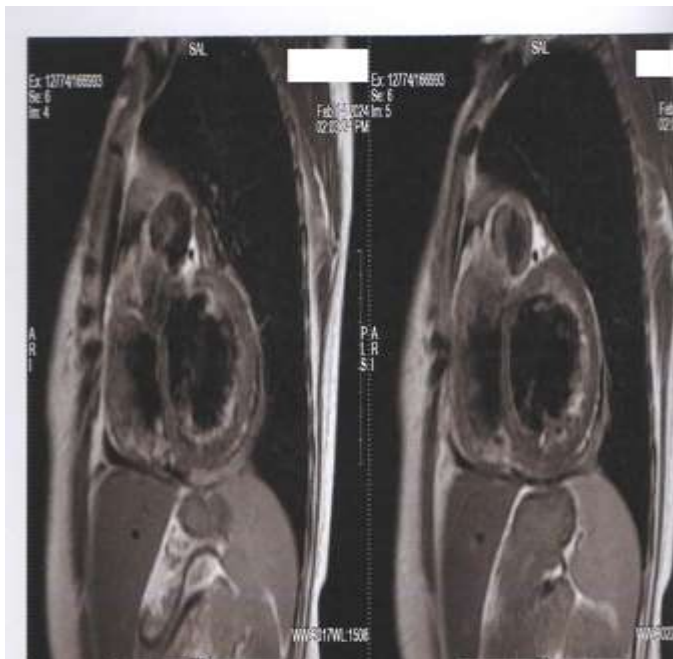


Figure 5: Cardiac magnetic resonance imaging showed non-ischemic dilated cardiomyopathy

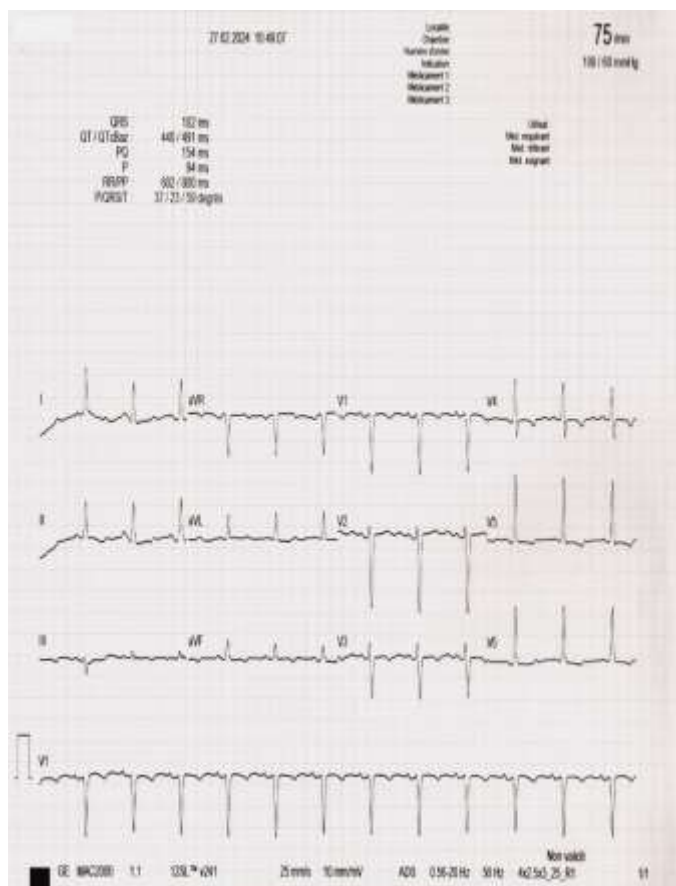


Figure 6 : Surface ECG after hypocalcemia correction, showed sinus rhythm, diffuse negative T waves and shortening of QT interval = 440 msec, corrected QT interval = 491 msec using Bazett's formula.

Discussion

Dilated cardiomyopathy is a progressive disease of heart muscle that is characterized by ventricular chamber enlargement and contractile dysfunction.

Dilated cardiomyopathy has many causes such as: inherited

disease, ischemia, hypertension, valvular disease, myocarditis, toxins, endocrine diseases, tachycardia or bradycardia, but hypocalcemia is a rare cause of dilated cardiomyopathy with congestive heart failure. [1] [2]

Severe hypocalcemia is often induced by Hypoparathyroidism secondary to surgical thyroidectomy, because thyroid resection may induce iatrogenic damage of parathyroid glands or their arteries; PTH is secreted by the parathyroid glands and is the critical regulator of blood calcium concentration.

Calcium is an essential element for constitution of several organs such as bones, muscles, eyes, and plays an important role in a wide range of biological functions, such as muscle contraction and nerve impulse transmission. [3]

Cardiac contractility is regulated by changes in intracellular Calcium; normal function requires that calcium be sufficiently high in systole and low in diastole

Calcium binds to troponin resulting in sliding of the thick and thin filaments, cell shortening, and thence the development of pressure within the ventricle and ejection of blood, so heart contractility depends on the amount of Calcium bound to troponin. [4]

Our patient had developed Hypoparathyroidism secondary to surgical thyroidectomy, but she had not received calcium supplementation; so she had severe hypocalcemia which induced progressive systolic left ventricular dysfunction over the years; all other causes of dilated cardiomyopathy were unlikely, we also excluded Post COVID19 myocarditis in our patient.

She also presented long QT interval on the surface ECG and very low serum calcium level, these two parameters were in favor of hypocalcemic dilated cardiomyopathy.

Hypocalcemic dilated cardiomyopathy may be reversible in some patients [5][6], but some irreversible cases were reported in the literature. [7] [8]

After calcium supplementation and conventional treatment of congestive heart failure, we have seen correction of hypocalcemia and shortening of QT interval, but not improvement in left ventricular systolic dysfunction, may be because severe hypocalcemia lasted a long time, with severe myocardial damage.

Conclusion

Severe Hypocalcemia is a rare cause of dilated cardiomyopathy, but should be suspected after thyroidectomy intervention which may induce hypoparathyroidism; long QT interval and low serum calcium level are the two principal parameters of hypocalcemic cardiomyopathy diagnosis, and the strategy treatment is based on calcium supplementation.

Hypocalcemic dilated cardiomyopathy may be irreversible in patient with progressive severe myocardial damages.

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