Cardiomyopathy Secondary to Hypocalcemia

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Abstract

The contractility of the heart is a result of the interplay of ionic movements in and out of its individual cells. Calcium ion plays an important role in the ventricular function. We report a 34 year old female who presented with cardiogenic shock having had no cardiac symptoms previously. She had underwent a total thyroidectomy five years back for medullary thyroid cancer. Laboratory tests revealed her calcium levels to be very low and a diagnosis of hypocalcemic cardiomyopathy secondary to hypopituitarism was made. With vigorous calcium supplementation apart from diuretics the patient had rapid clinical improvement.

Introduction

Calcium plays a central role in the sequence of myocardial excitation-contraction coupling and myocardial relaxation. Hypocalcemia manifests with a number of clinical manifestations however, observations of congestive heart failure in hypocalcemic patients are rather rare.[1] Here, we report a female patient with hypocalcemia-induced reversible cardiomyopathy with no underlying myocardial disease secondary to total thyroidectomy for medullary thyroid cancer.

Case Report

A 34-year-old female, with a history of thyroidectomy performed five years ago, secondary to carcinoma thyroid presented with worsening breathlessness and orthopnea. The patient had been on thyroxine and calcium supplements since the thyroidectomy but had stopped thyroxine and switched over to herbal nutritional supplement instead of elemental calcium since the past three months. Her BP was 70/40mm of Hg with pulse rate of 75/min with raised JVP. She had periorbital and pedal oedema with bilateral basal crepitations.

She was diagnosed to be in cardiogenic shock and was immediately started on diuretics with inotropic support (Dobutamine). Chest radiography demonstrated cardiac enlargement with pulmonary congestion and pleural effusion. Electrocardiography showed sinus rhythm with a rate of 75 beats/min. The corrected Q-T interval was 0.58 s, with T-wave inversions in leads VI to v6,II,III,aVF. M-mode and two-dimensional echocardiography revealed chamber enlargement (left ventricular internal diameter diastolic 5.4cm, end-systolic diameter 4.6cm) and severe generalized hypokinesis of the left ventricle with an ejection fraction of 30% (Figure 1). No pericardial effusions were identified. Color

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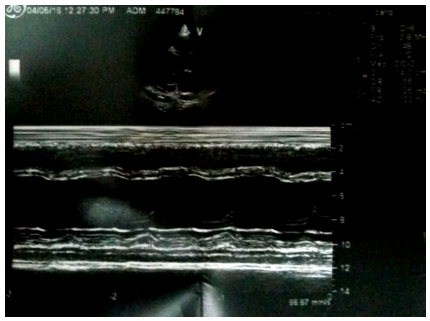

Fig. 1: Echocardiography at admission. M mode and two-dimensional echocardiography revealed chamber enlargement (left ventricular internal diameter diastolic 5.4 cm, end-systolic diameter 4.6 cm

Doppler echocardiography revealed moderate mitral regurgitation. The patient was not known to have any history of cardiac disease in the past and hence evaluation for other causes of cardiomyopathy was undertaken. Her laboratory data is elobarated in Table 1. She was found to be severely hypocalcemic with serum total calcium of 4mg/dl and ionic calcium at 2 mg/dl. A diagnosis of hypocalcemic cardiomyopathy was made and the patient was given 90 mEq of elemental calcium intravenously followed by an infusion of 50 mEq/hr for the next 24 hrs after which it was reduced to 20 mEq/hr. She received a total of 2000mEq of elemental calcium over 48 hrs. After which she was switched over to oral calcium supplements.

The patient improved clinically over the next three days with repeat calcium levels at 7mg/dl. Repeat echocardiography showed improved LV function with EF of 45% .The patient was discharged on the fifth day with thyroxine and calcium supplements.

The patient was completely asymptomatic on follow up after one month with echocardiogram showing normal chambers and EF of 48%.

Table 1: Laboratory parameters

Parameter	Value	Units
Hemoglobin	12.8	gm/dl
Total leucocyte count	11500	cells/microliter
Serum calcium	4.3	mg/dl
Ionised calcium	2.2	mg/dl
Serum magnesium	1.9	mg/dl
Serum phosphorus	6.4	mg/dl
Serum sodium	135	mg/dl
Serum potassium	3.8	mg/dl
Serum chloride	103	mg/dl
Serum creatinine	1.26	mg/dl
Serum albumin	3.3	gm/dl
Serum bilirubin	0.8	mg/dl
25-Hydroxyvitamin D	23.93	ng/ml
PTH levels	<2	ng/dl

Discussion

Ionic calcium plays a pivotal role in the maintenance and regulation of normal heart function; the changes in the concentrations of calcium in the different compartments of the cell determine the myocardial contractile force. The calcium influx from the extracellular compartment of muscle fiber facilitates the interaction of actin and myosin proteins bringing about contraction and relaxation. The influx of calcium into the cell triggers the release of calcium stored in the sarcoplasmic reticulum into the intracellular space next to the contractile proteins, resulting in the contraction of the muscle fiber. Decoupling of the same proteins occurs secondary to reentry of the calcium back into the sarcoplasmic reticulum causing drop in cystolic calcium levels.[2-4] Thus, normal levels of serum calcium are necessary for optimal myocardial function and hypocalcemia, regardless of its cause, can affect heart function to severe degrees.

The incidence of permanent hypocalcaemia after total thyroidectomy varies from 2-33%.[5] Although hypocalcaemia usually occurs soon after surgery, progressive atrophy of the parathyroid glands can result in late presentation.[6,7] As those patients may no longer be receiving regular follow up, the diagnosis is easily missed. All doctors need to be aware of the potential for hypocalcaemia to present in a non-specific manner including cardiogenic shock as in our patient. Patients with symptoms of cardiac failure with a history of thyroid surgery should always be checked for serum calcium.

Development of cardiomyopathy in hypoparathyroidism might include hypomagnesaemia, 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.

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