CASE REPORT

HYPOCALCAEMIA AS A RARE REVERSIBLE CAUSE OF DILATED CARDIOMYOPATHY

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Dilated cardiomyopathy is characterized by dilation and enlargement of one or both ventricles with reduced systolic function. Calcium plays a key role in myocardial contraction. Hypocalcaemia can lead to a decrease in contraction, left ventricular systolic dysfunction, and heart failure with reduced ejection fraction (EF). Hypocalcaemia is a rare reversible cause of dilated cardiomyopathy. The author presents a case who presented with complaints of shortness of breath on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, numbness and crampy muscular pains. He had a high JVP, systolic murmur on auscultation, hepatomegaly, pedal oedema and crackles on chest auscultation. His ECG showed sinus rhythm with prolonged QT interval. His echocardiography showed dilated cardiomyopathy with reduced ejection fraction, moderate mitral regurgitation and mild tricuspid regurgitation. His Calcium levels and PTH levels were both low. He was treated with ionotrophes, diuretics, vitamin D and calcium supplements, including both intravenous and oral. With the correction of calcium levels, he was weaned off the ionotrophic support and his ejection fraction improved. Calcium levels if low should be corrected in patients with dilated cardiomyopathy.

Keywords: Hypocalcaemia; Dilated cardiomyopathy; Hypoparathyroidism

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INTRODUCTION

Calcium plays an important role in the contraction of all types of muscles. Calcium has an important role in excitation contraction coupling with hypocalcaemia decreasing myocardial contractility.[1] Decreased myocardial contraction and even congestive cardiac failure have been described with hypocalcemia.[2] Here we describe a case of dilated cardiomyopathy caused by hypocalcaemia due to primary hypoparathyroidism.

CASE DETAILS

4. 18 years 18-year-old male patient presented with complaints of shortness of breath on exertion for the past two weeks, associated with orthopnoea and paroxysmal nocturnal dyspnoea. There was also associated generalized body weakness, numbness and crampy muscular pain for the previous four weeks. He denied any fever. He did not have any significant previous medical history. There was no recent travel or recent vaccination history and he was not on any regular medications. There was no family history of sudden cardiac death or any cardiomyopathy.

On examination he was sitting in a propped-up position, had a blood pressure of 85/60, was tachypneic and tachycardic with a heart rate of 130, pulse was low volume and regular. A general physical examination showed grade two pedal oedema and pallor. His JVP was 10 cm from a sternal angle with positive abdominal jugular reflux. The apex beat was displaced inferolateral to the 6th intercostal space and 3cm lateral to the midclavicular line. On auscultation, there was S3 gallop and pan-systolic murmur more pronounced at the mitral area and left lower sternal border. Chest examination showed bilateral crackles up to mid zone, more on the right side. Abdominal examination showed tenderness in the right hypochondrium with palpable liver 3 cm below the right costal margin. He also had a positive trousseaus and chovstek sign.

Chest X-ray of the patient showed cardiomegaly with features of left atrial enlargement along with pulmonary congestion. His ECG showed sinus rhythm with prolonged QT interval and inverted T waves. His echocardiography showed a dilated left ventricle with reduced ejection fraction (29 percent) moderate mitral regurgitation and right ventricular dysfunction with PASP 30, along with mild tricuspid regurgitation. The patient had a bilirubin level of 0.45 mg/dl and an ALT of 525U/l. His renal functions were deranged with urea of 98 mg/dl and creatinine of 2.02 mg/dl. Renal ultrasound was normal. His CRP was negative and ESR was 14. His serum calcium was 3.59 mg/dl (8.0-10.0). His ANA was negative. Repeated Calcium was persistently low with normal phosphate level. His PTH level was sent which turned out to be less than 3 pg/ml.

He was diagnosed with heart failure due to dilated cardiomyopathy secondary to hypocalcaemia due to primary hypoparathyroidism based on low calcium, PTH and normal renal ultrasound.

Hypoparathyroidism is associated with other autoimmune conditions such as poly endocrine syndromes which is a combination of autoimmune thyroid diseases, type 1 diabetes mellitus, hypoparathyroidism and Addison disease in various combinations. His thyroid function tests and HbA1C were normal and there were no electrolyte abnormalities, postural hypotension or hyperpigmentation to suggest Addison's disease.

Table-1: Investigations

Investigation	Value	Normal range	Investigation	Value	Normal range
Calcium	3.59mg/dl	8.0-10.0 mg/dl	Blood Cultures	No Growth	
Albumin	3.15 g/dl	3.5-5.0 g/dl	Hb	11.8 g/dl	11.5-17.5 g/dl
Corrected Calcium	3.87 mg/dl	8.0-10.0 md/dl	WBC	10.5	4-11
Vitamin D	48.64 ng/ml	40-100 ng/ml	troponin	0.294 ng/mL	<0.6 ng/mL
PTH	<3 pg/ml	16-87 pg/ml	ANA	Negative	
Urea	98 mg/dl	18-45 mg/dl	TSH	0.63 µIU/ml	0.3-4.2 µIU/ml
Creatinine	2.02 mg/dl	0.3-0.9 md/dl	T4	8.73 µg/dl	5.1-14.1 µg/dl
Bilirubin	0.45 mg/dl	0.1-1.0 mg/dl	HCV	Negative	
ALT	525 U/l	10-50 U/l	HBS Ag	Negative	
Covid 19	Negative				

He was started on dobutamine and furosemide infusion, spironolactone was added later on when blood pressure was stabilized. Endocrine consultation was made for hypocalcaemia due to hypoparathyroidism and he received calcium gluconate, and vitamin D. Following the commencement of Calcium and Vitamin D he improved both clinically and biochemically, he was weaned of the ionotrophic support and his renal and liver functions improved.

He is seen in the clinic every six months. He is currently on four heart failure medications including carvedilol 6.25 mg BD, rampiril 2.5mg OD, spironolactone 40mg and empagliflozin 5mg. He had echocardiography done after six months of the initial echo which showed improvement of ejection fraction from 28 to 48 percent. He is on oral vitamin D and Calcium replacements.

DISCUSSION

Dilated cardiomyopathy is a disease caused by dilation and enlargement of one or both ventricles, with reduced contractility (ejection fraction <40%). Dilated cardiomyopathy has many causes the familial causes are classified as idiopathic. Secondary causes of cardiomyopathy include myocardial viral infections, ischaemia, hypertension, medications, alcohol, HIV, peri partum and infiltrative diseases.[3] Hypocalcaemia is a rarely reported cause of dilated cardiomyopathy. Hypocalcaemia can be caused by vitamin D deficiency or hypoparathyroidism. In children, it is usually caused by vitamin D deficiency.[4]

Calcium in the blood can be attached to albumin, or it can be free (also called ionized calcium). Ionized calcium plays an important role in myocardial contraction. During the action, potential ionized calcium enters the myocardium through depolarization-activated calcium channels and in turn, triggers the release of calcium from the sarcoplasmic reticulum. Hypocalcaemia can change the contractility of the myocardium by altering the amplitude of calcium transit or sensitivity of myofilaments to calcium.[5]

Patients with hypocalcaemia may be asymptomatic if mild and chronic. Moderate and severe hypocalcaemia is associated with paraesthesia of fingers and circumoral areas and on examination trousseaus and chovstek signs can be elicited. Severe hypocalcaemia can cause seizures, carpaly spasms, bronchospasms, laryngospasms and QT prolongation.[6]

Our patient presented with signs and symptoms of heart failure, and echocardiography demonstrated features of dilated cardiomyopathy. His young age, absence of chest pain and negative troponins made ischemic causes very unlikely. He did not have a family history of any cardiac conditions. There were no signs or symptoms of viral illness and together with negative CRP made the viral aetiology very unlikely. He denied any alcohol intake and he was not on any regular medications. His ionized calcium was very low, along with low PTH levels. He was therefore started on calcium and vitamin D along with other medications for heart failure in the form of ionotrophic support and diuretics. With the correction of Calcium levels, he improved objectively, we were able to wean him off the ionotrophic support and on follow-up after six months his ejection fraction improved from 29 to 48 %. The patient did not have any underlying cardiomyopathy as with correction of calcium his ejection fraction has improved from 29 percent to

48 percent. His ejection fraction did not improve initially with medications for heart failure only, his ejection fraction improved only after the correction of hypocalcaemia. Also, once cardiomyopathy has developed due to any cause such as myocarditis, metabolic causes, toxic causes or hypocalcaemia ultrastructural abnormalities persist despite correction or removal of underlying cause and heart failure therapy is recommended to be continued as there are high risk of recurrence on stoppage of therapy

Cardiomyopathy due to hypocalcaemia is usually resistant to conventional treatments, however, there is a good recovery in cardiac function with the restoration of normal calcium levels.4 Furosemide used in the treatment of heart failure can worsen hypocalcaemia therefore strict monitoring of calcium levels should be done in dilated cardiomyopathy with hypocalcaemia. Also, discontinuation of calcium supplements can lead to the reappearance of features of heart failure.[7]

The patient did not have any underlying cardiomyopathy as with correction of calcium his ejection fraction has improved from 29 percent to 49 percent on 6 monthly follow up. His ejection fraction did not improve initially with medications for heart failure only, his ejection fraction improved only after correction of hypocalcaemia. Also, once cardiomyopathy has developed due to any cause such as myocarditis, metabolic causes, toxic causes or hypocalcaemia ultrastructural abnormalities persist despite correction or removal of underlying cause and heart failure therapy is recommended to be continued as there are high risk of recurrence on stoppage of therapy

CONCLUSION

Hypocalcaemia though rare is an important reversible cause of dilated cardiomyopathy. Calcium levels should be checked in all cases of dilated cardiomyopathy. As dilated cardiomyopathy due to hypocalcaemia usually doesn't respond to conventional treatments, therefore Calcium levels should be corrected in patients where the aetiology of cardiomyopathy is not clear. Also, discontinuation of calcium supplementation can lead to the appearance of symptoms of heart failure.

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