A Rare Cause of Heart Failure - Primary Hypoparathyroidism

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Abstract

We report a patient who presented with congestive heart failure (ejection fraction 24.4%) and who had previous history of convulsions. Our investigations found him to be a case of primary hypoparathyroidism. He showed a dramatic response with the addition of calcium infusion therapy with almost full recovery of left ventricular function (67% ejection fraction after 16 days of the initial echo). We conclude that in a young patient a thorough investigation for heart failure is never complete without looking for endocrine and metabolic causes. The prognosis in these cases is much better, identification and treatment of the same will yield dramatic results.

INTRODUCTION

Parathyroid hormone (PTH) works to regulate total body calcium. The body secretes PTH in response to hypocalcemia. Primary hypoparathyroidism results in deficient PTH secretion and is characterized by hypocalcemia and hyperphosphatemia. It may occur as familial isolated hypoparathyroidism and may show autosomal dominant, autosomal recessive, or X-linked inheritance. It is known that the hypocalcemic state reduces myocardial contractility; however the incidence of congestive heart failure due to hypocalcemia is quite rare in clinical practice.

CASE REPORT

An 18 year old boy presented with a history of sudden onset of breathlessness for one day with orthopnea, cough and mild grade fever. On further enquiry the patient gave history of convulsions off and on for the last two and half years. There was also history suggestive of tetany. Previous records revealed lowered serum calcium levels. He had no history of pre-existing cardiac illness. On examination, his respiratory rate was 28 per minute; his temperature was 37.4°C, pulse 120 /minute and BP - 80/40 mm Hg. There was no pallor. Chest auscultation revealed marked crepitations bilaterally. Cardiac examination revealed tachycardia with audible S1 and S2, and a grade 2-3/6 short systolic murmur was audible in the mitral area. Abdominal and neurological examination was normal.

Laboratory investigations revealed: Hb- 10.2 g/dl, TLC - 13,100 cells/µL, with 75% polymorphonuclear cells. Liver function tests and renal function tests were found to be within normal limits. An electrolyte study revealed hypokalemia (2.6mmol/L) (ref range: 3.5-5.0mmol/l), hypocalcemia (1.77mmol/L) (ref range; total calcium: 2.1-2.6mmol/L), hyperphosphatemia (3.75mmol/L) (ref range: 0.8-1.45mmol/L) and mild hypomagnesemia (0.6mmol/L) (ref range: 0.75-1.25mmol/L). His 24 hour urine calcium level was 0.135mmol/d, (ref range: 2.5-7.5mmol/d) showing a hypocalciuric state. His ECG revealed prolonged QTc interval (0.55sec) (Fig. 1). CT Scan Head showed intracranial calcifications in the basal ganglia. Chest skiagram revealed cardiomegaly (cardiothoracic ratio – 0.54) with bilateral hilar congestion. Parathormone value was <0.1 pmol/L (ref. range: 1.0-7.5 pmol/L). The

Fig. 1 : ECG at time of admission showing QTc prolongation.
abdominal ultrasonography report was normal.

On emergency echocardiography, the left ventricular internal diameter (LVID) (diastolic) measured 4.48cm, and the LVID (systolic) measured 4.08cm. The interventricular septum thickness was 0.81cm. The ejection fraction was poor (24.4%) with marked diastolic dysfunction. There was moderate mitral regurgitation (Fig. 2A). These findings suggested that the patient was in severe left ventricular failure.

Treatment was initiated with inotropes to raise the blood pressure. Digoxin was added to the regimen. Calcium, magnesium and potassium were supplemented. The patient had one episode of convulsions at the time for which a duty doctor put him on intravenous phenytoin. The convulsions receded but the following day patient developed increasing episodes of tetany. 10 ml of 10% calcium gluconate was given intravenously over 10 minutes which relieved his tetany temporarily. His serum calcium (ionic and total) levels were repeated and found to be further reduced (0.85mmol/L and 1.32mmol/L respectively) [ref range; ionic calcium: 1.05 -1.45mmol/L].

The patient’s respiratory distress had until now only slightly improved. Dopamine drip was withdrawn once the blood pressure became normal. Magnesium was stopped after supplementing for three days. Phenytoin was replaced by sodium valproate as phenytoin has hypocalcemic properties. Now, 50 ml of 10% calcium gluconate was added to 1 L of 5% glucose in water solution and given as a slow continuous infusion at 2 mg/kg/hr, following which, the patient’s condition improved rapidly. Once the serum levels reached normal limits, oral calcium carbonate 1g daily along with calcitriol 0.25µg daily was initiated. Repeat chest skiagram after 2 weeks showed normal sized heart (cardiothoracic ratio – 0.47) and ECG showed normal QTc interval (0.41sec).

Echocardiogram repeated on the 16th day demonstrated the almost complete recovery of the left ventricular function. The LVID (diastolic) was now 3.71cm, whereas the LVID (systolic) was 2.46cm. The interventricular septal thickness was 0.96cm. The Ejection fraction had increased to 67% with normal systolic and diastolic functions. There was only a trivial mitral regurgitation.

![Fig. 2a: Initial echocardiogram. Increased left ventricular dimensions moderate mitral regurgitation. Interventricular septal thickness 0.81 cm and ejection fraction 24.4%.

![Fig. 2b: Repeat echocardiogram on Day 16. Normal left ventricular dimensions, trivial mitral regurgitation, interventricular septal thickness 0.96 cm and ejection fraction 67%.]
present (Fig. 2B). The patient was diagnosed as a case of Primary Hypoparathyroidism with hypocalcemic heart failure.

**DISCUSSION**

This patient was a documented case of hypocalcemia, but had never been evaluated previously. And so he had remained symptomatic for so long. The cause for the heart failure had us stumped initially as we couldn’t identify a cause. Also his condition was refractory to conventional treatment of heart failure.

In this patient, there was initially a diagnostic problem since such cases are rarely seen in clinical practice. The inadequate improvement with use of digoxin and ionotropic agents and the complete reversal of the heart failure with calcium infusion is strong evidence in favour of hypocalcemia induced heart failure. It is a known fact that hypocalcemia prolongs the duration of phase two of the action potential of cardiac muscle. Calcium is released from the sarcoplasmic reticulum of the cardiac myocyte. This release is regulated mainly by the amount of calcium within the sarcoplasmic reticulum, which has direct effect on the systolic calcium and thereby on the contraction of the cardiac muscle. In other words, the concentration of the extracellular calcium ion is considered to have a direct effect on the strength of the myocardial contraction through excitation-contraction coupling.

The review of literature mentioned very few similar cases worldwide and we did not come across any such case reports from India. The main metabolic causes of heart failure include thyroid disorders, vitamin deficiencies including thiamine, ascorbic acid, endocrine abnormalities such as acromegaly, diabetes mellitus, pheochromocytoma, and rare disorders like porphyria. Primary hypoparathyroidism is a very rare cause of heart failure and must be remembered in the etiological assessment of a young patient presenting with heart failure.

**REFERENCES**


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**Announcement**

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