

Hypoparathyroidism – A Cause of Reversible Dilated Cardiomyopathy

Pankaj V Jariwala, B Sudarshan, MS Aditya, L Praveer, K Sarat Chandra

Abstract

Hypoparathyroidism is associated with chronic hypocalcaemia which can have a variety of manifestations including cardiovascular changes. We describe here a case of primary hypoparathyroidism associated with systolic dysfunction of left ventricle presenting as a dilated cardiomyopathy in addition to other manifestations.

Introduction

Dilated cardiomyopathy with left ventricular systolic dysfunction can be reversible in a wide variety of conditions including peripartum cardiomyopathy, alcoholic cardiomyopathy and so on. Metabolic conditions like hypothyroidism can be associated with left ventricular systolic dysfunction. We describe here a case of hypoparathyroidism which was associated with reversible left ventricular systolic dysfunction that recovered completely with treatment.

Case Report

A 24 year old man presented with history of seizures for the previous one year. He was being treated with carbamazepine. He was referred to us for evaluation of recent onset shortness of breath Class II and pedal edema. There was no history of chest pain, orthopnea or paroxysmal nocturnal dyspnea.

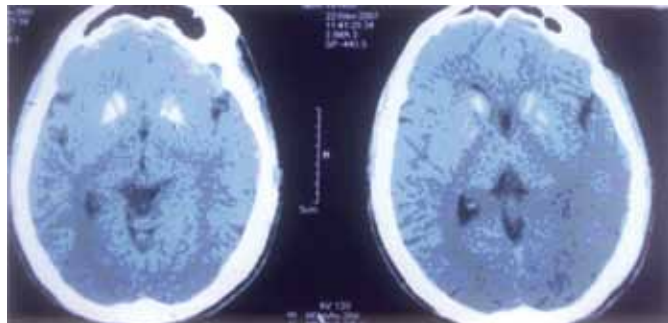


Fig. 1 : CT scan demonstrating bilateral basal ganglia calcification (Arrows)



Fig. 2 : Photograph showing mature cataract in the left eye.

Nizam's Institute of Medical Sciences, Punjagutta, Hyderabad – 500 082.

Received: 18.02.2009; Accepted: 01.10.2009

There was H/o loss of weight. Appetite was normal. He never underwent any surgery of thyroid gland in the past. His developmental history was significant for delayed mile stones and poor scholastic performance. CT scan done one year ago showed calcification of basal ganglia bilaterally (Fig. 1).

On physical examination he was under built with a height of 150cm and weight of 50kgs. Heart rate was 96/min, BP was 100/70 mm Hg and there was LV S3. Trousseau's sign and Chvostek's sign were positive. There was mature cataract in the left eye (Fig. 2). There was pedal edema bilaterally, however JVP was not elevated and liver was not palpable.

Serum Calcium was 4.5 mg/dl (8.7–10.2 mg/dL). Serum Phosphorus 5.6 mg/dl (2.5–4.3 mg/dL). Serum albumin 4.4 g/dl. Alkaline phosphatase was 272 IU/L (270 being ULN) Serum Magnesium 0.9 mmol/l (0.62–0.95 mmol/L). He had anemia suggestive of iron deficiency. The laboratory reports of the patient is shown in Table 1.

A diagnosis of primary Hypoparathyroidism was considered in view of hypocalcemia with tetany, hyperphosphatemia, seizures, bilateral basal ganglia calcifications and cataract. We suspected that the congestive heart failure - NYHA Class II was a sequel of severe chronic hypocalcemia. ECG revealed a prolonged QT interval (QTc 518 ms), lengthening of ST segment, T wave inversions in leads V2 - V6, I, aVL, II, III, aVF (Fig. 3). Echocardiography revealed dilated cardiac chambers, global hypokinesia with left ventricular Ejection fraction of 35% and moderate LV systolic dysfunction (Fig. 4). Intact parathormone levels were very low at 2.5 pg/ml. 25 hydroxy D3 levels were in normal range at 36.76 ng/ml. Primary hypoparathyroidism probably idiopathic

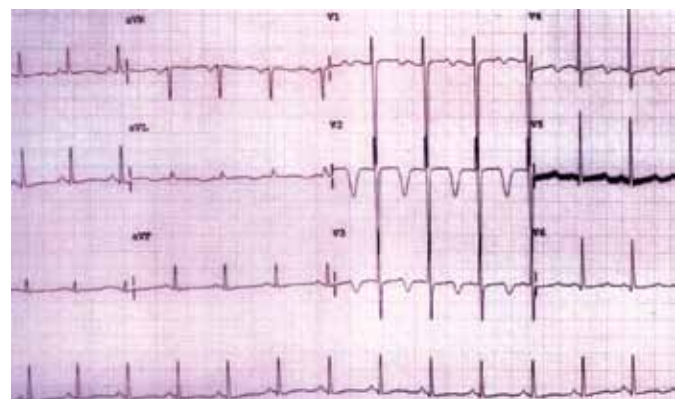


Fig. 3 : Electrocardiogram showing QT prolongation, seen well in the precordial leads.



Fig. 4 : M-mode echocardiogram showing reduced ejection fraction and Dilated LV at the time of diagnosis

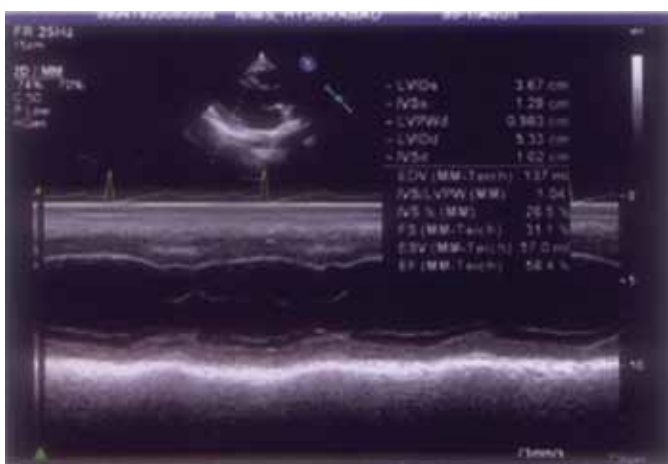


Fig. 5 : Six weeks after starting treatment the LV shows recovered function with EF of 58%

was diagnosed and the patient was started on calcium supplementation.

Initially calcium gluconate infusion was given and later he was switched to oral calcium supplements and 1,25 hydroxy D3. Thiazide diuretic was started. He improved symptomatically with decreased seizures and improvement in functional class. Echo parameters of LV function along with LV dimensions showed improvement at 6 weeks follow up (Fig. 5).

Follow up

He became normocalcaemic over a period of four days with a QTc of 0.40 sec. Left ventricular function improved within two weeks. His heart failure and seizures completely subsided. Later he underwent surgery for cataract uneventfully.

Discussion

Common cardiac manifestations of hypocalcemia include prolonged QT interval and arrhythmias. Chronic hypocalcemia is a relatively uncommon and reversible cause of congestive heart failure. Reports of congestive heart failure secondary to hypocalcemia are rather rare. Hegglin reported in 1939 that hypoparathyroidism with hypocalcemia involves the heart.¹ Most of the cases reported till date were in children and post thyroidectomy patients with Idiopathic hypoparathyroidism being reported uncommonly.²⁻⁵

The Cardiovascular manifestations of hypocalcaemia

Table 1: Laboratory reports

	Value	Normal Range
Serum calcium	4.5mg/dl	8.7-10.2mg/dl
Serum phosphorus	5.6mg/dl	2.5-4.3mg/dl
Serum potassium	3.4meq/l	3.5-5.5meq/l
Serum magnesium	0.9mmol/l	0.62-0.95mmol/l
Serum creatinine	1.0 mg/dl	0.6-1.5mg/dl
Serum albumin	4.4gm/l	3.0-5.0gm/l
Alkaline phosphatase	272.0IU/l	170-370 IU/l
Hb	9.1gm/dl	13-17 gm/dl
PCV	28.7%	40-50%
MCV	65.7	83-101fl
MCH	20.9	27-32 pg
MCHC	31.8	31.5-34.5gm/dl
TLC	4400	4000-10000/cumm
Neutrophils	68	40-80%
Lymphocytes	29	20-40%
Monocytes	2	2-10%
Eosinophils	1	1-6%
Serum 25, hydroxy vitamin D	36.76ng/ml	9.0-37.6ng/ml
Parathyroid hormone	<2.5 pg/ml	14.0-72pg/ml

Hb-Hemoglobin, PCV-Packed cell volume, MCV-Mean corpuscular volume, MCH-Mean corpuscular hemoglobin, MCHC-Mean corpuscular hemoglobin concentration, TLC-Total leucocyte count

include prolongation of QT interval, refractory life threatening hypotension ventricular arrhythmias and dilated cardiomyopathy.¹⁻⁹

Pathophysiology of Cardiac Failure in Chronic Hypocalcemia

Calcium has a crucial role in regulating the contraction and relaxation phases of the cardiac cycle. The details of the associated calcium ion fluxes that link contraction to the wave of excitation (excitation-contraction coupling) have now been reasonably well delineated.¹¹

Hypocalcaemia leads to decreased myocardial contractility, clinically this may present as congestive heart failure. The congestive cardiac failure in hypocalcaemia is refractory to diuretics and digitalis but rapidly responds to restoration of calcium concentrations to normal.¹²

Calcium infusion increases both cardiac output and blood pressure in hypoparathyroid patients suggesting a subclinical direct cardiac dysfunction due to hypocalcaemia.¹³ Hypomagnesaemia and reduced circulating parathyroid hormone may also be involved in causing dilated cardiomyopathy in hypoparathyroidism.⁹

Our patient initially presented with seizures which were inadequately controlled with carbamazepine, subsequent presentation for congestive heart failure led to more thorough metabolic evaluation.

Investigations revealed hypocalcemia, mild hyperphosphatemia, normal alkaline phosphatase, normal 25 hydroxy D3 and low intact parathormone levels.

Hypocalcemia is an important cause of reversible cardiomyopathy and lack of awareness of this etiology may lead to inappropriate therapy of cardiac failure with loop diuretics, leading to a worsening of hypocalcaemia and its possible acute life threatening manifestations (laryngeal spasm, prolonged QTc, ventricular arrhythmias, and refractory hypotension), by

increasing renal excretion of calcium.

Conclusion

Hypocalcemia should be considered as a possible cause of dilated cardiomyopathy when the patient presents with heart failure due to left ventricular systolic dysfunction in association with seizures and other neurologic manifestations and the electrocardiogram shows prolonged QT interval. Recognition of the condition is important as it is highly treatable.

References

1. Hegglin R. Herz und Hypocalzämie. *Helv Med Acta* 1939;5:584.
2. Pollard AB, Brenton DP, Gonzales J. Hypocalcemic cardiac failure. *Postgrad Med J* 1980;54:633.
3. Bashour T, Basha HS, Cheng TO. Hypocalcemic cardiomyopathy. *Chest* 1980;78:663.
4. Giles TD, Iteld BJ, Rives KL. The cardiomyopathy of hypoparathyroidism another reversible form of heart muscle disease. *Chest* 1981;79:225-229.
5. Heart failure, a thick tongue, and an abnormal cranial computed tomogram *Postgrad Med J* 2001;77:537g.
6. Doroghazi RM, Childers R. Time-related changes in the Q-T interval in acute myocardial infarction: possible relation to acute hypocalcaemia. *Am J Cardiol* 1978;41:684-688[Medline].
7. Reber PM, Heath H III. Hypocalcaemic emergencies. *Med Clin North Am* 1995;79:79-92[Medline].
8. Reddy CVR, Gould L, Gomprecht RF. Unusual electrocardiographic manifestations of hypocalcaemia. *Angiology* 1974;25:764-768.
9. Eastell R, Heath H III. Hypocalcaemic states: their differential diagnosis and management. In: Coe FL, Favus MJ, eds. Disorders of bone and mineral metabolism. New York: Raven Press 1992;571-585.
10. Wong CK, Lau CP, Cheng CH, et al. Hypocalcemic myocardial dysfunction: short and long term improvement with calcium replacement. *Am Heart J* 1990;120:381-386[Medline].
11. Opie LH: Mechanisms of cardiac contraction and relaxation. In: Zipes DP, Libby P, Bonow R, Braunwald E, ed. Heart Disease. A Textbook of Cardiovascular Medicine, 7th ed. Philadelphia: Elsevier Saunders; 2005:457-489.
12. Levine SN, Rheams CN. Hypocalcemic heart failure. *Am J Med* 1985;78:1033-1035[Medline].
13. Zonszein J, Sonnenblick EH. Endocrine diseases and the cardiovascular system. In: Alexander RW, Schlant RC, Fuster V, et al, eds. Hurst's the heart. New York: McGraw-Hill, 1998;2117-2142.