



Correspondence

Rare case of massive pericardial effusion secondary to primary hypoparathyroidism



1. Introduction

Calcium is essential for excitation contraction coupling in skeletal and smooth muscles, so for heart. Studies have revealed that hypocalcemia as an unusual cause of reversible cardiomyopathy leading to heart failure and very rarely to massive pericardial effusion. Herein we present a case of idiopathic hypoparathyroidism with multiple systemic manifestations along with massive pericardial effusion.

2. Case presentation

A 32 years old male was a known case of seizure disorder since last 12 years for which he was on carbamazepine and topiramate. He had no addictions or significant family history. Exploration of history revealed that he had history of frequent tetany episodes for last 3–4 years and underwent bilateral cataract surgery 3 years back. He had generalised weakness for last 2–3 months. During routine examination he had muffled heart sounds and cardiomegaly in chest x-ray for which echocardiography was done which revealed massive pericardial effusion with no features of tamponade (Fig. 1)

Patient was admitted for evaluation of pericardial effusion. In ECG, QTc was prolonged (QTc- 465 ms) and Chvostek and

Trousseau's signs were present. He had normocytic normochromic anemia (Hb-11.0 g%), normal blood urea (blood urea-26 mg/dl) and serum creatinine (0.74 mg/dl), low serum calcium (3.5 mg/dl), elevated phosphorus level (8.66 mg/dl), low magnesium level (1.5 mg/dl) with albumin on lower side (3.3gm%) and total serum protein within normal range (7.7 g%). Thyroid function tests were normal (T₃- 0.825, T₄- 5.05, TSH- 4.98, Anti TPO antibody- 5.0 IU/ml), iPTH was very low (3.57 pg/ml) and Vitamin-D level was on higher side (44.27 ng/ml). His FBS was- 116 mg/dl and HbA1c was 6.0%. Diagnostic pericardiocentesis showed TLC of 200 with 90% lymphocytes in pericardial fluid with elevated proteins (7 g%) and low sugar (11.0 mg/dl). Gram staining was negative, aerobic and anaerobic cultures were sterile, tubercular work up of pericardial fluid was negative (GeneXpert- negative, TB PCR-negative, ADA- 16.0 unit/litre, negative AFB staining and mycobacterial culture) and malignant cytology of pericardial fluid was also negative (Fig. 2).

Patient had generalised weakness for last 2–3 months and his CK-NAC levels were elevated (1237 U/L). Rheumatoid factor and ANA were negative. CECT chest and abdomen done after diagnostic pericardiocentesis had moderate pericardial effusion with pigtail in situ and normal pericardial thickness and moderate right sided and mild left sided pleural effusion. There were bilateral basal ganglia, thalamic and cerebellar calcification in NCCT head and also in CEMRI brain there was altered signal intensity suggestive of calcification in same areas.



Fig. 1. Apical 4C view showing massive pericardial effusion.

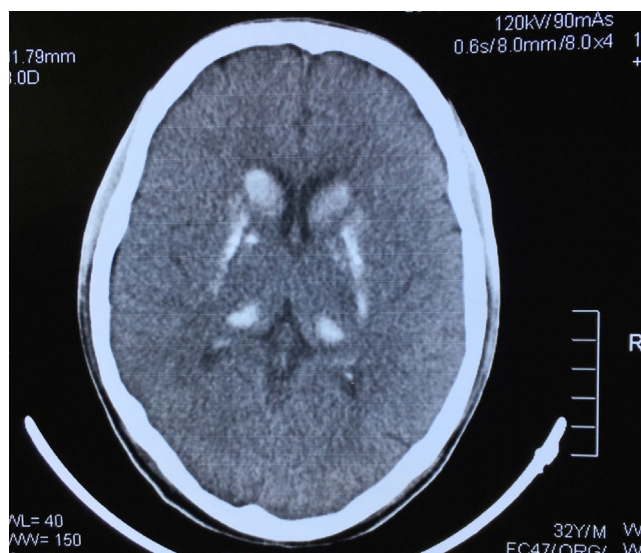


Fig. 2. Basal ganglia and thalamic calcification in NCCT head.

3. Discussion

Calcium plays a vital role in excitation contraction coupling and heart failure caused by hypocalcaemia is known as hypercalcaemic cardiomyopathy. It has been reported that hypocalcaemia associated with hypoparathyroidism leads to pericardial effusion in patient without heart failure but usually small effusion are reported. While reviewing the literature, we came across a single case report of massive pericardial effusion with hypercalcaemic cardiomyopathy.¹ Best to our knowledge massive pericardial effusion secondary to primary hypoparathyroidism in absence of cardiomyopathy along with other systemic manifestation of hypoparathyroidism has not been reported. In our case, patient had history of seizures which were probably because of hypocalcaemia. Glucose intolerance secondary to hypocalcaemia has been reported which improves with correction of hypocalcaemia, as occurred in our patient.² Premature bilateral cataract in our patient was also most likely because of hypocalcaemia for which he underwent cataract surgery. In literature also recurring seizures and tetany with bilateral cataract in idiopathic hypoparathyroidism is well described.³ There was bilateral basal ganglia, bilateral thalamic and cerebellar calcification secondary to hypoparathyroidism.⁴ Patient had generalised weakness along with elevated muscle enzymes. An inverse relation between degree of hypocalcaemia and elevation of muscle enzymes has been reported in idiopathic hypoparathyroidism myopathy.⁵ Patient was managed with calcium supplementation, activated vitamin D analogue and cholecalciferol. Gradually patient improved and at the time of discharge patient had only minimal pericardial effusion, no tetany episodes and improvement in his generalised weakness.

4. Conclusion

Hypoparathyroidism is a known cause of reversible dilated cardiomyopathy with pericardial effusion but massive pericardial effusion in absence of cardiomyopathy along with multiple

systemic manifestation of hypoparathyroidism has not been reported. Clinician should be cautious of this rare etiology while routinely managing cases of pericardial effusion.

Conflict of interest

None.

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Suraj Khanal*

3rd Floor Block-C, Advanced Cardiac Centre, PGIMER, Chandigarh,
160012, India

Rakesh Sharma

Sudhanshu Budakoty

Advanced Cardiac Centre, PGIMER, Chandigarh, 160012, India

* Corresponding author.

E-mail addresses: khanalsuraj74@gmail.com (S. Khanal),
sharmarakesh.med33@icloud.com,
sharmarakesh.med33@yahoo.com (R. Sharma),
sudhanshu_1811@yahoo.co.in (S. Budakoty).

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