DOI: 10.7860/JCDR/2025/74966.20609

Case Report

Internal Medicine Section

Cardiac Failure Secondary to Idiopathic Hypoparathyroidism: A Case Report

MANSI HARALE[1], SREEVIDYA YEKKALURU[2], ANITA BASVARAJ[3]

ABSTRACT

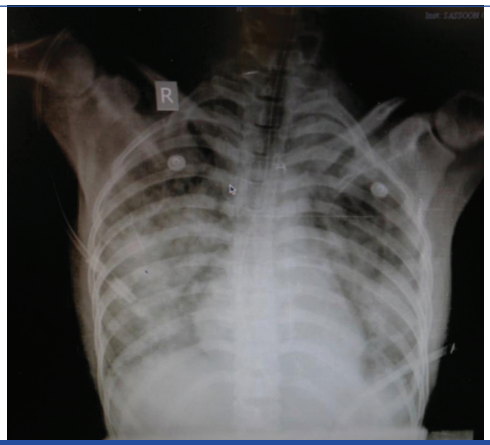
Hypoparathyroidism causes low calcium levels due to insufficient Parathyroid Hormone (PTH). This can lead to cardiovascular issues, including arrhythmias and rarely, hypocalcaemic cardiac failure. In cardiac failure, calcium dysregulation impairs contractility and increases myocardial stress. Hypoparathyroidism-induced hypocalcaemic cardiac failure is an exceptionally rare cause of heart failure, often unresponsive to standard treatments. This report highlights a case of a young female with undiagnosed idiopathic hypoparathyroidism who developed acute cardiac complications postoperatively after an appendectomy. Sixteen hours postsurgery, she experienced sudden onset breathlessness, with her oxygen saturation dropping to 50%. A chest X-ray showed bilateral homogeneous opacities consistent with pulmonary oedema. She was intubated, placed on invasive mechanical ventilation, and required pressor support before being transferred to the medicine unit. Initial Electrocardiography (ECG) findings were unremarkable. On the second day, carpal spasm was observed, prompting serum calcium testing, which revealed significantly low levels, as did serum PTH. Subsequent ECG demonstrated changes associated with hypocalcaemia, including a short PR interval and prolonged QT interval. Echocardiography indicated left ventricular hypokinesia with an ejection fraction of 25-30%, although cardiac enzymes were normal. N-terminal prohormone of brain natriuretic peptide (NT-proBNP) levels were elevated, and arterial blood gas analysis showed metabolic alkalosis and hypokalemia. The hypocalcaemia was aggravated by metabolic alkalosis due to gastric drainage. A diagnosis of heart failure secondary to hypocalcaemia from idiopathic hypoparathyroidism was established after excluding other aetiologies. Treatment focusing on heart failure management and calcium correction led to significant clinical improvement, and the patient was successfully extubated. This case highlights the importance of measuring serum calcium in all patients presenting with heart failure, as hypocalcaemia is a treatable cause. A comprehensive evaluation of endocrine and metabolic factors is essential in young patients with unexplained heart failure for accurate diagnosis and effective management.

Keywords: Hypocalcaemia, Metabolic alkalosis, Parathyroid hormone

CASE REPORT

A 28-year-old female presented to the surgical outpatient department with abdominal pain typical of appendicitis, which was initially around the umbilicus and then radiated to the right iliac fossa over the past day. She had no significant previous medical history. On examination, guarding and rigidity were present. Ultrasonography suggested appendicitis. Preoperative investigations, including echocardiography, chest roentgenogram, and erect abdominal X-ray, were normal. Laboratory reports were normal except for anaemia, with a haemoglobin level of 9 g/dL. She underwent an appendectomy under general anaesthesia on the same night. One unit of Packed Cell Volume (PCV) was transfused during the operative procedure. The patient tolerated the procedure well, and the surgical specimen was sent for histopathological examination. Following surgery, she was on nil by mouth, and aspiration was performed every two hours via a gastric tube. About 16 hours postsurgery, she developed acute onset breathlessness and orthopnoea. Her oxygen saturation was 50%, pulse rate was 90 beats per minute, blood pressure was 80/60 mmHg, respiratory rate was 22 breaths per minute, and temperature was 98.4°F. On auscultation, bilateral crepitations were present. A chest X-ray revealed bilateral homogeneous opacities likely suggestive of pulmonary oedema [Table/Fig-1].

The patient was intubated, and invasive ventilation was initiated. Intravenous antibiotics, vasopressor support, diuretics, and hydrocortisone were administered, and she was subsequently shifted to the medicine unit. An electrocardiogram revealed non-specific ST-T changes in the inferior and lateral leads. Cardiac enzymes were normal. Blood gas analysis revealed metabolic alkalosis and hypokalemia. On day 3, a repeat chest X-ray showed some resolution of pulmonary oedema [Table/Fig-2].

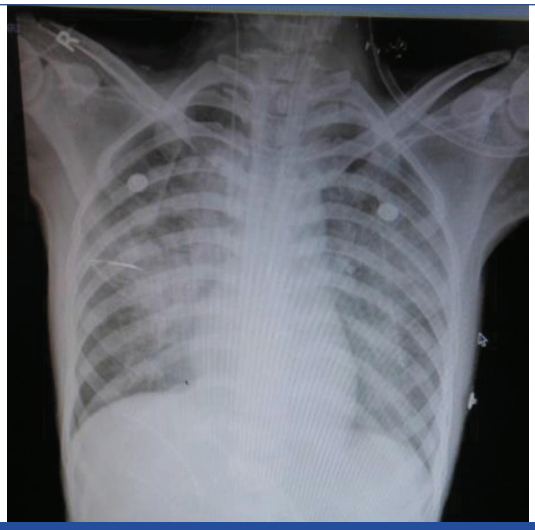


[Table/Fig-1]: Chest radiograph showing bilateral homogenous opacities.

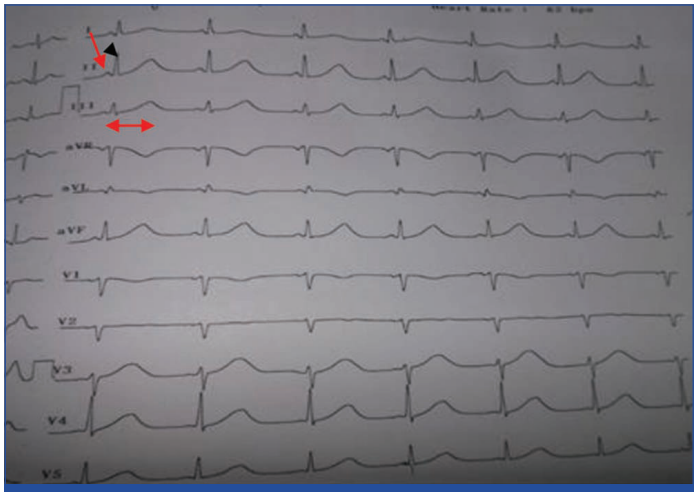
The patient developed carpal spasm the following day, which was treated with intravenous calcium gluconate. Serum calcium was found to be decreased, measuring 7.6 mg/dL. The patient experienced carpal spasm two more times on the same day. A 10-minute infusion of 1 g (10 mL) of calcium gluconate relieved both instances. The electrocardiogram revealed changes consistent with hypocalcaemia (short PR interval and prolonged QT interval) [Table/Fig-3]. NT-pro BNP level was elevated at 1120 pg/mL.

Persistent hypokalemia and hypocalcaemia were treated with calcium gluconate and potassium chloride. Meanwhile, the patient improved, was weaned off mechanical ventilation and pressor support, and was extubated.

Histopathological examination of the surgical specimen showed chronic appendicitis. The patient's past history revealed that she had an uneventful pregnancy five years ago. During her pregnancy, she



[Table/Fig-2]: Repeat chest radiograph showed minor resolution of pulmonary oedema.



[Table/Fig-3]: An ECG showing changes of hypocalcaemia (short PR interval and prolonged QT interval).

was taking calcium supplements and haematinics, with no history of co-morbidities. A laboratory work-up for hypocalcaemia was conducted [Table/Fig-4], which revealed low PTH levels, while serum albumin, serum creatinine, and thyroid profile were normal.

Test	Level	Normal levels
Urinary calcium (millimoles/day)	0.5	2.5-7.5
Parathyroid Hormone (PTH) (pg/mL)	8.76	10-60
25(OH) vitamin D (ng/mL)	4.63	21-100
Magnesium (mg%)	2	1.6-2.2
Amylase (U/L)	30	30-118
Lipase (U/L)	83.3	13-60
Serum calcium (c) (mg%)	7.6	9-11
Serum phosphorus (mg%)	6.4	2.5-4.5

[Table/Fig-4]: Laboratory tests for hypocalcaemia.

A hypokalemia work-up was also performed [Table/Fig-5]. Renal loss of potassium was ruled out as urinary potassium loss was low. The hypokalemia was attributed to low intake and loss of gastrointestinal fluid due to aspirate loss through the Ryle's tube drainage. Arterial blood gas analysis revealed metabolic alkalosis [Table/Fig-6]. The 2D echocardiography showed global left ventricular hypokinesia with an ejection fraction of 25-30% [Table/Fig-7].

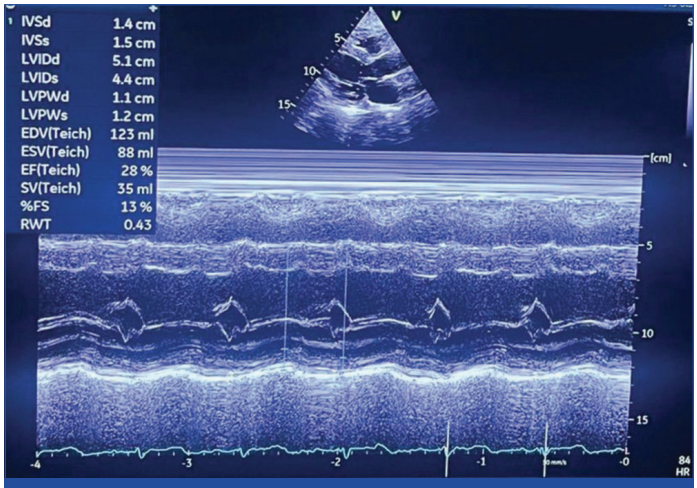
Thus, the case was diagnosed as acute onset cardiac failure and cardiogenic shock due to hypocalcaemia due to idiopathic hypoparathyroidism, precipitated by metabolic alkalosis caused by continuous gastric aspiration. At the time of discharge, the patient was prescribed tablet sacubitril/valsartan 50 mg twice a

Test	Level	Normal level
Urinary K (mEq/day)	12	25-100
Plasma K (mEq/L)	2.2	3.5-5
Plasma osmolality (mosmol/kg)	264.1	285-295
Urine osmolality (mosmol/kg)	300.1	300-900
Trans tubular potassium gradient	4.8	4-8

[Table/Fig-5]: Laboratory tests for hypokalemia.

Arterial blood gas	Value	Normal level
pH (Potential of Hydrogen)	7.48	7.35-7.45
$PaCO_2$ (Partial pressure of carbon dioxide) (mmHg)	36	35-45
PaO_2 (Partial pressure of oxygen) (mmHg)	90	80-100
HCO_3 (Bicarbonate) (mEq/L)	30	22-26

[Table/Fig-6]: Arterial blood gas analysis.



[Table/Fig-7]: The 2D ECHO showed global left ventricular hypokinesia with reduced ejection fraction.

day, hydrochlorothiazide 25 mg/day, spironolactone 20 mg/day, and alfacalcidol 0.5 mcg once a day. During routine check-ups, the patient's calcium levels improved.

DISCUSSION

Hypocalcaemia affects cardiac function by decreasing cardiac contractility and causing a decline in left ventricular work, stroke index, and cardiac index. The causes of hypocalcaemia with low PTH levels can be post-thyroidectomy hypoparathyroidism, hypomagnesemia, or idiopathic hypoparathyroidism (where there is no family history and the cause is unknown) [1]. In this case, since magnesium levels were normal and the patient had no history of thyroidectomy, hypocalcaemia was diagnosed as idiopathic hypoparathyroidism [2,3]. Clinical signs and symptoms of hypocalcaemia are varied and affect almost all major physiological systems, including the respiratory, neurological (such as fatal convulsions), cardiovascular, and renal systems [4]. When serum calcium falls below 8.8 mg/dL, PTH output rises proportionately, releasing more calcium from the bone. Through negative feedback, PTH regulates its own synthesis and encourages the kidneys to produce 1,25(OH)2D, which in turn drives the mobilisation of calcium from the gut and bone. In hypoparathyroidism, low calcium levels do not cause an increase in PTH; thus, low vitamin D levels result [5].

The physiologically active component of blood calcium is free-ionised calcium. Bound hydrogen ions separate from albumin when blood becomes alkaline, allowing albumin to bind with more calcium and lowering the amount of total serum calcium that is freely ionised. The cerebral vasoconstriction that results in common symptoms such as light-headedness, fainting, and paraesthesia is partly caused by this hypocalcaemia associated with alkalosis [6].

Serum calcium is known to play a role in the contraction and stimulation of cardiac muscle fibers. The quantity of cytosolic calcium

that can bind to troponin C increases when external calcium is added to that from the activated sarcoplasmic reticulum [7]. Muscle contraction results from this interaction with the troponin-tropomyosin complex, which creates cross-bridges between actin and myosin [8]. Despite the fact that the kinetics of intracellular calcium are clearly linked to muscle contraction and relaxation, the mechanism of myocardial dysfunction that results from hypocalcaemia remains not entirely clarified. However, calcium plays a role in cardiomyocyte repolarisation; hypocalcaemia is thought to cause the QT interval to lengthen and the T-wave to invert, which is what occurred in this instance. Therefore, the lack of efficacy of a single conventional medication for heart failure (such as furosemide, which may even exacerbate hypocalcaemia) and the fact that ejection fraction and QT interval can be restored with the correction of hypocalcaemia may help explain the features of hypocalcaemic cardiac failure [9].

Gastric aspiration leads to the loss of hydrogen ions and chloride, causing metabolic alkalosis. This results in low chloride and high bicarbonate levels, elevated CO_2, and increased blood pH. The body compensates by retaining bicarbonate, which further worsens the alkalosis. Additionally, potassium shifts into cells, causing hypokalemia, which can lead to muscle weakness and arrhythmias. In patients with cardiac failure, calcium levels should be monitored, and they should be evaluated for other causes, including metabolic and endocrine factors. One should replace gastric aspirate with Ringer's lactate to avoid hypokalemia. Other studies have also revealed similar results, indicating that hypocalcaemia is one of the reversible causes of cardiac failure [Table/Fig-8] [10-12].

Author's name	Publication year	Age and Gender	S. Calcium and S. Parathyroid Hormone (PTH) levels	Diagnosis	Treatment
Rupasinghe N et al., [10]	2024	54/F	Serum calcium 2.0 mg/dL Parathyroid hormone 2 pg/mL	Dilated cardiomyopathy due to hypocalcaemia	Calcium and vitamin D replacement therapy given.
Kharel M et al., [11]	2024	55/F	Total Calcium: 3.5 mg/dL Parathyroid hormone <1 pg/L	Hypocalcaemic cardiomyopathy with heart failure	Corrected hypocalcaemia and vitamin D supplementation.
Wang C et al., [12]	2021	65/F	Serum calcium 1.33 mmol/L Parathyroid hormone <1.0 pg/mL	Reversible congestive heart failure associated with hypocalcaemia	Calcium gluconate infusion.
Present study	2025	28/F	Serum calcium 7.6 mg% Parathyroid hormone 8.76 pg/mL	Cardiac failure secondary to idiopathic hypoparathyroidism	Calcium gluconate i.v. with oral calcium supplements.

[Table/Fig-8]: Similar cases from the literature [10-12].

CONCLUSION(S)

Hypoparathyroidism with severe hypocalcaemia is a significant and reversible cause of heart failure. Prompt recognition and treatment with calcium are essential, as mismanagement with loop diuretics can worsen hypocalcaemia and lead to life-threatening complications. Recognising metabolic causes is crucial for evaluating unexplained cardiomyopathy or heart failure.

REFERENCES

[1] Bastepe M, Gensure RC. Hypoparathyroidism and Pseudohypoparathyroidism. [Updated 2024 May 8]. In: Feingold KR, Anawalt B, Blackman MR, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK279165/.

[2] Suzuki T, Ikeda U, Fujikawa H, Saito K, Shimada K. Hypocalcaemic heart failure: A reversible form of heart muscle disease. Clin Cardiol. 1998;21(3):227-28.

[3] Gulden T, Yahyavi SK, Lodding IP, Jensen JEB, Blomberg Jensen M. Severe hypocalcaemia due to hypoparathyroidism associated with HIV: A case report. Bone Rep. 2021;15:101119.

[4] Xie Y, Yang J, Li K, Liu F, Yi Y, Zhang P. Case report: A case of hypocalcaemic cardiomyopathy with non-reversible cardiac fibrosis. Front Cardiovasc Med. 2023;10:1166600.

[5] Lofrese JJ, Basit H, Lappin SL. Physiology, Parathyroid. [Updated 2023 Jul 17]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482510/.

[6] Tinawi M. Pathophysiology, evaluation, and management of metabolic alkalosis. Cureus. 2021;13(1):e12841. Doi: 10.7759/cureus.12841.

[7] Abate EG, Clarke BL. Review of hypoparathyroidism. Front Endocrinol (Lausanne). 2017;7:231563.

[8] Sadiq NM, Naganathan S, Badireddy M. Hypercalcaemia continuing education activity. [Updated 2024 May 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430714/.

[9] Wen Y, Luo X. Hypocalcemic cardiomyopathy: A case report. Front Cardiovasc Med. 2022;9:999550. Doi: 10.3389/fcvm.2022.999550.

[10] Rupasinghe N, Ranasinghe P, Wanninayake L. Dilated cardiomyopathy due to hypocalcaemia: A case report. J Med Case Rep [Internet]. 2024 Dec 1 [cited 2024 Nov 19];18(1):01-04. Available from: https://jmedicalcasereports.biomedcentral.com/articles/10.1186/s13256-024-04505-3.

[11] Kharel M, Subedi A, Hossain MF. Hypocalcaemic cardiomyopathy with heart failure: A rare case report. Clin Case Rep [Internet]. 2024 Sep 1 [cited 2024 Nov 16];12(9):e9463. Available from: https://onlinelibrary.wiley.com/doi/full/10.1002/ccr3.9463.

[12] Wang C, Dou LW, Wang TB, Guo Y. Reversible congestive heart failure associated with hypocalcaemia: A case report. World J Clin Cases [Internet]. 2021 [cited 2024 Nov 16];9(34):10659. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC8686125/.

PARTICULARS OF CONTRIBUTORS:

1. Associate Professor, Department of General Medicine, Dr. D. Y. Patil Medical College and Research Institute, Pune, Maharashtra, India.
2. Junior Resident, Department of General Medicine, Dr. D. Y. Patil Medical College and Research Institute, Pune, Maharashtra, India.
3. Professor and Head, Department of General Medicine, Chatrapati Sambhaji Maharaj Government Medical College, Satara, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Sreevidya Yekkaluru,
87/510, Prem Nagar Colony, Kurnool, Andhra Pradesh, India.
E-mail: sreevidyayekkaluru@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Sep 07, 2024
- Manual Googling: Jan 04, 2025
- iThenticate Software: Jan 07, 2025 (5%)

ETYMOLOGY: Author Origin

EMENDATIONS: 8

AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Sep 02, 2024**
Date of Peer Review: **Nov 02, 2024**
Date of Acceptance: **Jan 09, 2025**
Date of Publishing: **Feb 01, 2025**