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CASE REPORT

Persistent QTc Prolongation Due to Hypocalcemia from Hypoparathyroidism in a Heart Failure Patient: A Case Report

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ABSTRACT

The QT interval on an electrocardiogram reflects ventricular depolarization and repolarization, with prolonged QTc indicating a defect in myocardial repolarization. This condition increases the risk of arrhythmias, torsade's de pointes, and sudden cardiac death. We present the case of a 40-year-old female with heart failure and reduced ejection fraction who exhibited persistent QTc prolongation due to hypocalcemia resulting from hypoparathyroidism secondary to a thyroidectomy performed 15 years prior. Management included standard heart failure treatment alongside active vitamin D and calcium supplementation, leading to clinical improvement. This case underscores the importance of early identification and management of secondary causes of QTc prolongation in heart failure patients.

Keywords: Hypocalcemia, QTc prolongation, Heart failure with reduced ejection fraction, Hypoparathyroidism.

Authors' Contribution:
[1,2]Conception; Literature research; manuscript design and drafting; [3,4] Critical analysis and manuscript review; [5,6]Data analysis; Manuscript Editing.

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Introduction

Prolonged QT interval on an electrocardiogram indicates delayed myocardial repolarization.[1] Hypocalcemia can reduce myocardial contractility and extend the QT interval.[2] Prolonged QT can be congenital or acquired, with common acquired causes including medication and metabolic abnormalities such as hypocalcemia.[3] Early detection and calcium supplementation can often reverse this condition.[4] Post-operative hypoparathyroidism, a potential complication of thyroidectomy, can lead to persistent hypocalcemia. This report details a case where a patient developed symptoms of hypoparathyroidism 15 years after thyroid surgery, presenting with heart failure and electrolyte abnormalities.

Case Presentation

We describe a 40-year-old married female who presented to the cardiology ward with a three-day history of chest pain, shortness of breath, and multiple vomiting episodes. The chest pain was severe and radiated to the left arm, persisting for hours. The patient had a history of heart failure that worsened from NYHA Class II to IV. She had a significant medical history of hypertension and underwent thyroidectomy for Graves' disease 15 years ago. On examination, the patient appeared anxious with notable head titubation's and a visible neck scar. Vital signs showed blood pressure of 110/70 mmHg, pulse rate of 88/min, respiratory rate of 24/min, and oxygen saturation of 95% on room air. Distended jugular venous pressure and

peripheral edema were observed. She experienced carpopedal spasms during her hospital stay, prompting the administration of intravenous calcium gluconate. Signs of Trousseau's and Chvostek's were present. Cardiovascular examination revealed S3 gallop and a pan-systolic murmur consistent with mitral regurgitation, while respiratory examination showed bilateral basal crackles.

Investigations:

- **ECG:** Sinus rhythm with a QTc interval of 643msec, in figure 1.
- **Echocardiography:** Severe hypokinesia of the infer posterior segment and moderately severe mitral regurgitation, with an ejection fraction of 35%. Figure 2, 3
- **Coronary Angiography:** Moderate right coronary artery disease. Figures 4, 5,6.
- **Laboratory Findings:**
    - Serum Calcium: 5.19 mg/dL (normal range: 8.6-10.2 mg/dL)
    - Serum Magnesium: 2.25 mg/dL (normal range: 1.6-2.6 mg/dL)
    - Serum Phosphate: 9.11 mg/dL (normal range: 2.5-4.5 mg/dL)
    - Serum Potassium: 3.52 mmol/L (normal range: 3.5-4.5 mmol/L)
    - PTH: 1.85 pg/mL (normal range: 10-65 pg/mL)
    - Thyroid Function Tests: T3, T4, and TSH levels within specified ranges.
    - High-sensitivity Troponin-T: 12.8 ng/L.

Given her history and laboratory findings, the prolonged QT interval was attributed to hypocalcemia due to hypoparathyroidism.

Management:

The patient received IV calcium gluconate and magnesium sulfate to correct hypocalcemia, along with standard heart failure treatment (ARNIs, IV diuretics, SGLT2 inhibitors, spironolactone, and ivabradine) to manage her heart failure. Anti-emetics were provided to control vomiting.

Continuous ECG monitoring was conducted, and improvements in QT interval were noted after initiating active vitamin D3 (calcitriol) to enhance calcium absorption. Upon discharge, the patient was prescribed oral calcium, active vitamin D supplements, and ongoing heart failure management. Follow-up after one month showed improvement in left ventricular ejection fraction to 45% shown in Figure 8,9 and serum calcium levels increased to 7.61 mg/dL, with QTc reduced to 539 msec shown in Figure 8,9.

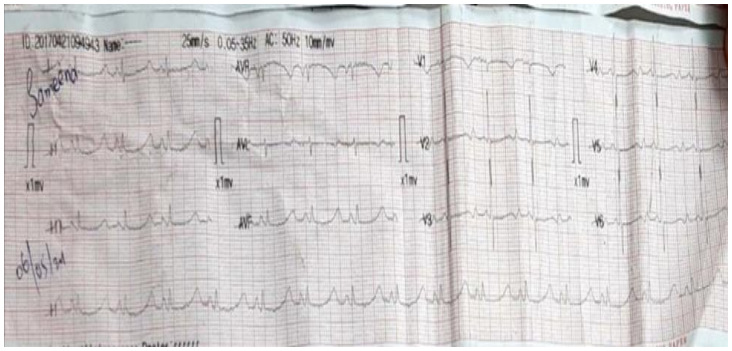

Figure 1: The Electrocardiogram On admission shows sinus rhythm with prolonged QTc of 643msec and biphasic T waves in the anterior chest leads

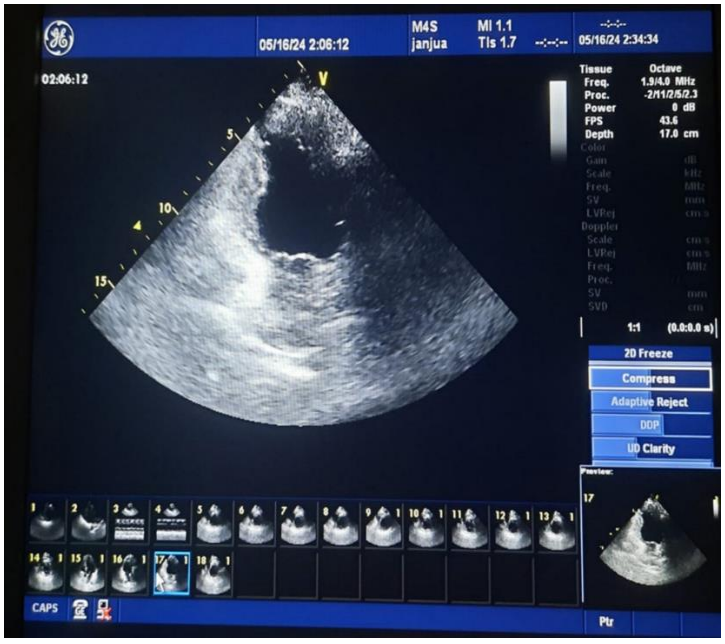

Figure 2: The echography on admission; Parasternal short axis view showing hypokinesia of inferno posterior segment with aneurysm formation.

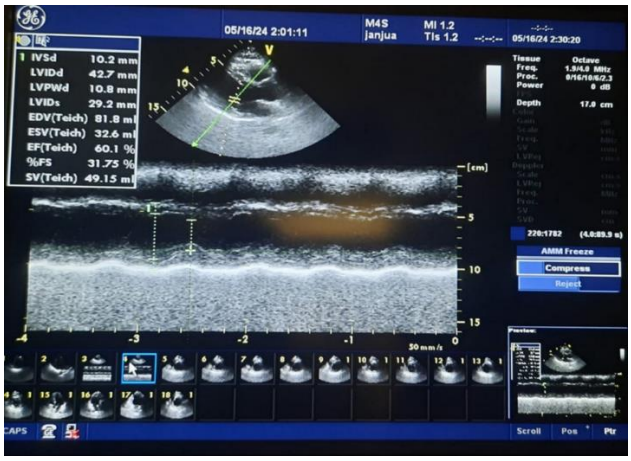

Figure 3: PLAX view depicting LV dimensions

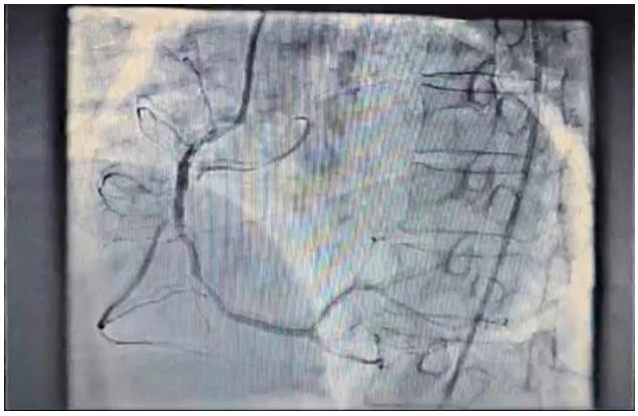

Figure 4: The Right coronary artery is a dominant vessel, moderate proximal disease followed by a long segment of moderate mid-disease.

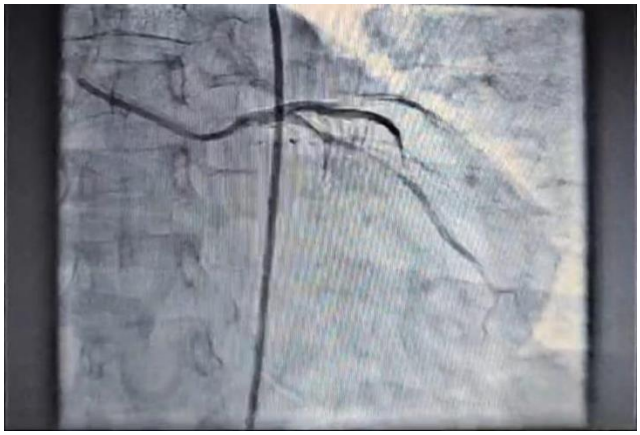

Figure 5: Left anterior descending artery findings; Good sized vessel, mild mid-disease. A fair-sized diagonal branch shows moderate ostial disease.

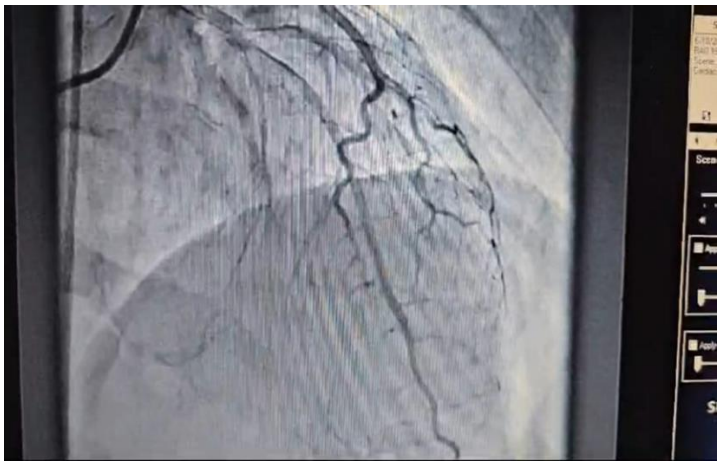

Figure 6: Left circumflex artery is a non-dominant artery with, mild proximal disease.

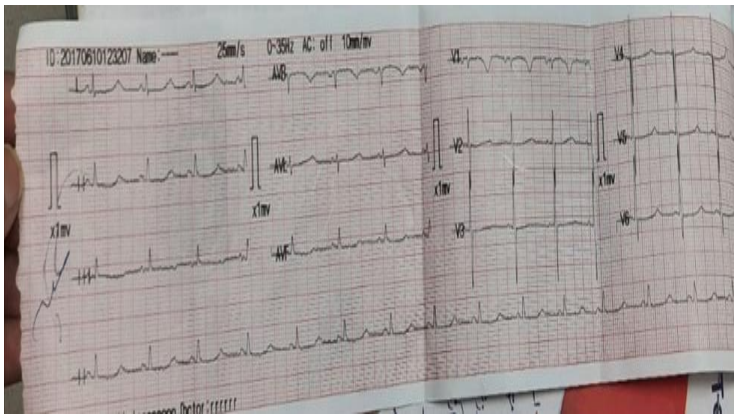

Figure 7: This electrocardiogram after one month shows sinus rhythm with QTc 539msec and t wave flattening in lead V2 to V4

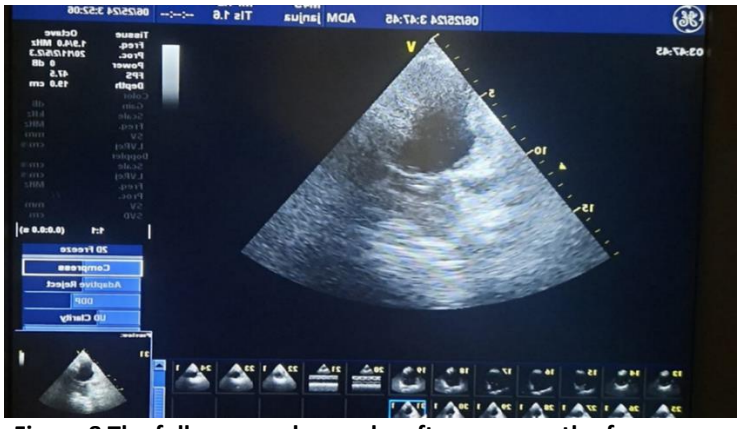

Figure 8 The follow-up echography after one month of treatment; Parasternal short axis view showing improvement in left ventricular ejection fraction.

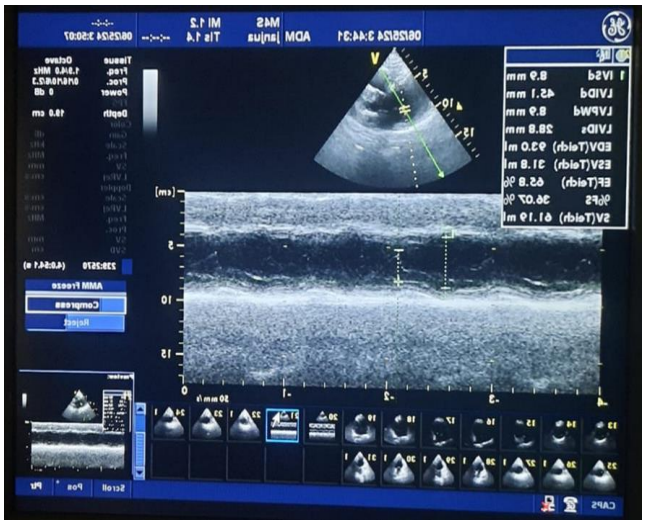

Figure 9: The echocardiography showing LV dimensions after one month of treatment.

Discussion

This case encourages us to detect immediate electrolyte imbalance in patients with cardiovascular diseases to prevent early dreadful arrhythmia, hypocalcemia, though rare can further aggregate the cardiac complications in patients with underlying heart failure.[5] Immediate Ca-level correction is essential in preventing arrhythmia and improving patient outcomes.[6]

The causes of acquired QT prolongation include medications, electrolyte imbalances, and medical conditions. Electrolyte abnormalities can either be the primary cause of QT prolongation or exacerbate an underlying long QT syndrome. In this case, the patient's prolonged QT interval prompted an electrolyte analysis, revealing hypocalcemia, which is known to elongate the ST segment and prolong the QT interval.[7] During an outpatient cardiology evaluation for QT prolongation, the basic electrolyte analysis led to the diagnosis of hypoparathyroidism. Hypocalcemia associated with hypoparathyroidism also causes reversible acquired QT prolongation, as documented in previous case reports.[8,9]

This case report introduces clinical data demonstrating a connection between QTc prolongation and serum calcium levels. Hypocalcemia rarely occurs in isolation, so identifying the underlying cause of disturbed calcium homeostasis is crucial, considering factors such as hormonal imbalances, acid-base disturbances, renal issues, and iatrogenic factors.[10] While most of the body's calcium is stored in the skeletal system or bound to albumin in plasma, free calcium levels are tightly regulated by parathyroid hormone (PTH). These calcium gradients across cell membranes are essential for muscle, neuron, and myocyte excitability. Myocardial contraction relies heavily on extracellular calcium, prolonged and severe hypocalcemia can lead to heart failure.[11]

One significant cause of hypocalcemia is thyroid surgery, which poses the risk of damaging or removing the parathyroid glands. This can result in hypoparathyroidism and subsequent hypocalcemia.[12] Severe symptoms of hypocalcemia include confusion, muscle spasms, numbness in the hands, feet, and face, hallucinations, muscle cramps, brittle nails, and an increased risk of bone fractures. The symptoms of Chvostek's and Trousseau's signs are associated with either the rapid onset of hypocalcemia or severe cases of the condition.[13]

Post-thyroidectomy hypocalcemia can lead to acute cardiomyopathies, which are often reversible with calcium replacement and vitamin D supplementation. This condition is a known cause of QT prolongation, as it extends the plateau phase of the cardiac action potential. This prolonged phase keeps calcium ion channels open for a longer duration, allowing for late calcium inflow, which can result in early afterdepolarizations.

Conclusion

This case underscores the importance of early diagnosis and management of electrolyte imbalances to prevent severe complications in patients with heart failure. A multidisciplinary approach involving endocrinology and cardiology is essential for managing complex conditions affecting both systems.

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