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# Severe Hypercalcemia-Induced Electrocardiographic Abnormalities as the Presenting Manifestation of Multiple Myeloma: A Case Report

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**Abstract** **Case Report**

***Background:*** Severe hypercalcemia may cause neuropsychiatric manifestations, acute kidney injury, and characteristic electrocardiographic (ECG) abnormalities. In older adults, these findings may mimic acute coronary syndromes or primary rhythm disorders, resulting in diagnostic delay. ***Case presentation:*** An 82-year-old woman with cardiovascular risk factors and chronic vitamin D supplementation presented with a 10-day history of agitation and behavioral change, without chest pain, syncope, or dyspnea. After an initial emergency department discharge, persistent symptoms led to re-evaluation where modest troponin elevation and abnormal ECG findings were detected. On admission, she was confused but hemodynamically stable. The ECG demonstrated an irregular non-sinus rhythm with concave ST-segment depression and a markedly shortened corrected QT interval, followed by atrioventricular dissociation with retrograde atrial activation and persistent short QT. Laboratory testing revealed severe hypercalcemia with acute kidney injury and inflammatory markers elevation. Transthoracic echocardiography showed preserved biventricular function without segmental wall motion abnormalities and a small pericardial effusion. The patient underwent intensive intravenous hydration and urgent hemodialysis because of symptomatic severe hypercalcemia with renal dysfunction. Following calcium reduction, the ECG normalized with restoration of sinus rhythm and QT interval lengthening. Etiological workup demonstrated suppressed parathyroid hormone, normal thyroid function, and imaging findings suggestive of malignancy (heterogeneous splenomegaly with multiple masses, adrenal lesions, and osteolytic bone lesions), with further investigations establishing the diagnosis of multiple myeloma as the underlying cause of hypercalcemia. ***Conclusion:*** Hypercalcemia can produce dynamic ECG abnormalities that simulate ischemia or intrinsic conduction disease. Recognizing short QT interval and the reversibility of ECG findings after calcium correction is critical, particularly in elderly patients with neuropsychiatric symptoms. PTH-independent severe hypercalcemia should prompt urgent treatment and evaluation for malignancy, including multiple myeloma in the presence of osteolytic bone lesions.

**Keywords:** Hypercalcemia, Electrocardiogram, Short QT interval, Multiple Myeloma.



## INTRODUCTION

Hypercalcemia is a frequent metabolic disorder encountered in clinical practice, most commonly attributable to primary hyperparathyroidism or malignancy. Severe hypercalcemia constitutes a medical emergency due to its multisystem effects, including neurologic impairment, renal dysfunction, gastrointestinal symptoms, and cardiovascular complications [1]. In patients with cancer, hypercalcemia is usually PTH-independent and may result from humoral mechanisms (parathyroid hormone-related peptide), increased calcitriol production, or osteolytic bone involvement [2]. In multiple myeloma, hypercalcemia is primarily associated with increased osteoclast-mediated bone resorption resulting from tumor-induced disruption of bone remodeling [3].

The electrocardiographic (ECG) signature of hypercalcemia is typically a shortened QT interval, reflecting abbreviation of ventricular repolarization. Additional ECG manifestations may include ST-segment changes and J-point elevation, sometimes described as Osborn-like waves [4]. More severe hypercalcemia may also be associated with delayed cardiac conduction, including PR and QRS prolongation and atrioventricular

conduction abnormalities [5]. These patterns may be confused with acute coronary syndromes, early repolarization, or Brugada-type ECG patterns in patients with severe hypercalcemia [6 5]. Interpretation may be further complicated by elevated cardiac troponin in the setting of non-ischemic myocardial injury or renal impairment [7].

We report a case of severe hypercalcemia presenting with neuropsychiatric disturbance and dynamic ECG abnormalities that resolved after metabolic correction and ultimately revealed multiple myeloma as the underlying cause of hypercalcemia.

## CASE REPORT

An 82-year-old woman was referred to our cardiology department for evaluation of abnormal ECG findings and elevated troponin in the context of acute behavioral changes. Her medical history included long-standing untreated arterial hypertension, osteoarthritis treated chronically with vitamin D supplementation, and remote cholecystectomy (20 years prior). There was no documented history of coronary artery disease, heart failure, diabetes mellitus, or chronic kidney disease. She did not smoke and did not report alcohol use.

The patient developed progressive agitation, irritability, and disorientation over approximately 10 days. There was no chest pain, syncope, presyncope, or dyspnea. No fever or focal neurologic deficits were reported. She initially presented to the emergency department and was treated symptomatically before being discharged on the same day because vital signs were stable. Persistent symptoms prompted reconsultation in a private facility, where laboratory testing reportedly showed a mild troponin elevation and an abnormal ECG, leading to referral for cardiologic assessment.

On admission, the patient appeared confused and disoriented but was hemodynamically stable. Blood pressure was 138/87 mmHg, heart rate 76 beats/min, respiratory rate 18 cycles/min, and oxygen saturation 97% on room air. She was afebrile. Physical examination revealed no clinical signs of heart failure, no respiratory distress, and no focal neurologic deficits. Peripheral pulses were palpable and symmetric.

The initial ECG performed at the referring facility demonstrated an irregular non-sinus rhythm with bradycardia (approximately 52 beats/min), concave ST-segment depression (inferior and lateral leads), and a markedly shortened corrected QT interval (QTc approximately 335 ms) (Figure 1). The admission ECG in our department demonstrated AV dissociation with retrograde P waves, a ventricular rate around 57 beats/min, persistent repolarization abnormalities, and continued QT shortening (QTc approximately 350 ms) (Figure 2). These findings raised diagnostic concern for non-ST-elevation acute coronary syndrome; however, the short QT interval and atypical ST morphology suggested a metabolic etiology.

Laboratory investigations showed severe hypercalcemia with acute kidney injury. Total serum calcium was markedly elevated (155 mg/L) with normal serum albumin. Mild hyponatremia was present. Renal function was impaired, with an estimated glomerular filtration rate of approximately 19 mL/min/1.73 m². Inflammatory markers were elevated, and mild hepatic cytolysis was noted. Cardiac troponin was elevated but remained modest relative to the ECG appearance and clinical presentation.

Transthoracic echocardiography demonstrated preserved left ventricular systolic function (ejection fraction approximately 60%), no segmental wall-motion abnormalities, no significant valvular disease, and a small pericardial effusion without hemodynamic compromise. Right ventricular size and function were preserved and there was no evidence of pulmonary hypertension.

Given the severe hypercalcemia and renal dysfunction, etiological evaluation was initiated. Parathyroid hormone was suppressed, supporting PTH-independent hypercalcemia. Thyroid-stimulating hormone and vitamin D levels were within normal limits, making thyrotoxicosis and vitamin D intoxication unlikely.

Abdominal ultrasonography revealed heterogeneous splenomegaly with multiple focal masses. Thoracoabdominopelvic computed tomography revealed adrenal lesions and osteolytic bone lesions, raising strong suspicion for an underlying malignant process as the cause of hypercalcemia of malignancy. Further hematological investigations subsequently established the diagnosis of multiple myeloma as the underlying cause of the severe hypercalcemia.

The patient was managed as a hypercalcemic emergency. Intensive intravenous hydration with isotonic saline was initiated to correct intravascular volume and promote calciuresis. Due to symptomatic severe hypercalcemia with acute kidney injury, urgent hemodialysis was performed using an appropriate dialysate calcium concentration.

After dialysis and partial correction of calcium levels, the patient's mental status improved. ECG monitoring demonstrated restoration of sinus rhythm and lengthening of the QT interval (QTc approximately 480 ms) (Figure 3). The dynamic reversal of ECG abnormalities in parallel with calcium reduction supported the diagnosis of hypercalcemia-induced ECG changes rather than primary ischemia or intrinsic conduction disease.

The recurrence tendency of hypercalcemia despite initial correction reinforced the diagnosis of hypercalcemia of malignancy secondary to multiple myeloma, and further oncologic evaluation, including tissue diagnosis strategy, was planned.

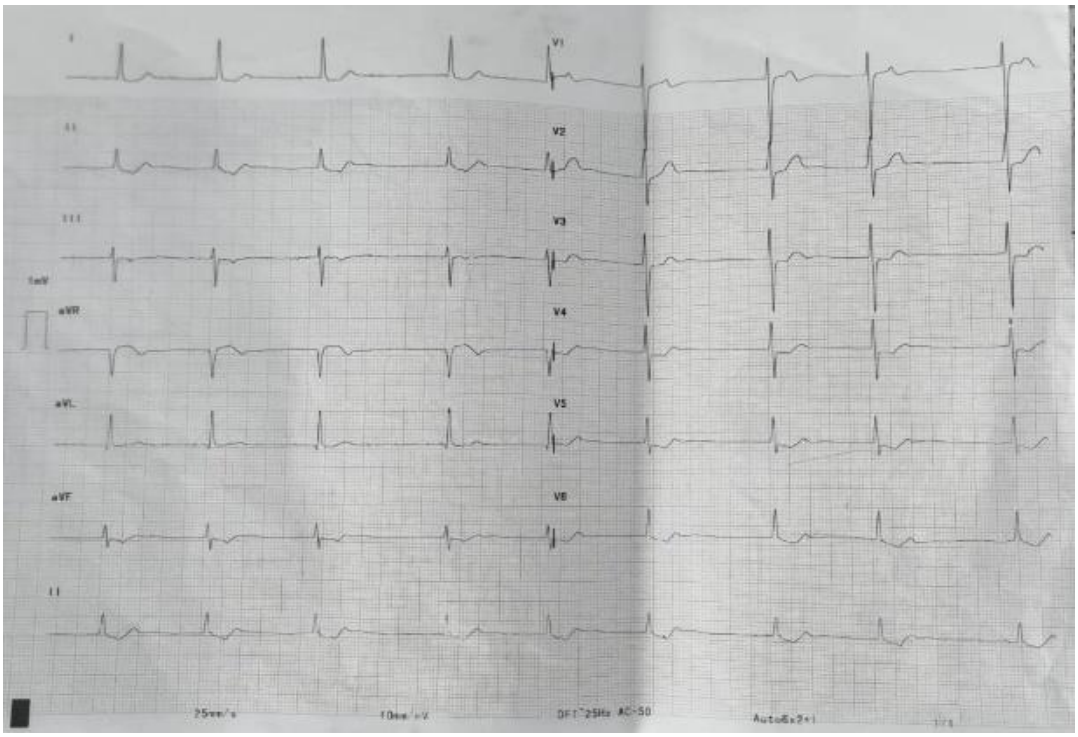


**Figure 1: ECG performed at the referring facility showing an irregular non-sinus rhythm with bradycardia (≈52 beats/min), concave ST-segment depression in the inferior and lateral leads, and marked QTc shortening (≈335 ms)**

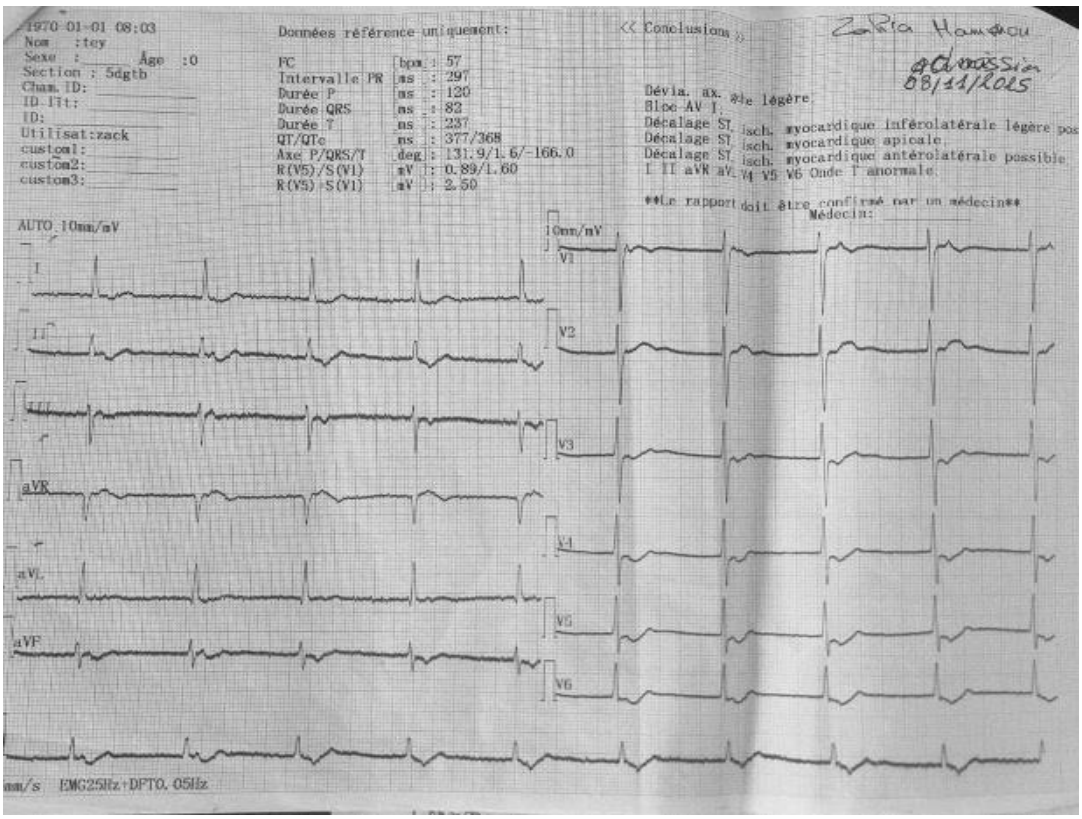


**Figure 2: Admission ECG at our department demonstrating atrioventricular dissociation with retrograde P waves, a ventricular rate of approximately 57 beats/min, persistent repolarization abnormalities, and marked QTc shortening (≈350 ms)**

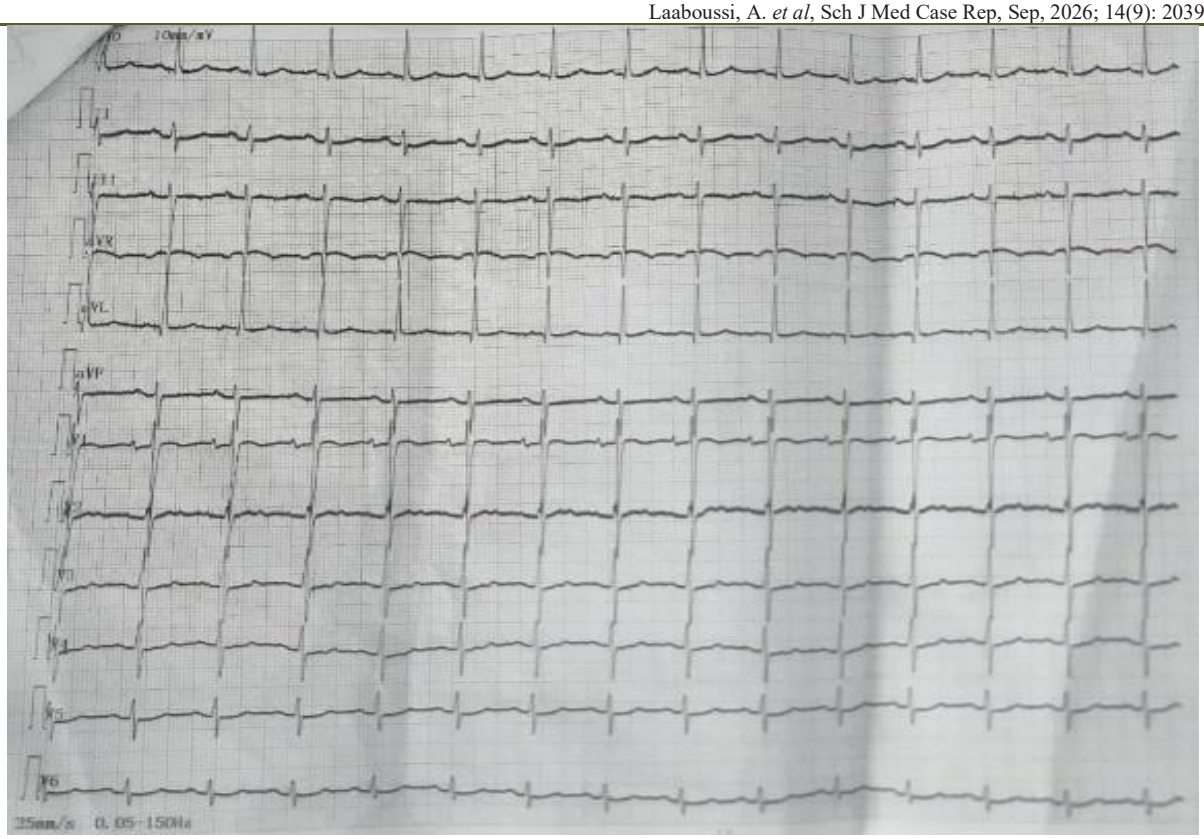

**Figure 3 : Post-dialysis ECG demonstrating restoration of sinus rhythm and marked prolongation of the QTc interval (≈480 ms) following partial correction of serum calcium levels**

## DISCUSSION

This case illustrates the importance of recognizing hypercalcemia-induced ECG abnormalities in an elderly patient presenting with neuropsychiatric symptoms. Severe hypercalcemia can produce a spectrum of ECG manifestations. The most consistent finding is QT interval shortening, largely due to abbreviation of the ST segment [4]. In more severe cases, atrioventricular (AV) conduction abnormalities and junctional rhythms may occur, potentially leading to AV dissociation and bradyarrhythmias [8]. These findings can be misinterpreted as ischemia or intrinsic conduction disease, particularly when ST-segment abnormalities and troponin elevation are present [9].

Troponin elevation in severe metabolic disturbances may reflect myocardial injury related to supply–demand imbalance or other non-coronary mechanisms, while elevated troponin levels are also common in patients with renal impairment and may complicate the interpretation of acute coronary syndromes [7]. In patients with atypical clinical features and no echocardiographic evidence of regional wall-motion abnormalities, metabolic and electrolyte disturbances, particularly calcium abnormalities, should be considered as potential contributors to ECG and troponin abnormalities to avoid misdiagnosis and unnecessary antithrombotic or invasive coronary interventions [9 7].

The presence of suppressed PTH strongly indicates PTH-independent hypercalcemia, for which malignancy is an important cause. In this patient, the presence of osteolytic bone lesions, together with severe hypercalcemia, raised suspicion of an underlying malignant process, and further investigations established the diagnosis of multiple myeloma. Hypercalcemia is a recognized myeloma-defining event and is generally related to increased bone resorption associated with the myeloma bone disease [3]. Hypercalcemia of malignancy is associated with advanced disease and poor prognosis, emphasizing the importance of early recognition and treatment [2].

From a management standpoint, the initial treatment of severe hypercalcemia consists of intravenous volume expansion with isotonic fluids, when clinically tolerated. Antiresorptive therapy with an intravenous bisphosphonate or denosumab is recommended for hypercalcemia of malignancy, while calcitonin may be added in severe cases because of its rapid but transient effect [10]. In patients with severe renal dysfunction, refractory hypercalcemia, or contraindications to standard medical therapy, hemodialysis with a low-calcium dialysate may provide rapid correction and can be life-saving [2].

## CONCLUSION

Severe hypercalcemia can cause neuropsychiatric symptoms and dynamic ECG

abnormalities that mimic acute coronary syndromes and conduction system disease. A markedly shortened QT interval is a key clue. Rapid correction of calcium levels may normalize rhythm and repolarization changes, potentially preventing fatal arrhythmias. In PTH-suppressed severe hypercalcemia, clinicians should urgently evaluate for malignancy, including multiple myeloma, particularly in the presence of osteolytic bone lesions, as early diagnosis and targeted treatment are critical for prognosis.

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**Declaration of competing interest**
The authors declare no competing interests.

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**Patient consent**
Written informed consent for publication of this case report was obtained from the patient's legal representatives in accordance with institutional policy.

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