Received: 17 June 2024 | Revised: 15 October 2024 | Accepted: 6 November 2024

DOI: 10.1002/jgf2.754

Journal of General and Family Medicine Open Access WILEY

CASE REPORT

Extreme hypercalcemia secondary to parathyroid adenoma obscured by excessive coffee consumption and premature ventricular complexes

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Abstract

Severe hypercalcemia has an arrhythmogenic effect. We present a case of a young male falsely diagnosed with premature ventricular complexes attributed to heavy coffee consumption, while symptoms and electrocardiographic findings of hypercalcemia were disregarded. Delayed work-up identified extreme hypercalcemia, and subsequently, parathyroid adenoma was diagnosed. Medical and surgical management resulted in complete resolution. The misdiagnosis led to a substantial diagnostic delay.

KEYWORDS
coffee consumption, diagnosis bias, electrocardiogram, hypercalcemia, premature ventricular complexes

1 | INTRODUCTION

Despite the technological advancement that usher in a new era of modern medicine, diagnosis bias is unfortunately part of clinical practice, as physicians miss or fail to evaluate some information provided or to include the range of diagnostic possibilities.[1] The classic presentation of hypercalcemia is often memorized by the mnemonics 'moans, bones, stones and groans', referring to the gastrointestinal, musculoskeletal, renal, and psychiatric manifestations.[2] It is evident that hypercalcemia, particularly in severe cases, may elicit a wide variety of cardiac arrhythmias including bradycardia, sinus arrest, ectopic beats, and in rare forms ventricular fibrillation.[3] We present an unusual case of severe hypercalcemia and primary hyperparathyroidism in a young male initially diagnosed as suffering from symptomatic premature ventricular complexes (PVCs) related to heavy coffee consumption.

2 | CASE PRESENTATION

An otherwise healthy man in his late 40s presented to the outpatient clinic with the chief complaint of episodic palpitations during the past 2 weeks. He had no personal nor family history of hypertension or diabetes, and there was no personal or family history nephrolithiasis. He takes no herbal, prescriptions or over-the-counter medications. On review of systems, he reported fatigue and daytime somnolence in the recent month that was managed by drinking 9–10 cups of coffee daily. The patient declined the consumption of energy drinks, other caffeinated products, alcohol use, or illicit substance abuse. He noticed being nauseous, feeling thirsty with more frequent urination than usual, all of which he related to heavy coffee consumption, since all the complaints appeared roughly in the same time frame. His physical examination, vitals and glucose finger stick were unremarkable, except for a regularly irregular heart sounds. A 12-lead electrocardiogram (ECG) was obtained (Figure 1) and the diagnosis of symptomatic PVCs was given. He was reassured and instructed to avoid coffee, other caffeinated beverages and to perform blood tests. Five days later, he was referred to the hospital due to "abnormal test results". Upon arrival, clinical examination was unremarkable and the ECG was similar to previous tracing. Laboratory tests were notable for serum calcium of 20.4 mg/dL (reference, 8.6–10.3 mg/dL), intact parathyroid hormone (PTH) level was 1430 pg/mL (reference, 14–72 pg/mL), serum creatinine was 1.1 mg/dL, and serum phosphate was 2.5 mg/

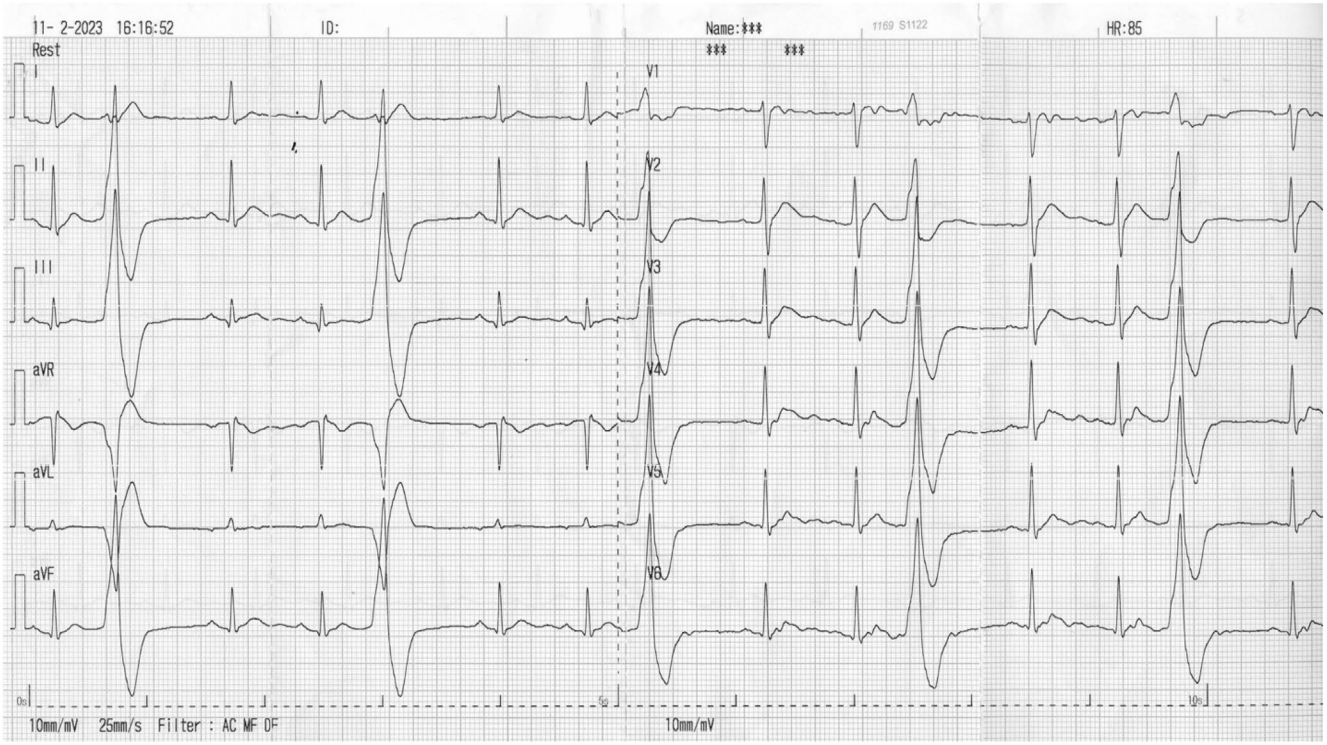

FIGURE 1 ECG obtained in the outpatient clinic showing a sinus rhythm of 78 beats per minute, incomplete right bundle branch block, QT interval of 308 milliseconds, QTc interval of 350 milliseconds, J-point and ST segment elevation, and trigeminy with unifocal PVCs.

dL (reference, 2.8–4.5 mg/dL). Aggressive intravenous fluid resuscitation and pamidronate were initiated with gradual normalization of calcium levels over the next days. A neck ultrasound was conducted, and color Doppler revealed a mass that appears more vascularized than the thyroid parenchyma. Tc-99 m sestamibi scan demonstrated a mass at the posterior lower aspect of the thyroid gland, measured at 14 × 27 millimeters (Figure S1).

The patient underwent parathyroidectomy and a lobular 2.4 g mass with hypercellular morphology was removed, consistent with parathyroid adenoma. The adjacent anatomical structures were uninvolved. Intraprocedural PTH documentation revealed a decrease from 751 ng/L to 102 ng/L after removal of the adenoma. With the resolution of the hypercalcemia, during a 72-h continuous cardiac monitoring PVCs were not observed. The post-operative ECG is shown in Figure 2, with normalization of the corrected QT interval (QTc) and disappearance of the J point elevation. The post-operative period was complicated by mild electrolyte abnormalities including hypocalcemia and hypophosphatemia. During 3 months of follow-up including ambulatory ECG monitoring, he remained asymptomatic.

3 | DISCUSSION

Uniquely in our case, the extreme hypercalcemia was brought into light by symptomatic PVCs, which were our patient main concern. Indeed, review of systems revealed fatigue, polyuria and polydipsia that were falsely attributed to excessive coffee intake. Obviously, the extreme hypercalcemia accounts for these symptoms. The present case emphasizes the fact, that errors attributed to flaws in clinical reasoning might occur. These are most prevalent in the primary healthcare setting, though diagnostic error rates were reported to account for between 5% and 10% of all emergency department clinical decisions.[4]

Arrhythmias from electrolyte imbalance can be life-threatening. Five days went by between the initial ECG recording and hospital admission. During this period, the patient was unattended and could have suffered a fatal arrhythmia. This could have been prevented by a better ECG interpretive skill, data integration and analytic processes.

Various electrocardiographic manifestations of hypercalcemia have been reported. Characteristically, a shortened ST segment, shortened QT, and QTc intervals could be noted. Interestingly, as our case depicted, the QTc interval normalizes upon correction of serum calcium level. Additional findings among patients with hypercalcemia include an increased incidence of J waves and ST segment elevation, Brugada-like ECG pattern, prolongation of the PR and QRS intervals. Repolarization abnormalities may also result in fattened or biphasic T waves and prominent U waves.[5]

Our patient's ECG showed a sinus rhythm of 78 beats per minute, incomplete right bundle branch block, a QT interval of 308 milliseconds, QTc interval of 350 milliseconds, J-point and ST segment elevation, and trigeminy pattern with unifocal PVCs. The acceptable definition of short QT is QTc interval <350 milliseconds for males and 360 milliseconds for females. The differential diagnosis includes the congenital short QT syndrome, hypercalcemia, hyperkalemia, acidosis, hyperthermia, and drug effects (e.g., digitalis).[6]

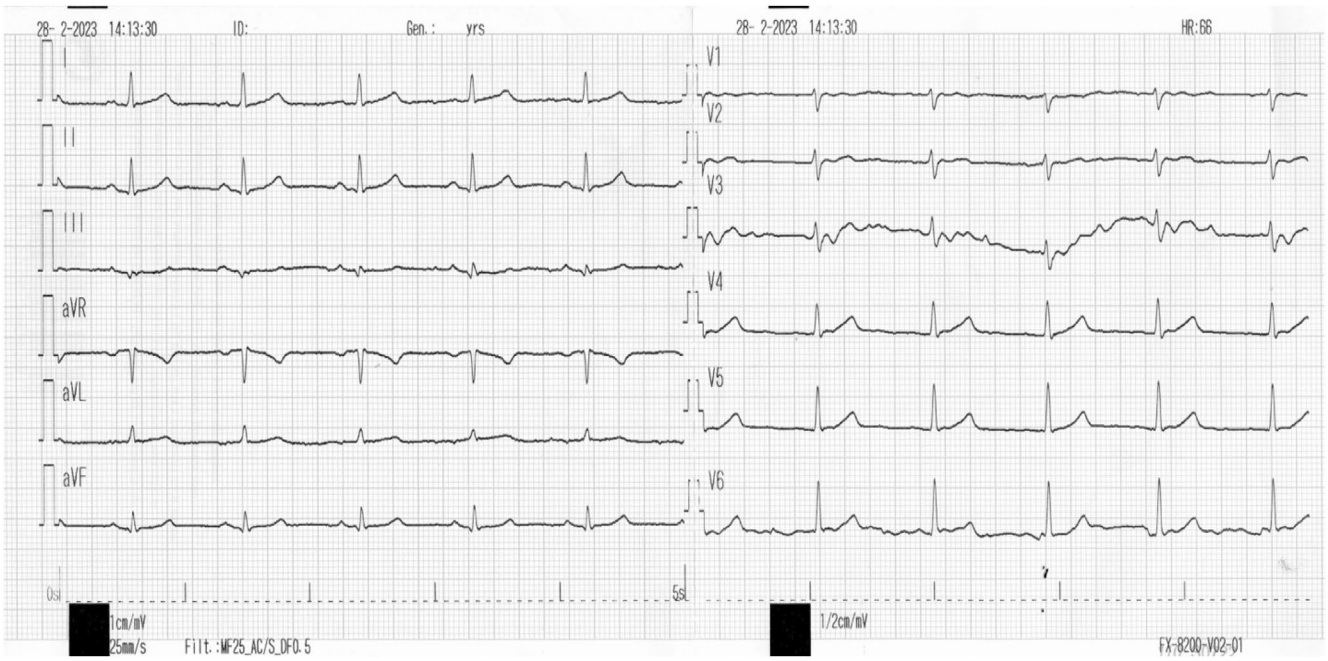

FIGURE 2 ECG recorded post-operatively day 2, showing normalization of the QT interval.

PVCs are not uncommonly encountered in middle-aged men, with a prevalence of 4%–5% on routine ECG exam.[7] Since they are conceived to be relatively benign in nature, PVCs are often disregarded. However, frequent PVCs are associated with increased cardiovascular risk and mortality.[8] The initial assessment should include full history taking, physical examination, and blood analysis for electrolyte abnormalities. In this case, the ECG displayed findings suggestive of hypercalcemia that were overlooked, namely, short QT and J-point elevation. Caffeine is suggested to exert sympathomimetic effects, thereby resulting in elevated plasma levels of catecholamines, and as a result, potentially increasing premature extra beats. Although both patients and clinicians tend to associate consumption of caffeinated products with arrhythmogenesis, there is little data to support this relationship, as most observational and experimental studies were negative.[9] Nevertheless, guidelines recommended to eliminate possible contributing factors, such as caffeine and alcohol as part of the management of premature beats.[10]

Another intriguing point in our case is the extremely elevated calcium and PTH levels. There is a great evidence in the literature suggesting that such an extreme elevation is more commonly encountered with parathyroid cancer and is unusual for adenoma.[11]

4 | CONCLUSION

We present a case of severe hypercalcemia with an unusual presentation initially misattributed to coffee consumption. In patients with PVCs, a careful examination of the ECG for clues indicating underlying pathology is essential. While comprehensive history-taking remains integral in clinical practice, physicians must be cautious of patients' potential bias in rationalizing their symptoms as less severe. It is imperative for physicians to meticulously observe and integrate all findings, including physical examinations and laboratory tests, while maintaining objectivity to minimize the risk of diagnostic errors.

AUTHOR CONTRIBUTIONS

All authors have contributed to the manuscript.

CONFLICT OF INTEREST STATEMENT

The other authors have stated explicitly that here are no conflicts of interest in connection with this article.

ETHICS STATEMENT

Ethics approval statement: None.
Patient consent statement: None.
Clinical trial registration: None.

INFORMED CONSENT STATEMENT

Informed consent was obtained from the patient and is available upon request.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Avidan Y, Kassem S. Extreme hypercalcemia secondary to parathyroid adenoma obscured by excessive coffee consumption and premature ventricular complexes. J Gen Fam Med. 2025;26:263–266. https://doi.org/10.1002/jgf2.754