Case Report

Hypocalcemia and Reversible Cardiomyopathy Assessed by Magnetic Resonance Imaging

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Introduction

Calcium is a fundamental component of biological systems and plays a key role in excitation-contraction coupling in cardiac myocytes. Calcium entry triggers a mechanism known as calcium-induced calcium release, in which calcium released from the sarcoplasmic reticulum is modulated by the influx of calcium from the extracellular space. Muscle contraction subsequently occurs through the interaction between calcium and the troponin-tropomyosin complex. In the heart, intracellular calcium is the primary regulator of contractile force (inotropism).[1]

Hypocalcemia-induced cardiomyopathy is a rare condition with few reports in the scientific literature. We present a clinical case associated with hypoparathyroidism, one of the main causes of chronic hypocalcemia.[2] This case report includes a novel element: the use of cardiac Magnetic Resonance Imaging (cMRI), which, to our knowledge, has not been previously described in the literature.

Case report

A 74-year-old female patient with a history of thyroid cancer underwent total thyroidectomy 10 years ago, resulting in hypoparathyroidism as a consequence of the procedure. Other relevant medical history includes type 2 diabetes and hypertension. Cardiac function was evaluated eight months prior to the current consultation using transthoracic Doppler echocardiography, which reported a left ventricular ejection fraction (LVEF) of 55%. In the three months leading up to the consultation, the patient discontinued her pharmacological treatment with calcium, cholecalciferol, and calcitriol.

The patient complained at the emergency department with exertional dyspnea, asthenia and adynamia. Bilateral edema of the lower limbs and moist rales in the lung fields. She denies history of chest pain, palpitations, infections or exposure to cardiotoxic agents. Chvostek and Trousseau signs were absent.

Keywords

Cardiomyopathies; Hypocalcemia; Case Reports

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A 12-lead electrocardiogram (ECG) was performed (Figure 1), showing sinus rhythm at 80 beats per minute, narrow QRS complexes, diffuse T wave inversions, and a prolonged isoelectric ST segment, with a corrected QT interval (QTc) of 700 ms.

Initial blood tests revealed a serum calcium level of 4 mg/dL (reference range: 8.5–10.2 mg/dL) and an ionized calcium level of 0.61 mmol/L (reference range: 1.1–1.3 mmol/L). NT-proBNP was elevated at 2000 pg/mL (normal <300 pg/mL), while troponins were undetectable (<40 ng/dL). Renal function tests, standard electrolyte panel, complete blood count, and thyroid profile were all within normal limits.

Subsequently, a TTE was performed (Figure 2), showing a left ventricle with normal dimensions, diffuse hypokinesia, a LVEF of 28% (measured by the Biplane Simpson method), and severe secondary mitral regurgitation.

Intravenous and oral calcium supplementation was initiated. Intravenous furosemide was also started, along with bisoprolol (2.5 mg daily), ramipril (2.5 mg daily), and spironolactone (12.5 mg daily). Within seventy-two hours of starting treatment, the patient showed clear clinical improvement. Follow-up blood tests revealed a serum calcium level of 9.5 mg/dL and an ionized calcium level of 1.23 mmol/L. A repeat ECG showed positive T waves and a corrected QT interval (QTc) of 480 ms (Figure 3).

A new TTE revealed a LVEF of 35% and mild mitral insufficiency. All TTEs were performed by the same technician and using the same equipment.

Three days later, a cMRI was performed (Figure 4), showing global hypokinesia of the left ventricle with, a LVEF of 45% and mild mitral insufficiency. T1 mapping was 1061 ms and T2 mapping was 49 ms. Late gadolinium enhancement was observed at the intramyocardial level in the inferior, inferoseptal and inferolateral basal segments, consistent with a non-ischemic pattern. The late enhancement mass measured 4g representing 3.4% of the left ventricular mass.

The patient was discharged on calcium, calcitriol, and vitamin D supplementation, along with treatment for systolic dysfunction. Four weeks later, a TTE was performed, showing a LVEF of 60% (Figure 5).

Discussion

The link between calcium and myocardial contractility was established more than 150 years ago by Dr. Sidney Ringer.[3] During the 20th century, the relationship between serum calcium levels and ventricular function was experimentally demonstrated, revealing a linear relationship between calcium levels and inotropism.[4]

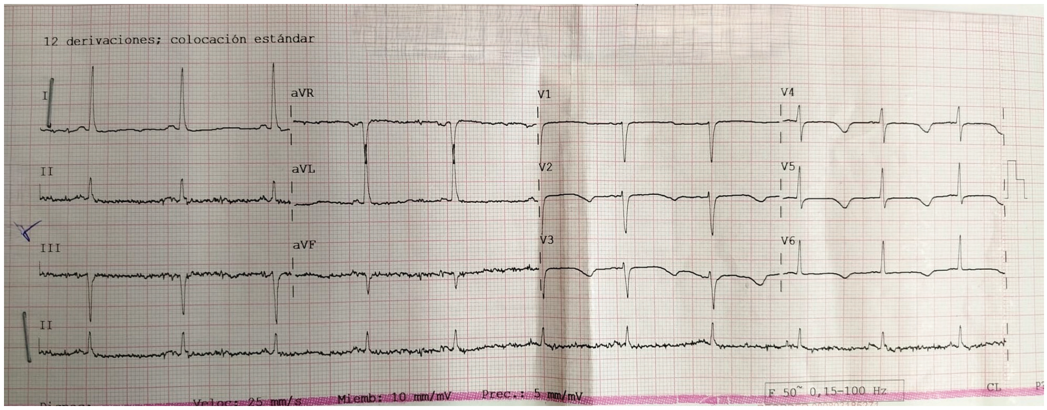

Figure 1 – *Echocardigram showing sinus rhythm, narrow QRS complexes, diffuse inverted T waves and a QTc of 700 ms.*

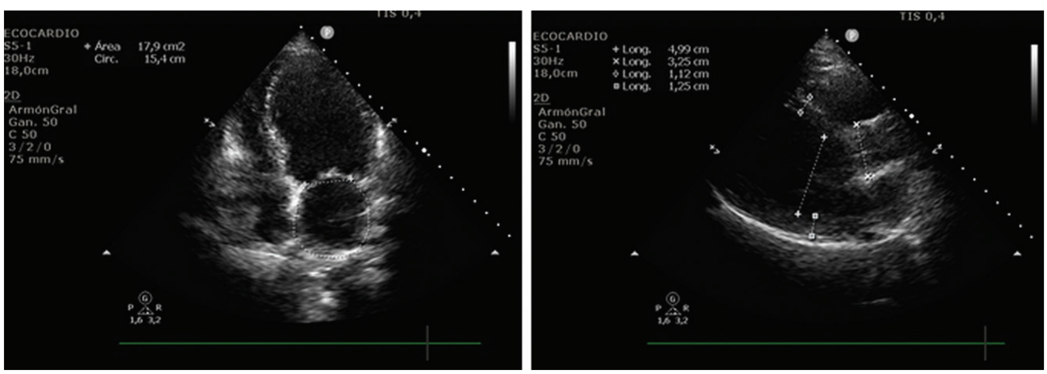

Figure 2 – *Transthoracic echocardiogram showing diffuse hypokinesia of the left ventricle and a left ventricular ejection fraction (LVEF) of 28%.*

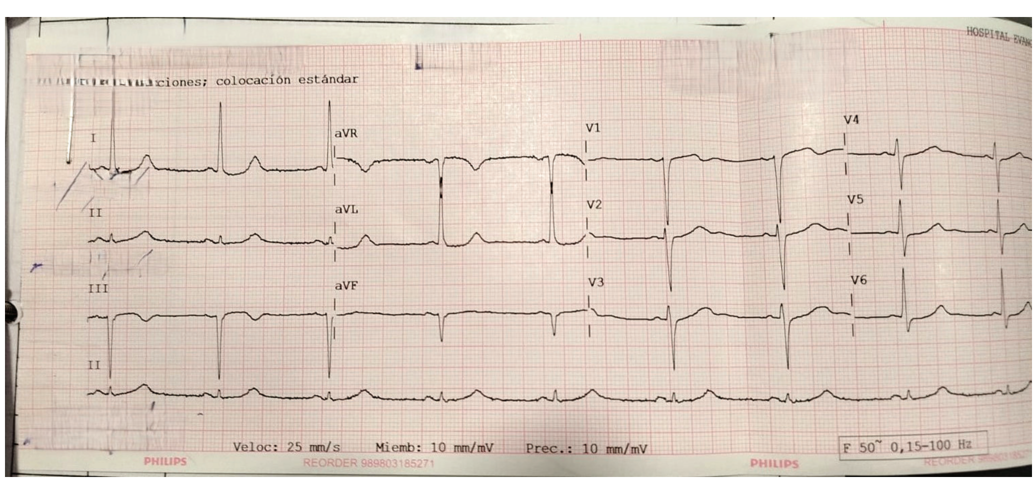

Figure 3 – *Echocardiogram showing positive T waves with a corrected QT interval of 480 ms.*

Hypocalcemia is a rare etiology of cardiomyopathy, with few reports in the literature. The only systematic review published to date includes 47 patients from case series and reports, most of them secondary to hypoparathyroidism,[5] which is the main cause of chronic hypocalcemia.[6]

Most case reports highlight three features that characterize this condition as a distinct subtype of cardiomyopathy: a) a limited response to standard heart failure therapy, b) an association with chronic hypoparathyroidism, and c) its reversibility upon normalization of serum calcium levels.[5]

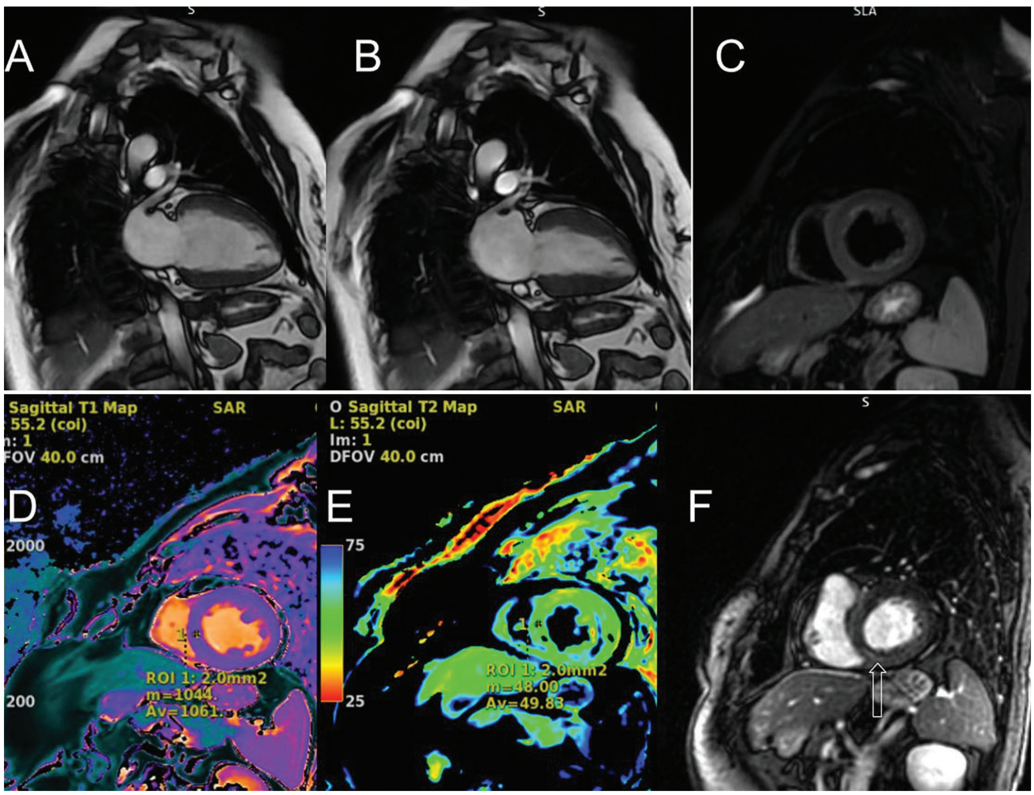

Figure 4 – *ardiac-MRI. Panels A and B show diastole and systole, respectively, in a two-chamber projection (FFPP). Panel C displays a T2-enhanced sequence. Panels D and E show the T1 and T2 mapping, respectively. Panel F displays a late gadolinium enhancement sequence (PSIR); the arrow indicates the site of enhancement.*

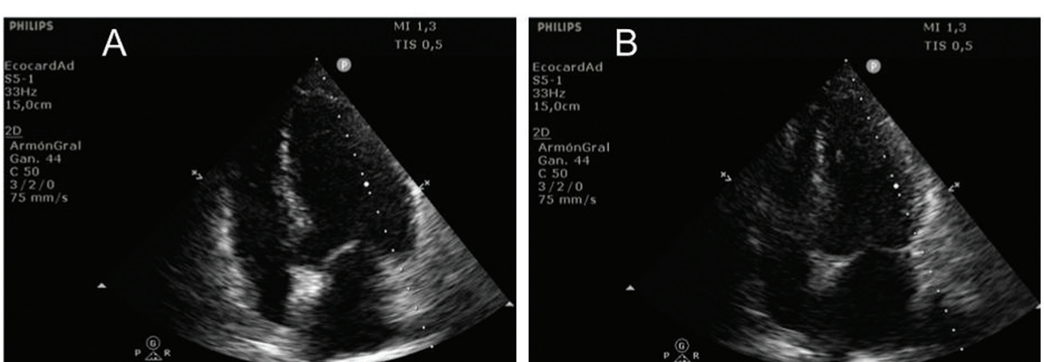

Figure 5 – *Transthoracic echocardiogram performed four weeks after discharge. Panel A shows the left ventricle in diastole, and Panel B in systole; the left ventricular ejection fraction (LVEF) was calculated at 60%.*

Regarding the pathophysiology of this condition, the leading theory suggests that reduced calcium influx through L-type channels impairs the sarcoplasmic reticulum's ability to elevate intracellular calcium levels via the calcium-induced calcium release mechanism. This disruption negatively affects actin–myosin cross-bridge formation, thereby reducing cardiac inotropism.[7-9]

Phase 2 of the cardiac action potential corresponds to the ST segment and T wave on the ECG. The ST segment reflects a balance between inward calcium currents and outward potassium currents. In hypocalcemia, the QTc interval is prolonged due to an extended isoelectric ST segment (plateau), which distinguishes it from other causes of QT prolongation. This phenomenon occurs because the termination of transmembrane calcium current relies on calcium flow through L-type channels via a mechanism known as calcium-mediated inactivation. Consequently, reduced calcium flow prolongs the duration of this current, leading to an extended ST segment.[10]

Our patient presented with transient systolic dysfunction, which showed rapid clinical and echocardiographic improvement following calcium administration. This supports the diagnosis of myocardial dysfunction secondary to severe hypocalcemia.

Cardiac MRI represents a novel diagnostic element in this context. The T2 mapping and T2-weighted sequences showed no evidence of edema, helping to rule out differential diagnoses such as acute myocarditis. The normal T1 values suggest minimal impact on overall myocardial composition. However, the observed fibrosis pattern evokes the possibility of a harmful agent or toxin affecting the myocardium. Could hypocalcemia trigger a degree of cellular dysfunction leading to myocyte necrosis and fibrosis?

Based on the rapid recovery of ventricular function following calcium replacement, as observed in this case and others, it is expected that this condition does not induce fibrosis or significant structural alterations. Instead, it likely involves a substrate of reversible myocardiopathic dysfunction linked to impaired actin–myosin interaction. In this context, the abnormalities seen on cardiac MRI may reflect an intercurrent condition such as chronic-phase myocarditis, hypertensive or diabetic cardiomyopathy, or a genetically determined cardiomyopathy, which could have amplified the myocardial depressant effects of hypocalcemia.

In conclusion, this case highlights a rare clinical entity that can be suspected based on clinical history and electrocardiographic findings. It was characterized through cardiac MRI and is associated with an excellent prognosis.

Author Contributions

Conception and design of the research: Lenzi B, Tortajada G, Rabaza V, Cairolli E, Parma G. Acquisition of data: Lenzi B. Analysis and interpretation of the data: Lenzi B, Tortajada G, Cairolli E. Writing of the manuscript: Lenzi B, Rabaza V. Critical revision of the manuscript for intellectual content: Tortajada G, Cairolli E, Parma G.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Mutualista Hospital Evangélico under protocol number 110. All procedures involved in this study are in accordance with the Declaration of Helsinki of 1975, as revised in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

References

1. Boron WF, Boulpaep EL. Medical physiology. 3rd ed. Philadelphia: Elsevier; 2016.
2. Bove-Fenderson E, Mannstadt M. Hypocalcemic Disorders. Best Pract Res Clin Endocrinol Metab. 2018;32(5):639-56. doi: 10.1016/j.beem.2018.05.006.
3. Ringer S. Concerning the Influence Exerted by Each of the Constituents of the Blood on the Contraction of the Ventricle. J Physiol. 1882;3(5-6):380-93. doi: 10.1113/jphysiol.1882.sp000111.
4. Lang RM, Fellner SK, Neumann A, Bushinsky DA, Borow KM. Left Ventricular Contractility Varies Directly with Blood Ionized Calcium. Ann Intern Med. 1988;108(4):524-9. doi: 10.7326/0003-4819-108-4-524.
5. Newman DB, Fidahussein SS, Kashiwagi DT, Kennel KA, Kashani KB, Wang Z, et al. Reversible Cardiac Dysfunction Associated with Hypocalcemia: A Systematic Review and Meta-Analysis of Individual Patient Data. Heart Fail Rev. 2014;19(2):199-205. doi: 10.1007/s10741-013-9371-1.
6. Bjornsdottir S, Ing S, Mitchell DM, Sikjaer T, Underbjerg L, Hassan-Smith Z, et al. Epidemiology and Financial Burden of Adult Chronic

Case Report

Hypoparathyroidism. J Bone Miner Res. 2022;37(12):2602-14. doi: 10.1002/jbmr.4675.

7. Bansal B, Bansal M, Bajpai P, Garewal HK. Hypocalcemic Cardiomyopathy-Different Mechanisms in Adult and Pediatric Cases. J Clin Endocrinol Metab. 2014;99(8):2627-32. doi: 10.1210/jc.2013-3352.

8. Santos TS, Carvalho AC. Hypocalcemic Cardiomyopathy - A Rare and Reversible Entity. Am J Emerg Med. 2022;51:426.e1-426.e3. doi: 10.1016/j.ajem.2021.06.060.

9. Lozano PJM, Junyent JMG. Hypocalcaemia: An Unusual and Potentially Reversible Cause of Dilated Cardiomyopathy. Med Clin. 2011;136(3):133-4. doi: 10.1016/j.medcli.2009.11.010.

10. Kramer DB, Zimetbaum PJ. Long-QT Syndrome. Cardiol Rev. 2011;19(5):217-25. doi: 10.1097/CRD.0b013e3182203504.