Case report

Acute ST-segment elevation myocardial infarction: to be or not to be?

Maria Carolina Manzur Barbur[1], Maria Cristina Martínez-Ávila[1,*], Angélica Imitola Madero[2], José Fernando García Nuñez[3], Juan Camilo Garcia Dominguez[4]

[1]Department of Internal Medicine, Centro Hospitalario Serena del Mar, Cartagena, Colombia; [2]Division of Endocrinology, Diabetes and Metabolism. Centro Hospitalario Serena del Mar, Colombia; [3]Department of Cardiology, Interventional Electrocardiology. Centro Hospitalario Serena del Mar, Colombia; [4]Department of Cardiology, Echocardiography and Cardiac Imaging. Centro Hospitalario Serena del Mar, Colombia

Abstract

Calcium has a significant effect on cells in the myocardium, affecting conduction, intracellular signaling and contraction of muscle fibers. In fact, calcium levels could affect myocardial action potential and affect heart conduction. Hypocalcemia manifest on ECG with arrythmias or QT prolongation, in some extraordinary cases, it could present with variations in the ST segment, which requires the study of differential diagnoses of cardiac origin, such as acute myocardial infarction with ST elevation (STEMI) or pericarditis. We expose a case of a 28-year-old male patient who arrived at the emergency department with chest pain and was misdiagnosed with STEMI, after a complex process finally it was a severe hypocalcemia mimicking acute myocardial infarction.

Keywords: *hypocalcemia; electrocardiogram; ST segment elevation myocardial infarction; hypoparathyroidism*

Introduction

Chest pain is a frequent symptom in the triage and emergency clinic. Prompt recognition and exclusion of life-threatening causes such as aortic dissection, ST segment elevation myocardial infarction (STEMI), pericarditis with tamponade, pulmonary embolism, esophageal perforation, tension pneumothorax has a high impact on morbidity and mortality [1]. Diagnosis strategies include serum chemistry, X ray, electrocardiogram (ECG), echocardiogram, or even invasive interventions to help differentiate among various pathological entities [1].

Received: February 2022; Accepted after review: March 2022; Published: March 2022.

*Corresponding author: Maria Cristina Martínez-Ávila, Department of Internal Medicine, Centro Hospitalario Serena del Mar, Vía Al Mar, Serena del Mar, Cartagena, Bolívar, Colombia
Email: maria.martinez@chsm.com

Acute myocardial infarction is one of the leading causes of chest pain that can turn to serious complications and death [1]. Acute myocardial infarction can be divided in two categories: STEMI and non-ST segment elevation MI (NSTEMI). STEMI generally indicates a total blockage of a coronary artery, that causes heart muscle to die; this is reflected in a distinct and characteristic pattern on the ECG [1]. However, some non-ischemic causes can exhibit with ST segment elevation not associated with STEMI, making patients with such ECG pattern candidates for invasive and unnecessary interventions. Among non-ischemic etiologies electrolyte derangements are known [2].

Herein, we describe the case of a 28-year-old man who arrive to the emergency department complaining of acute onset chest pain, highlighting the clinical importance of considering hypocalcemia as a likely reversible cause of ECG changes.

Case report

The patient reported 22-hours of retrosternal chest pain at rest, radiated to his back, jaw and left arm. He was obese and had history hypertension being risk factors for coronary artery atherosclerosis, as well as history of thyroidectomy with postoperative hypoparathyroidism more than 3-years ago. Vitals were unremarkable, blood pressure was normal: 124/79 mm Hg, heart rate 60 beats/minute, respiratory rate was 18 breaths/minute, oxygen saturation was 96% at room air. Physical examination evidenced facial muscle spasms after cheek percussion (Chvostek sign). The heart was normal on percussion and auscultation with regular pulse at physical examination. Due patient symptoms and medical history a 12-lead ECG was obtained. ECG depicted ST elevation in inferior and lateral leads (Figure 1). Initial high-sensitivity cardiac troponin was 8,858.30 ng/mL (normal: 0-11.6 ng/mL). Inflammation markers were negative (leukocytes 6,000/mm^3, C reactive protein 0.5 mg/L). Portable anterior-posterior chest x-ray was normal.

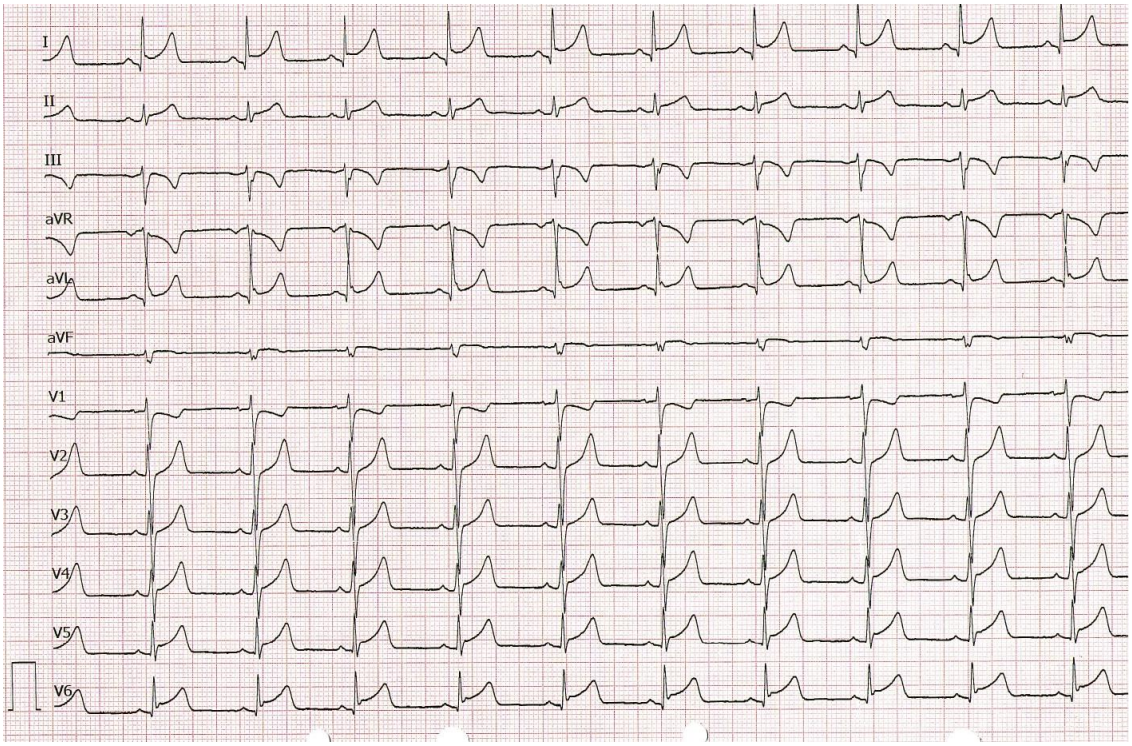

Fig. 1. ECG showing ST elevation in leads I, II, aVL and V5, V6

Diagnosis. The classic appearance of acute ST segment elevation, suggestive of antero-lateral myocardial infarct, is of major concern needing immediate diagnosis and treatment reason to why patient was immediately transfer to coronary angiography. Before coronary intervention, he received dual antiplatelet agents, chewed full dose of aspirin, P2Y12 platelet inhibitor: ticagrelor and intravenous heparin infusion. However, emergent percutaneous coronary intervention did not reveal obstructive coronary artery disease. Echocardiogram was performed to evaluate anatomical and function of the myocardium as well to seek for differential diagnoses; per contra it displayed preserved myocardial function. The septum and free left ventricle thickness were normal, with no regional wall motion abnormalities, and with a left ventricular ejection fraction of 62%.

Seeking differential diagnosis due patient risk factors and retrosternal chest pain duration (about 22-hours) as a great angina crisis; pulmonary embolism and myocarditis

were ruled out by angio-CT and Cardiac MRI respectively. Regardless, ECG suggests rather pericarditis because ST segment is upper concave, inflammation markers were negative, and imaging had no pericardial inflammation decreasing this suspicion.

On further questioning, the patient admitted he stopped oral calcium supplements. Laboratory evaluation showed severe hypocalcemia (ionized calcium 0.53 mmol/L), normal serum magnesium (2.21 mg/dL), potassium (3.8 mEq/L) and thyroid stimulating hormone (0.49 µU/mL). Renal function was conserved (creatinine 0.86 mg/dL, blood urea nitrogen 20 mg/dL). Additionally arterial blood gases show no acid-base imbalance and a normal alkaline reserve.

Treatment. After diagnosing severe hypocalcemia and ruling out other electrolyte disturbances, treatment was established. The treatment was settled according to the severity, presence of symptoms, and etiology of hypocalcemia. The administration of a continuous infusion of 2 grams of calcium gluconate in association with vitamin-D was the treatment of choice. The high sensitivity cardiac troponin taken in absence of chest pain showed 10.35 ng/mL as a result. An ECG was performed when serum ionized calcium reached its normal range and demonstrated normalization of the ST segment deviations and QTc interval (Figure 2).

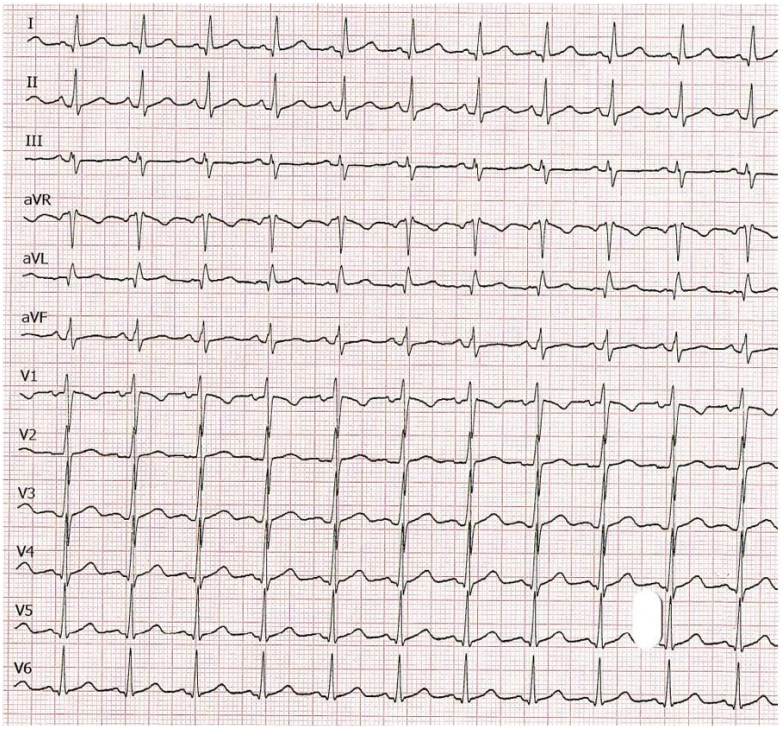

Fig. 2. ECG after treatment with intravenous calcium showed normalization of the ST segment deviations and QTc.

Follow-up and outcomes. Patient was discharged fully asymptomatic with normal laboratory values in phosphocalcium metabolism without further complication.

Discussion

When the ST segment is elevated on the ECG a careful attention to the ST-T and QRS-complex configurations is indispensable to distinguish among the possible underlying causes of ST segment elevation [1, 3]. The differential diagnosis of ST elevation embraces ischemic and non-ischemic causes as STEMI, early repolarization, myopericarditis, pulmonary embolism, hyperkalemia, and ST elevation secondary to an abnormality of the QRS complex as preexcitation, left ventricular hypertrophy or left bundle branch block [4].

Other abdominal conditions include acute pancreatitis and very large hiatal hernias [3].

Even though, electrocardiographic ST segment deviation is common finding, among all ischemic and non-ischemic causes, hypocalcemia is an uncommon etiology involving the ST-segment [2]. Rare cases of hypocalcemia have been described in the literature [5–8], presenting electrocardiographically with a ST segment elevation and simulating acute myocardial infarction. However, in most cases hypocalcemia was associated with further electrolytic imbalance, such as hypokalemia or hypomagnesaemia, which are associated with more notoriously electrocardiographic alterations that can mimic an acute STEMI. In other cases of hypocalcemia previously described, in addition to the ST segment elevation on ECG an alteration of the contractile function of the left ventricle was found on echocardiogram, in the absence of coronary artery disease [5–8]. The presented case is atypical because is just calcium disorder without QT alterations and normal contractile function, making it an interesting medical illustration as it shows a curious ECG manifestation caused by electrolyte derangements such as severe hypocalcemia.

In this case, although the patient had risk factors for disorders in phospho-calcium metabolism (hypoparathyroidism and Chvostek sign), his symptoms pointed to be due cardiac arrest.

During the comparison of the presented ECG with clinical setting and specific electrocardiographic criteria of the differential conditions we could affirm this pseudo-infarct pattern was the result of hypocalcemia. First of all, we could consider an early repolarization pattern in view of notched J-point and ST elevation not exceeding 3 mm, however, in our ECG the ST segment / T wave ratio and the ST segment upper concave morphology pointed out to be more like a pericarditis. Nevertheless, non-criterial diagnosis rather than ECG features without pericardial friction, effusion, biomarkers nor imaging with inflammation disparage this diagnosis. Secondly, ST segment morphology mimics STEMI, but there are enough signs to the contrary, as the absence of T inverted waves, Q waves or reciprocal ST depression; summed to that normal coronary arteriography disesteem the diagnosis. Other possibilities were ruled out by its own such as hyperkalemia, acidosis.

At the end of a complex diagnostic process, that included cardiac enzymes, electrolyte profile, echocardiogram, coronary angiography, cardiac MRI and angio-CT, severe hypocalcemia was diagnose, which would also justify the electrocardiographic feature presented by the patient. Finally pseudo-infarct pattern on the ECG was outstanding severe hypocalcemia, transient metabolic changes, indicated by troponin levels, which reverse with intravenous treatment.

The mechanism by which low serum calcium results in the pseudo-infarct pattern on ECG is not fully elucidated. Calcium channels in normal conditions close at the end of phase 2 of the cardiac action potential, when severe hypocalcemia, the duration of phase 2 is prolonged which results in a ST-segment lengthening with subsequent prolongation of the QT interval [9]. A plausible explanation is that hypocalcemia dropped cell membrane potential, with consequential increased cell membrane permeability, liberation of cytoplasmic proteins, which cause alteration in depolarization/repolarization, cardiac injury, represented in the ECG with changes on ST segments that may imitate acute myocardial infarction, arrhythmias, conduction abnormalities or cardiomyopathy [2, 10].

The clinical manifestation of hypocalcemia depends on serum levels and its chronicity [10]. Symptoms ranges from asymptomatic, passing through mild symptoms as paresthesia that could be perioral or in extremities, neuromuscular irritability, muscle cramps, carpopedal spasm known as Trousseau's sign, facial muscle spasms after cheek percussion: Chvostek sign, psychiatric emotional lability: anxiety/depression [10]. Life threatening symptoms include papilledema, seizures, heart failure, spontaneous or latent tetany, laryngospasm and QT prolongation which evolved to torsades de points [10]. Management is realized according to the severity and presence of symptoms, and etiology of hypocalcemia. Normalization of

serum calcium levels is the standard intervention to revert symptoms and ECG manifestations. Nevertheless, vitamin D supplementation is usually indicated to endorse better calcium absorption. If there's any other electrolyte imbalance, it should be corrected first.

Conclusions

Conclusively, in patients with risk factors and chest pain is necessary to analyze entire clinical scenario, including serum chemistry values to aide in the interpretation of ECG abnormalities. Educating patients and physicians about the disease, symptoms, possible risks, complications, and treatments, is essential to assure adherence, prevent morbidity, as well as so that patients can seek medication adjustment in a timely manner. The diagnosis and treatment of hypocalcemia requires a multidisciplinary and comprehensive team.

Acknowledgements

Written informed consent was obtained from the patient for publication of this case report.

Conflicts of interest

The authors declare that they have no competing interests.

References

1. O'gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2013; 61(4):e78-e140. doi:10.1016/j.jacc.2012.11.019
2. Diercks DB, Shumaik GM, Harrigan RA, Brady WJ, Chan TC. Electrocardiographic manifestations: electrolyte abnormalities. *J Emerg Med*. 2004; 27(2):153-160. doi:10.1016/j.jemermed.2004.04.006. PMID: 15261358.
3. Rautaharju PM, Surawicz B, Gettes LS. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part IV: the ST segment, T and U waves, and the QT interval a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society Endorsed by the International Society for Computerized Electrocardiology. *J Am Coll Cardiol.* 2009; 53(11):982-991. doi:10.1016/j.jacc.2008.12.014
4. Gard JJ, Bader W, Enriquez-Sarano M, Frye RL, Michelena HI. Uncommon cause of ST elevation. *Circulation*. 2011; 123(9):e259-e261. doi:10.1161/CIRCULATIONAHA.110.002477
5. Ilveskoski E, Sclarovsky S, Nikus K. Severe hypocalcemia simulating ST-elevation myocardial infarction. *Am J Emerg Med.* 2012; 30(1):256.e3-6. doi:10.1016/j.ajem.2010.10.021
6. Khardori R, Cohen B, Taylor D, Soler NG. Electrocardiographic finding simulating acute myocardial infarction in a compound metabolic aberration. *Am J Med.* 1985; 78(3):529-532. doi:10.1016/0002-9343(85)90351-1
7. Reddy CV, Gould L, Gomprecht RF. Unusual electrocardiographic manifestations of hypocalcemia. *Angiology*. 1974; 25(11):764-768. doi:10.1177/000331977402501105
8. Lehmann G, Deisenhofer I, Ndrepepa G, Schmitt C. ECG changes in a 25-year-old woman with hypocalcemia due to hypoparathyroidism. Hypocalcemia mimicking acute myocardial infarction. *Chest*. 2000;118(1):260-262. doi:10.1378/chest.118.1.260
9. Pozzolini A, Rio T, Padeletti M, De Ponti R, Leonelli FM, Bagliani G. Complex arrhythmias due to reversible causes. *Card Electrophysiol Clin.* 2019; 11(2):375-390. doi:10.1016/j.ccep.2019.03.002
10. Melmed S, Auchus RJ, Goldfine AB, Koenig R, Rosen CJ. Williams Textbook of Endocrinology. 14th edition. Philadelphia: Elsevier; 2019.