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ST-Segment Elevation Mimicking STEMI Due to Hypercalcemia: A Case Report

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Abstract

ST-segment elevation myocardial infarction (STEMI) is an important, life-threatening diagnosis that requires quick diagnosis and treatment, characteristic ECG of which shows ST-segment elevation. Unfortunately, ST-segment elevation is nonspecific, which can be misleading if not careful to be interpreted, as in this case of hypercalcemia seen by us. A 48-year-old male was admitted to our emergency department with recurrent chest pain, nausea and vomiting. Medical history includes hypertension and diabetes. ST-segment elevation in V1 - V4 mimicking STEMI was present on admission. However, immediate coronary angiography revealed nearly normal coronary arteries, his troponin was negative in 6 hours and calcium was 2.95 mmol/L. It was thought that the ECG changes were not indicative of cardiac ischemia but hypercalcemia. He was managed with calcium reduction treatment such as intravenous normal saline and furosemide, with subsequent resolution of ST-segment changes.

Keywords

Hypercalcemia, Myocardial Infarction, ST Elevation, ECG

1. Introduction

ECG is an important non-invasive test in diagnosis of some severe cardiac diseases such as STEMI, which shows ST-segment elevation and usually requires urgent primary percutaneous intervention. However, ST-segment elevation is nonspecific and may be disease other than STEMI. There are several known mimickers of STEMI that are well documented, but hypercalcemia is rarely mentioned. It's well established that Characteristic ECG changes in hypercalcemia are shortened QT interval, prolonged PR, lengthened QRS interval, flattened or inverted T waves and variable degrees of heart block, few authors have reported

transient ST-segment elevation mimicking STEMI in patients with hypercalcemia. However, as Adam Orville Strand mentioned, hypercalcemia leading to ECG changes consistent with an STEMI is considered very unique but not rare as believed, but only under-reported with a lack of common association [1]. We report a patient with chest pain who showed ST-segment elevation due to hypercalcemia mimicking AMI. By recognizing this, clinicians should take into consideration of more mimickers on ST-segment elevation and prevent adverse outcomes that could easily be avoided.

2. Case Presentation

A 48-year-old male was admitted for recurrent chest pain, nausea and vomiting. He had intermittent retrosternal pain half a month prior, which is more serious after physical activity. An ECG performed in another hospital showed complete right bundle branch block (**Figure 1(a)**), and he refused further diagnosis and treatment. 1 hour ago, he perspired heavily after physical labor, and soon developed nausea, vomiting and fatigue, without chest pain, back pain and dyspnea. ECG in community health unit demonstrated new ST-segment elevation in V1 - V4 (**Figure 1(b)**) concerning STEMI, and patients were sent to our hospital by ambulance after giving 300 mg aspirin,180 mg ticagrelor and 20 mg rosuvastatin calcium.

Medical history includes poorly controlled hypertension that had not been treated medically 5 years before admission, long-term diagnosis of stage 2 diabetes

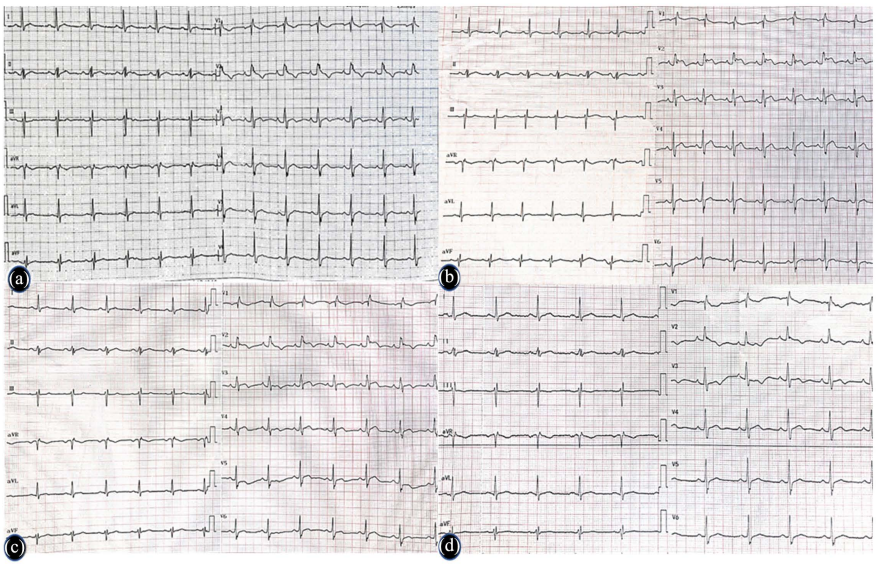

Figure 1. Dynamic ECG evolution of patients: ECG (a) in another hospital suggested incomplete right bundle branch block; ECG (b) in community health unit suggested 0.1 - 0.3 mV of ST-segment elevation in V1 - V4, QoTc 187 ms, QaTc 257 ms, QeTc 351 ms; Postoperative ECG (c) indicated that there was no-dynamic change, QoTc 190 ms, QaTc 268 ms, QeTc 358 ms; Re-examination of ECG (d) on the second day of admission indicated that ST-segment in V1 - V4 returned to normal, QoTc 230 ms, QaTc 312 ms, QeTc 396 ms.

which was controlled by acarbose, not monitoring blood sugar. The patient denied any other history of coronary heart disease, or kidney disease, and never smoked and drank.

In the emergency department, his blood pressure was 130/86mmHg, respirations at 20/min (12 - 20/min), heart rate of 82 beats per minute (60 - 100 beats per min), oxygen saturation of 94% (95% - 100%). There were no pertinent positive physical findings. Given the patient history and the ECG findings, STEMI should be taken into consideration, he undergo urgent coronary angiography, which revealed nearly normal coronary arteries (**Figure 2**).

Subsequently, the first set of cardiac enzymes showed elevated myoglobin 89.99 ng/mL (0 - 58 ng/mL), normal creatine kinase isoenzyme 4.25 ng/mL (0 - 5 ng/mL), troponin negative. All other laboratory values were within normal range except for white blood cell of 12.88 × 10^9/L, of creatinine 213 μmol/L (57 - 111 μmol/L), sodium of 135 mmol/L (137 - 147 mmol/L), chlorine of 93 mmol/L (99 - 110 mmol/L), calcium of 2.95 mmol/L (2.2 - 2.5 mmol/L), glucose of 15.56 mmol/L (3.9 - 6.1 mmol/L). Bedside echocardiography showed no obvious abnormalities 6 hours after the onset, and the second set of cardiac enzymes myoglobin returned to normal, while creatine kinase isoenzyme and troponin I were still negative, and repeated ECG showed persistent marked ST elevation (**Figure 1(c)**), which excluded myocardial infarction as the cause of his electrocardiographic changes. And through searching pertinent literatures, acute renal function injury with hypercalcemia was considered.

The patient was subsequently managed with calcium reduction treatment such as intravenous normal saline and furosemide 20 mg iv. His calcium and creatinine improved progressively (calcium 2.42 mmol/L, creatinine 106 umol/L) on the second day after admission, meanwhile, follow-up ECG showed ST-segment back to baseline in V1 - V4 (**Figure 1(d)**). In addition, his parathyroid hormone was normal.

Over the next few days, the patient's condition improved dramatically. He continued to deny any chest pain or pressure during his following admission and did not deteriorate clinically. He has subsequently discharged home on day four post-presentation.

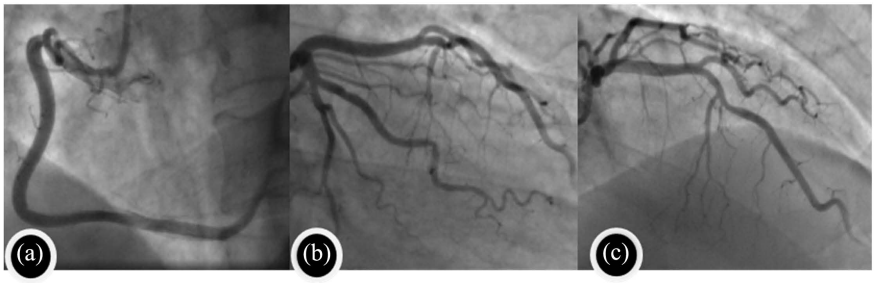

Figure 2. Coronary angiography results of the patient: coronary atherosclerosis in the middle part of the LAD, TIMI grade 3 in the distal blood flow, and no obvious abnormalities in other vessels.

3. Discussion

STEMIs are frequently encountered in hospital visitations and admissions. The earlier the reperfusion therapy is initiated, the greater the benefit. However, there are several known mimickers of STEMI as well as hypercalcemia. Several risks make the diagnosis of the patient STEMI likely: history of diabetes and smoking, chest pain related to physical labor. Eventually, a review after seeing the high-serum calcium levels clarified that ECG changes were all due to acute hypercalcemia.

Hypercalcemia is a common and important clinical problem, most commonly caused by primary hyperparathyroidism and malignant neoplasms, in addition to vitamin D poisoning, sarcoidosis, and other granulomatous diseases [2]. Its clinical manifestations are non-specific, including fatigue, nausea, vomiting, abdominal pain, etc., and can also cause hypovolemia with acute renal injury. The PTH is normal in the patient, who had no special drug taking and no history of tumors causing high calcium. Considering the patient perspired heavily before the onset, the author speculates that dewatering may lead to hypercalcemia.

As is known to all, hypercalcemia can also cause abnormal heart conduction system. The relationship between hypercalcemia and shortened QTc interval is close, including the intervals from the beginning of the QRS to the onset (QoTc), the apex (QaTc), and the end of T wave (QeTc), among which QoTc (<180 ms) and QaTc (<300 ms) have the highest specificity in predicting hypercalcemia [3]. In our case, in the hypercalcemic ST elevated phase, the QoTc and QaTc were 187 ms and 257 ms returned to 230 ms and 310 ms after hypercalcemia was corrected.

In a case-control study published in 2016, Keiko Sonoda, M. D found that J point elevation (including early repolarization and Brugada-type ECG) and ST-segment elevation in hypercalcemia were also common [4]. The patient's ECG was similar to STEMI, and he had risk factors associated with coronary heart disease that more likely led to confusing the diagnosis. ST-segment elevation in hypercalcemia mimicking AMI has been reported in a few cases (Ashizawa *et al.* described a patient with hypercalcemia caused by vitamin D poisoning with "ST-segment elevation" in lead V1 - V2 resembling acute myocardial infarction [5]. Turhan *et al.* reported a case of ST-segment elevation in lead I, II and V1 - V6 [6].) Littman *et al.* reported 16 cases of severe hypercalcemia presented with ST-segment elevation ECG changes in conditions other than AMI [7]. Their ST-segment elevation location was not fixed and was mainly found in the precordial leads. The elevated ST-segments always had a characteristic "scooped" appearance and typically were not followed by distinct T waves. The mean serum calcium was 14.3 ± 2.9 mg/dL and QTc 379 + 44 ms. ST-segment elevation in AMI is often accompanied by prolonged QT interval, ST-segment specular depression, and following characteristic dynamic evolution of ST-segment. The patient did not have typical angina pectoris, the electrocardiogram V1 - V4 was elevated, but the QTc was not prolonged, and there was no obvious specular depression of ST-segment. Troponin I was still normal within 6 hours of onset,

so the diagnosis of AMI was excluded. Of course, the differential diagnosis of ST elevation also needs to consider ventricular tumor, pericarditis, left ventricular hypertrophy, hyperkalemia, etc. Echocardiography and laboratory tests ruled out these possibilities. The ECG recovered after we corrected hypercalcemia.

The mechanism leading to ST-segment elevation in hypercalcemia remains unclear. Studies have shown that shortening QT due to hypercalcemia is mainly caused by shortening the duration of stages 2 and 3. The duration of action potential phase 2 is shortened by inward calcium flow through l-type calcium channels [8]. The higher calcium concentration in the extracellular fluid will promote the rapid influx of calcium through the channel, causing intracellular calcium ions to rapidly reach the threshold of closing l-type calcium channel, reducing the duration of action potential phase 2 [9]. The duration of phase 3 in the action potential is determined by outward potassium flow through delayed recirculating potassium channels [8]. Increased extracellular calcium concentration promotes intracellular entry of calcium ions, possibly activating these potassium channels in the frog heart. This will increase the outward potassium current in stage 3 of the action potential, thus speeding up the repolarization process and thus shortening the overall duration of the systolic phase [10].

4. Conclusion

Clinicians should be aware that high calcium levels may be associated with a pseudo-infarct pattern on the ECG. In patients who are less likely to have acute coronary syndrome, other causes should be explored in a timely manner prior to further intervention. Clinicians must weigh the urgency and risks of intervention against the benefits of waiting for more laboratory results. Nevertheless, if clinical discrepancies make the probability of a STEMI low, it is reasonable to wait for basic laboratories before any type of intervention is pursued. Measurement of QTc interval changes may help in the diagnosis. Dynamic troponin I and electrocardiogram testing are also important in patients with hypercalcemic nephropathy; performing coronary angiography for apparent ST-segment elevation could be harmful because it may result in further deterioration of renal function [11].

Informed Consent

The patient's legal representative has given permission for the publication of this report and the accompanying images.

Conflicts of Interest

The author declares that they have no conflicts of interest pertaining to this article.

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