Hypercalcemia-Induced New Onset Left Bundle Branch Block Mimicking Acute Myocardial Infarction in a Patient with Primary Hyperparathyroidism

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A 78-year-old women with a recent diagnosis of primary hyperparathyroidism presented with vague chest pain, and new onset left bundle block (LBBB) on the electrocardiogram (ECG) mimicking acute myocardial infarction (AMI). LBBB resolved without abnormal Q waves only after correction of hypercalcemia. The cardiac enzymes, including creatine kinase, creatine kinase-MB, and troponin-I were all within normal range. Hypercalcemia provoking ECG changes that mimic acute myocardial infarction is infrequently reported. To our knowledge, this is the first report of hypercalcemia-induced new onset LBBB mimicking AMI. Emergency physicians should include hypercalcemia-induced new onset LBBB on the ECG in the differential diagnosis of AMI.

Key Words: Acute myocardial infarction • Hypercalcemia • Hyperparathyroidism • left bundle branch block

INTRODUCTION

Acute myocardial infarction (AMI) is an important differential diagnosis in patients who present with acute chest pain and new onset left bundle branch block (LBBB) on the electrocardiogram (ECG). Unnecessary treatment can be harmful in patients who have new onset LBBB due to conditions other than AMI. We report a patient with primary hyperparathyroidism (pHPT) who presented with new onset LBBB on the ECG mimicking AMI.

CASE REPORT

A 78-year-old female presented to our facility with a 10-year history of hypertension. The patient denied any history of coronary artery disease, diabetes mellitus, or hyperlipidemia. She had been hospitalized in another institution for a deteriorating level of consciousness and diagnosed with pHPT-induced hypercalcemia. Laboratory studies revealed increased parathyroid hormone (PTH): 354 pg/ml (12-72), calcium: 11.2 mg/dl (8.4-10.2), blood urea nitrogen (BUN): 44 mg/dl (5-25), and creatinine (Cr): 1.2 mg/dl (0.7-1.4). ECG showed sinus rhythm and left ventricular hypertrophy by voltage (Figure 1A). After hydration with normal saline and diuretic therapy, the patient’s serum calcium level decreased to 10.2 mg/dl. She was discharged under stable condition with medication including calcium channel block, nitrate and diuretic. Five days after her discharge, lethargy, accompanied with constipation, weakness, anorexia, nausea and vomiting, developed progressively. Then, she was sent to our emergency department under the chief
complaint of vague chest pain for 3 hours.

On examination, the patient’s Glasgow Coma Score was 12. She appeared dehydrated, with a body temperature of 36.5 °C, blood pressure 160/96 mmHg, pulse 102 beats per minute, respiratory rate 18 breaths per minute and the oxygen saturation 100% with ambient air. The patient’s heart and lung examinations were unremarkable. Her abdomen was distended with hypoaactive bowel sounds. There were no focal neurological signs or other significant findings. Computed tomography of the brain was performed and disclosed only old lacunar infarction over bilateral corona radiata and basal ganglia. Chest radiograph revealed an enlarged heart and clear lung. An initial ECG showed new onset LBBB (Figure 1B) as compared with those in a previous ECG (Figure 1A). Blood testing revealed acute renal failure: BUN 86 mg/dl (5-25), and Cr 2.0 mg/dl (0.7-1.4). Her serum calcium level was 12.8 mg/dl. The cardiac biomarkers including creatine kinase, creatine kinase-MB, and troponin-I were within normal range. ST-elevation myocardial infarction (STEMI) was suspected as first, but the patient’s family declined primary primary coronary intervention. Subsequently, the second and third set of cardiac enzymes were all within normal range. A transthoracic echocardiogram demonstrated left ventricular hypertrophy with paradoxical septal wall motion, without obvious akinesia or dyskinesia of the left ventricle lateral wall. The patient was treated with aggressive hydration, and diuretic and intravenous pamidronate to correct hypercalcemia. Her condition improved with clear consciousness, with no further complaints of chest pain after a decrease in serum calcium level to 10.4 mg/dl. An ECG following reversal of the hypercalcemia (Figure 2)

Figure 1. (A) Sinus rhythm and left ventricular hypertrophy (Heart rate: 78 bpm, no QT shortening); (B) New onset left bundle branch block (LBBB) (Heart rate: 96 bpm, Q-aTc: 280 msec).

Figure 2. Resolution of LBBB, inverted T wave over V2, V3, flat T wave over V4 to V6 (Heart rate: 114 bpm, Q-aTc: 280 msec).
showed resolution of LBBB. The patient was discharged from our hospital with medication, including valsartan 40 mg qd, amlodipine 5 mg qd, bisoprolol 5 mg qd and furosemide 40 mg qd. She had no recurrence of hypercalcemia after regular follow-up at our department for about one year.

DISCUSSION

This patient had a distinct history of pHPT and developed symptoms suggestive of hypercalcemia, including lethargy, constipation, weakness, anorexia, nausea and vomiting, in addition to vague chest pain. However, it was unclear whether the chest pain was typical, or due to poor consciousness level. The mechanism by which hypercalcemia causes chest pain in patients with pHPT is not well understood. Several possible mechanisms had been reported. First, the secretion of parathyroid hormone (PTH) can induce hypertrophic cardiomyopathy via its effect on adult cardiomyocytes as a hypertrophic factor and accumulation of calcium in the cells, where the ion triggers protein kinase C activity.1 Hypertrophic cardiomyopathy can lead to increased oxygen demand of myocardium, which eventually causes chest pain. Second, hypercalcemia may increase cardiac contractility and irritability, also leading to increased oxygen demand of myocardium and subsequent chest pain. Third, hypercalcemia predisposes calcific deposition in the valve cusps, annuli, and coronary arteries, which could cause significant aortic valve stenosis and accelerate coronary atherosclerosis. Both can mismatch the oxygen supply and myocardium demand.2 Finally, a patient with severe hypercalcemia usually appears dehydrated due to osmotic diuresis for hypercalcemia. Insufficient volume status causes tachycardia resulting in a deterioration of the balance between myocardial oxygen supply and demand.

The effect of hypercalcemia on the ECG is well-known, by shortening the QT interval mainly due to shortening of the ST segment. The intervals Q-oTc (the interval from the beginning of the QRS complex to the beginning of the T wave) of less than 0.18 second, and Q-aTc of less than 0.30 second (measured from the beginning of the QRS complex to the apex of the T wave) are reliable indicators of clinical hypercalcemia.3 Hypercalcemia decreases ventricular conduction velocity and shortens the effective refractory period. Cardiac conduction abnormalities may occur, the most common of which are bradydysrhythmias.4 A spectrum of other ECG findings include decreased T wave amplitude, T wave notching or inversion,5 atrioventricular block,6 ventricular fibrillation, nonthermal Osborn or prominent J waves,7 Brugada-like electrocardiographic pattern8 and transient ST-segment elevation.9 Only a few studies have reported transient ST-segment elevation in hypercalcemia.9 In addition to hyperparathyroidism, other possible causes of hypercalcemia-induced transient ST-segment elevation on the ECG (thereby mimicking STEMI) have been demonstrated, such as vitamin D intoxication,9 multiple myeloma, non-Hodgkin’s lymphoma, squamous cell lung cancer with bone metastasis, although the mechanisms involved have not yet been fully elucidated. In clinical practice, it is crucial to differentiate hypercalcemia from AMI.10-12

In our patient, an initial ECG showed new onset LBBB accompanied by vague chest pain, so STEMI was first suspected at the emergency department. Serial ECGs illustrated no dynamic ST-T change. LBBB resolved without abnormal Q wave only after correction of hypercalcemia. Shortening of Q-aTc interval could also be observed on the ECG. Transthoracic echocardiogram showed no regional wall motion abnormalities over lateral wall, but only paradoxical septal wall motion. Sequential cardiac biomarkers were all normal. According to the above findings, new onset LBBB accompanied by vague chest pain in conditions other than AMI should be considered. The observation of shortening of the Q-oTc or Q-aTc interval should raise clinical suspicion of hypercalcemia. To our knowledge, this is the first report of hypercalcemia-induced new onset LBBB mimicking AMI. In another report, conditions like Takotsubo cardiomyopathy could also cause transient LBBB mimicking AMI.13

In conclusion, simultaneous pHPT and hypercalcemia can cause angina-like chest distress and new onset LBBB on the ECG, mimicking AMI. Measurement of Q-oTc or Q-aTc interval with no evolutionary ECG changes may help in the diagnosis.

REFERENCES

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