Hypercalcemia and electrocardiogram changes

Sir,

A 45-year-old female was referred to us for further management in case of primary hyperparathyroidism with left superior parathyroid adenoma. On admission, her serum calcium, phosphorus, alkaline phosphatase, and Vitamin D levels were 12.6 mg/dl, 2.8 mg/ dl, 676 IU/ml, and 40 ng/ml, respectively. Serum parathyroid hormone was 220 pg/ml, and technetium sestamibi scan was suggestive of left superior parathyroid adenoma. Her electrocardiogram (ECG) showed significant ST depression in inferior and lateral leads [Figure 1]. Repeat ECG was performed and showed similar changes along with occasional ventricular premature contractions (VPCs). Cardiology department was requested for further evaluation. In view of no relevant clinical history for coronary heart disease, ECG changes were thought due to hypercalcemia. Her dobutamine stress echocardiography was negative for inducible ischemia. Subsequent ECG, after correction of hypercalcemia with hydration and loop diuretic, shows reversal of ST segment depression although changes of short QT interval persist [Figure 2]. Coronary angiogram was not performed in view of negative clinical history, dobutamine stress echocardiography, and subsequent normal ECG. The patient was later on subjected to surgery for parathyroid adenoma.

ECG changes are common in hypercalcemia, and they can mimic myocardial ischemia or infarction.^[1] It is important to distinguish between these two conditions as it may lead



Figure 1: Electrocardiogram showing ST depression in leads II, III, and aVF

to a battery of further investigations, thus increasing the cost of treatment and add significant stress to patient mind. Short QT interval is the most common finding, and this is contributed mainly by the shortening of ST segment. The ST segment may be completely absent and replaced by inverted small T-wave directly after R-wave. The intervals Q-oTc (the interval from the beginning of the QRS complex to the beginning of the T-wave) of <0.18 s and Q-aTc of <0.30 s (measured from the beginning of the QRS complex to the apex of the T-wave) are reliable indicators of clinical hypercalcemia.^[2] Decrease T-wave amplitude and T-wave notching can be observed. J-point elevation mimicking as ST segment elevation is a common observation.^[1,3] Transient ST segment elevation is observed in a few studies, particularly in a patient with severe hypercalcemia. Osborn wave which is usually a feature of hypothermia can also be observed in severe hypercalcemia.^[4] Hypercalcemia seemed to decrease atrial activity and increase ventricular activity as evidenced by the appearance of bradycardia, sinus arrest, and premature ectopic beats. Hypercalcemia decreases ventricular conduction velocity and shortens the effective refractory period. Ventricular arrhythmias ranging from VPCs to Frank ventricular fibrillation can be seen in severe degree of hypercalcemia.^[5] Other common changes include PR prolongation and increase in the amplitude of QRS complex.

In summary, electrocardiographic changes are common findings in hypercalcemia and often mimic those of acute myocardial infarction. Knowledge of these changes is essential as it helps in improvement of diagnosis and management and significantly, reduces the financial burden on health-care system.



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Figure 2: Reversal of ST depression after correction of hypercalcemia although short QT interval persists

Conflicts of interest

There are no conflicts of interest.

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