Electrocardiographic J Wave as a Result of Hypercalcemia Aggravated by Thiazide Diuretics in a Case of Primary Hyperparathyroidism

Ramazan TOPSAKAL,1 MD, Hayrettin SAĞLAM,1 MD, Hüseyin ARINÇ,1 MD, Namik Kemal ERYOL,1 MD, and Servet ÇETIN,1 MD

SUMMARY

Electrocardiographically, the QT interval is shortened, ST segment is depressed, and T wave becomes negative in hypercalcemia. The use of diuretics in cases with hyperparathyroidism decreases the excretion of calcium, causes changes in bone-calcium turnover and parathyroid hormone activity, and forms hypercalcemia. A 67 year-old Turkish female patient in whom we electrocardiographically observed a J wave is presented as a hypercalcemic case with primary, hyperparathyroidism aggravated by the use of a thiazide diuretic. (Jpn Heart J 2003; 44: 1033-1037)

Key words: Hypercalcemia, J wave, Hyperparathyroidism, Thiazide diuretic

ELECTROCARDIOGRAPHICALLY, the QT interval is shortened, ST segment is depressed, and T wave becomes negative in hypercalcemia. A J wave was first seen in hypercalcemia by Kraus.1) The use of diuretics in cases with hyperparathyroidism decreases the excretion of calcium (Ca), which causes changes in bone-calcium turnover and parathyroid hormone activity, and forms hypercalcemia in a complex way.2) Here, a hypercalcemic case with primary hyperparathyroidism aggravated by the use of a thiazide diuretic in whom we electrocardiographically observed a J wave is presented.

CASE REPORT

A 67-year-old female patient had undergone coronary angiography a week earlier due to chest pain and her coronary arteries were found to be normal. Thallium-201 scintigraphy, performed in order to determine silent myocardial ischemia, was normal. She was admitted to our hospital with complaints of weakness, nausea, vomiting, and constipation one week after the treatment was started.
On physical examination, her blood pressure was 120/80 mmHg, pulse 45/minute, and temperature 36.5°C. A fourth heart sound (S₄) and a grade 2/6 systolic murmur were clearly heard at the second right intercostal space and cardiac apex. On ECG, ST elevation was present in leads II, III, aVF and V₄ to V₆, and ST depression was present in leads I and aVL. The ultrasonic echocardiographic examination findings were as follows; left atrial diameter: 37 mm, left ventricular diastolic diameter: 47 mm, left ventricular systolic diameter: 29 mm, ejection fraction: 68%, fractional shortening: 38%, mitral E/A < 1, IVRT: 140 ms, diastolic septal thickness: 16 mm, and diastolic posterior wall thickness: 15 mm. All wall motions were normokinetic. The aortic valve was calcified but no important gradient was detected. The mitral and tricuspid valves were normal. Blood urea nitrogen, creatinine, Na, K, and Ca²⁺ levels were 58 mg/dL, 2.5 mg/dL, 138 mmol/dL, 2.9 mmol/dL, and 18.5 mg, respectively. An ECG showed ST segment elevation in leads V₄ to V₆, II, III, and aVF with simultaneous depression in I and aVL. Her ECG was normal in August 1998 (Figure 1) and J waves were seen in her ECG in August 2000 (Figure 2). In hypercalcemia, the PR and QRS intervals lengthen and QRS amplitude increases. Another frequently encountered ECG finding is lengthening of the ST segment duration. In the ECG in our case, the

Figure 1. Normal ECG in August 1998.
QRS interval was 0.14 seconds, (it was 0.08 seconds 2 years earlier). Her QRS amplitude was 1.8 mV in I, and 1.4 mV in aVL (they were 1.3 mV in I, and 0.9 mV in aVL in the previous ECG). It was observed that these changes decreased to previous values associated with the decrease in calcium levels. The PR interval was normal. Considering that hypercalcemia might be associated with the use of thiazide, hydrochlorothiazide was stopped, following which her Ca level decreased to 14.9 g/dL and the K values returned to normal. As calcium levels did not completely decrease to normal, the etiologically measured parathyroid hormone level was higher than the normal 1070 pg/mL (normal range 9-55 pg/mL).

Parathyroid ultrasonography revealed a hypoechoic nodule with dimensions of 16 and 10 mm at the left upper lobe. Parathyroid scintigraphy was performed with Tc-99m MIBI. Increased activity was obtained in the left upper lobe and this activity persisted during late images, which is reported to be consistent with parathyroid adenoma. After parathyroidectomy her blood calcium level dropped to 10.5 g/dL. the ECG J wave had completely disappeared (Figure 3). Findings of hypercalcemia associated with serum calcium levels. There is usually no symptoms at calcium levels of 11.5-12 mg/dL. Findings of hypercalcemia on kidney, skin, cardiovascular system and gastrointestinal system occur at levels of 13 mg/dL.

Figure 2. Leads II, III, V₆, V₅, and V₄ showing J waves (arrows) during hypercalcemia (Ca⁺⁺ 18.5 mg/dL).
DISCUSSION

In our case, complaints and findings which were in agreement with high calcium levels were present. Calcium is rarely above 14 mg/dL in primary hyperparathyroidism and higher values are seen in parathyroid cancers. In our case, we believe the hypercalcemia was aggravated by the use of a thiazide diuretic which caused symptoms associated with hypercalcemia to occur. Thiazide diuretics are known to cause hypercalcemia. However, to the best of our knowledge, this is the first case in which hypercalcemia was aggravated by the use of a thiazide diuretic in a hypercalcemic case with primary hyperparathyroidism and J waves. As with hypothermia, the abnormality may occasionally, in some leads, appear more as a widening and notching of the QRS instead of a clear-cut separated J deflection. The pathogenesis of these transient J waves is unclear. Such an ST-junctional change could result from selective abbreviation of phase 2 of cellular action potentials in the subendocardium as compared with those in the subepicardium. After it was described by Kraus, J waves in hypercalcemia were reported in the presentation of a few cases in humans. In 1984, Sridharan, et al described the presence of J waves in 2 patients with hypercalcemia associated with malignancies. Douglas, et al reported one case with parathyroid adenoma.

Figure 3. Twelve-lead electrocardiogram at serum calcium concentration of 9 mg/dL after parathyroidectomy.
Transient J waves have been reported in rare cases although ECG changes associated with hypercalcemia are frequently seen. The presence of J waves is an indication of serious hypercalcemia.

REFERENCES