CASE REPORT

A Rare Electrocardiographic Manifestation of a Rare Form of Multiple Electrolyte Disturbances: Hyperparathyroid Crisis

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Abstract- The surface electrocardiogram (ECG) has been used as a useful method for detection of metabolic disturbances for a long time. However, it may be difficult to distinguish the exact disturbance when more than one metabolic abnormality exists in a patient simultaneously. Although, "classic" ECG characterizations of common electrolyte disturbances are well described, multiple concurrent electrolyte disturbances may lead to ECG abnormalities that may not be easily detectable. This ECG concerns a 60-year-old male presented with general fatigue, weakness, epigastric pain, anorexia, nausea and extreme hypercalcemia (serum total and ionized calcium levels 20.5 mg/dL and 12.02 mg/dl, respectively), hypokalemia and hypomagnesemia associated with elevated parathyroid hormone (1160 pg/ml) and normal serum vitamin D level (97 ng/ml) . This rare manifestation of primary hyperparathyroidism has been named hyperparathyroid crisis in the literature. Hyperparathyroid crisis is an emergency form of multiple electrolyte abnormalities that manifest as a life-threatening hypercalcemia and simultaneous hypokalemia and hypomagnesemia; these two later are believed to be caused by diuretic effect of calcium on the renal tubules. The unique pattern of ECG in our patient first was misdiagnosed as prominent T waves with prolongation of the QT corrected (QTc) interval, which has been reported several times in patients with hyperparathyroidism crisis, compatible with our patient. But more investigation revealed that, the QTc interval not only is not prolonged, it is shortened as it is expected from the effect of hypercalcemia on electrocardiogram. The exact pattern of the patient`s ECG (Figure 1) can be interpreted as it follows: (1) Flattening of the T wave, (2) a prominent U wave, (3) prolongation of the descending limb of the T wave such that it overlapped with the next U wave (4) virtual absence of ST segment and (5) shortening of the QT corrected interval. In conclusion, it should be emphasized when the T and U waves are separated by a very short segment they can mimic the appearance of a prolonged QT interval. However, more investigation can demonstrate the exact electrocardiographic pattern especially in multiple electrolyte disturbances, when "classic" ECG patterns are not expectable.

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Keywords: Electrocardiography; Electrolyte disturbances; Hyperparathyroidism

Introduction

The surface electrocardiogram (ECG) has been used as a useful method for detection of metabolic disturbances for a long time. While "classic" ECG patterns of common electrolyte imbalances are well recognized, the presence of multiple electrolyte disturbances may result in ECG patterns that are not easily identifiable. Hyperparathyroid crisis characterized by severe hypercalcemia and concurrent hypokalemia is an example of how multiple electrolyte disturbances can modify ECG components.

Case Presentation

A 60-year-old male was admitted to our hospital with a chief complaint of one month-lasting general fatigue and weakness, getting worse in the recent 5 days. He also complained of epigastric pain, anorexia, nausea and episodes of vomiting beginning from previous month and leading to a 9 kg loss of his body weight. In addition, in the recent 6 months he had been suffered from bone pain in his legs which has become more disabling in the recent weeks. He also had polyuria enough to make him incontinent from one week prior admission. His past medical history included a 10 year

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history of hypothyroidism, which was controlled with levothyroxine. His vital signs included a blood pressure of 80/pulse mm Hg, a heart rate of 120 beats per minute, a respiration of 30/min and a temperature of 36.5°C. Signs of severe dehydration, drowsiness, diffuse weakness with hypotonia in extremities were evident on physical examination. The rest of examination was otherwise unremarkable. Initial laboratory data showed hyponatremia with serum sodium level of 129 mEq/L, hypokalemia with serum potassium of 3 mEq/L and hypomagnesemia with serum magnesium level of 1.5mg/dl. Serum creatinine level was 3.28mg/dl and serum urea nitrogen was 86 mg/dL. He had serum calcium and inorganic phosphorus levels of 20.5 mg/dL (ionized calcium: 12.02 mg/dl) and 4.1 mg/dl respectively.

The ECG (figure 1) demonstrated normal sinus rhythm at 120 beats per min; QT interval of 0.26 sec (corrected QT interval: 0.31); and virtual absence of ST-segment. Serum level of troponin I was elevated (9.66ng/mL). Transthoracic echocardiographic examination demonstrated normal left ventricular function with ejection fraction of 50% to 55%. No sign of regional wall motion abnormalities was evident. There was no pericardial effusion and the pericardium appeared normal.

Given the high serum calcium level, and electrolyte abnormalities, the serum levels of parathyroid hormone and vitamin D were requested and the patient was treated with aggressive hydration and diuresis, pamindronate, calcitonin and KCl. Unfortunately no decrease was seen in serum calcium level and patient died due to cardiopulmonary arrest; before a trial of hemodialysis for reduction of serum calcium can be made. On the following day, the analysis of the patient`s blood sample revealed serum levels of parathyroid hormone (PTH) and vitamin D being 1160 pg/ml (reference values) and 97 (reference values), respectively.

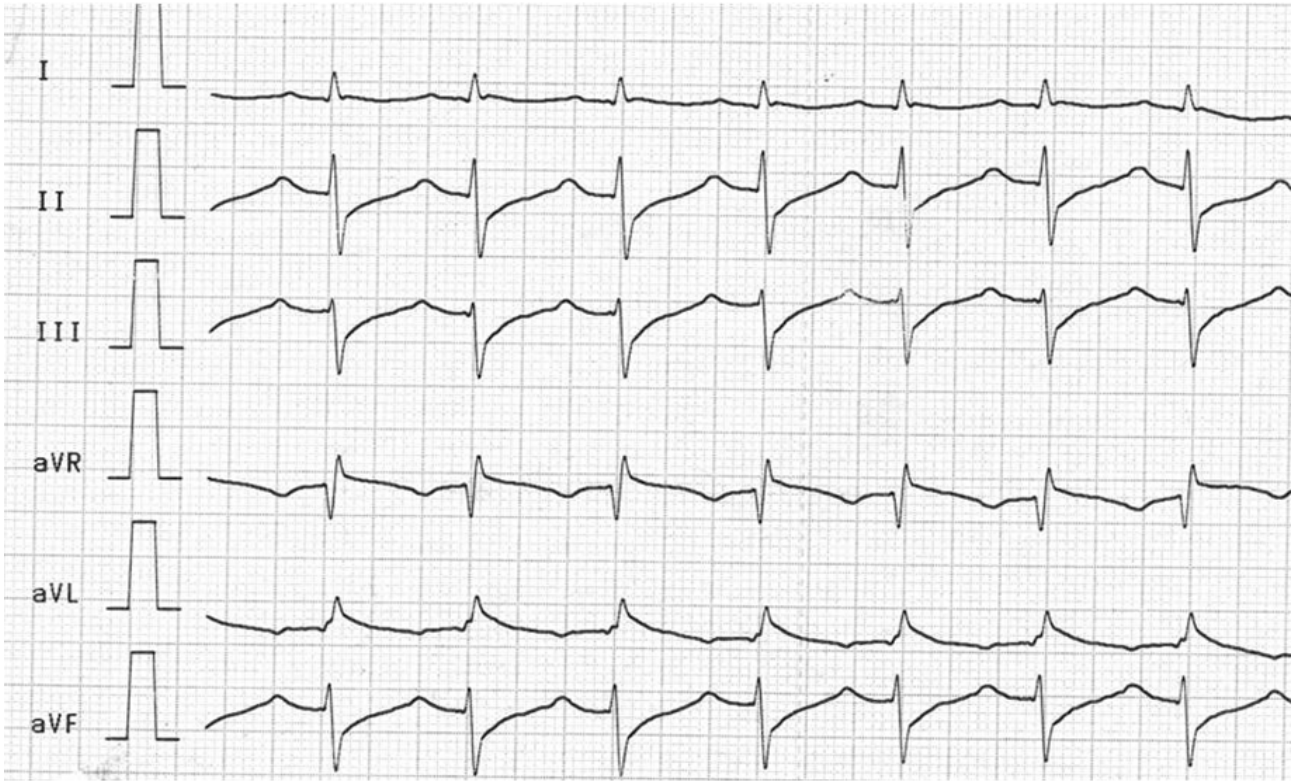

Figure 1. 12-lead electrocardiography of a 60 year-old male with hyperparathyroidism crisis: Note the following: (1) Flattening of the T wave, best shown in V6 (dashed arrows), (2) prominent U waves especially in leads V1, V5 and V6 (arrows), (3) virtual absence of ST segment and (4) shortening of the QT corrected interval. QoT or QaT and QeT intervals are shown with horizontal lines in V6 from above to bottom, respectively. The measured QT intervals are as follows: QoT: 0.12s; QaT: 0.20s, QeT: 0.24s and the corrected QT intervals are QoT: 0.15s; QaT: 0.25s, QeT: 0.31s. A; limb leads, B; precordial leads.

Discussion

The action potential in cardiac cells is generated by movement of ions across the cardiac cell membrane. Any alteration in the normal levels of these ions may lead to altered electrical activity. These changes may manifest as abnormalities in the surface electrocardiogram (ECG) and therefore, provide a useful instrument in the detection of metabolic disturbances. Some characteristic patterns, such as a peaked T wave in hyperkalemia or a shortened QT interval in hypercalcemia are virtually diagnostic. Furthermore, the severity of electrolyte imbalances can sometimes be estimated on the basis of these ECG changes. However, in the presence of multiple electrolyte disturbances, the ECG signal may be significantly distorted, making both the diagnosis of the metabolic disorder(s) and the estimation of severity of the metabolic disturbance(s) more difficult.

Hyperparathyroid crisis, also known as parathyroid storm or acute primary hyperparathyroidism, is a rare but potentially life-threatening endocrine emergency. It is characterized by severe hypercalcemia and is associated with central nervous system dysfunction, bone disease, nephrolithiasis, acute renal failure and cardiac electrophysiologic abnormalities. A wide range of electrocardiographic changes have been described in hyperparathyroidism crisis, and it seems that different mechanisms are responsible for each of them. The electrocardiographic pattern described here (Figure 1) is presumed to be caused by the simultaneous effects of hypokalemia, hypomagnesemia and hypercalcemia together. It is believed that hypercalcemia-induced polyuria leads to increased loss of serum electrolytes such as sodium, potassium and magnesium in the urine which may be high enough to cause hyponatremia, hypokalemia and hypomagnesemia. The electrocardiographic changes in this patient include (1) Flattening of the T wave, (2) a prominent U wave, (3) prolongation of the descending limb of the T wave such that it overlapped with the next U wave (4) virtual absence of ST segment and (5) shortening of the QT corrected interval. This unique pattern of ECG first was misdiagnosed as prominent T waves with prolongation of the QT corrected interval, which has been reported several times in patients with hyperparathyroidism crisis, compatible with our patient. However, more investigation revealed that the QTc interval not only is not prolonged, it is shortened as it is always expected from the effect of hypercalcemia on electrocardiogram.

Ahmed and Hashiba (1) classified QT intervals due to the effect of the shortened ST segment and revealed the intervals QoTc (the interval from the beginning of the QRS complex to the beginning of the T wave) of less than 0.18 second, and QaTc of less than 0.30 second (measured from the beginning of the QRS complex to the apex of the T wave) as reliable indicators of clinically moderate to severe hypercalcemia.

Ashizawa *et al.*, showed that hypercalcemia caused shortening of the QaTc (2). Nierenberg and Ransil found that QaTc correlates more closely than the QoTc or QeTc with the serum calcium levels. QaTc interval of 0.27 second or less was associated with hypercalcemia in more than 90 percent of the cases (3). In another study, by Saikawa *et al.* (4) reliability of corrected QT intervals (QoTc, QaTc, and QeTc) as indicators of clinical hypercalcemia were assessed which showed sensitivity of QoTc, QaTc, and QeTc in predicting high serum calcium was 83%, 57%, and 39%, respectively, and specificity was 100%, 100%, and 89%. They suggested that QT intervals can serve as an indicator of high serum calcium level and that the QoTc seems to be a reliable indicator of the three QTc's. Moreover, Ahmed *et al.* (1) showed that QeTc (the interval from the beginning of the QRS complex to the end of the T wave) interval has neither significant correlation with serum calcium nor any consistent pattern of change with development of hypercalcemia or normalization of serum calcium. Their study showed QoTc and QaTc intervals shorten with development of hypercalcemia and showed significant correlation with serum calcium. Combination of short QoTc (less than 0.18 s) and short QaTc (less than 0.30 s) was found to be highly specific for, and was present in 65% of ECGs, in moderate and severe hypercalcemia. Combination of normal QoTc (greater than 0.18 s) and normal QaTc (greater than 0.30 s) was not observed in moderate or severe hypercalcemia. Bronsky *et al.* (5) found that the QTc interval was inversely proportional to the serum calcium level up to 16 mg/dL. The interval between the onset of QRS and the onset of the T wave (QoTc) became shorter at higher calcium levels, although the T wave was prolonged and the QT interval became more normal. Lind and Ljunghall (6) examined ECG changes of 139 patients with primary hyperparathyroidism. A decrease in the ST segment and the QoTc interval correlated significantly with increasing serum calcium concentration. They reported that hypercalcemia usually does not alter the morphology of T wave, but a slight increase in T wave duration maybe is possible. This effect may sometimes produce a prolonged QT interval,

but the calculation of the corrected QT interval based on QoTc or QaTc may help in estimating its precise length.

As in most studies it is reported that QoTc and QaTc are more reliable indicators of hypercalcemia, it can be concluded that the main effect of hypercalcemia on QT interval is shortening of the ST segment duration rather than the duration of T wave. The very short or virtually absent ST segment in this ECG may be due to the sever hypercalcemia of our patient. The prominent U wave and the flattened T wave in this ECG may be a result of the concurrent hypokalemia. It can be concluded in the presence of hypokalemia along with hypercalcemia, the effect of calcium is yet maintained by the fact that it truly shortens ST segment *i.e.* QoTc and QaTc. Although it may prolongs QeTc, because of the increase in T wave duration. In conclusion, this case illustrates an interesting example of how multiple electrolyte abnormalities may modify various components of the ECG. The result in this case was an interesting combination of effects including Flattening of the T wave, a prominent U wave, prolongation of the descending limb of the T wave such that it overlapped with the next U wave, virtual absence of ST segment and shortening of the QT corrected interval. This appearance was attributed to a significant electrolyte disturbance consisting of concurrent hyperkalemia, hypocalcemia, and hypomagnesemia. Early recognition of this sign may contribute to rapid correction of electrolyte imbalance, preventing potential serious complications.

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