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Case Report

Monophasic Action Potential-Like Electrocardiogram Simulating Acute Myocardial Infarction

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Abstract. **Monophasic action potential-like electrocardiogram simulating acute myocardial infarction has been reported in patients with hyperkalemia. We report two cases presenting marked diffuse RS-T elevation resembling monophasic action potential. They had abnormal electrolyte levels, including hyperkalemia and hypocalcemia in case 1 and hypokalemia and hypocalcemia in case 2. The ECG abnormality returned to baseline after correction of potassium and calcium. We conclude that monophasic action potential-like electrocardiogram is caused by electrolyte disturbance other than acute myocardial infarction.**

Key Words. **electrocardiogram, electrolyte**

Introduction

Electrocardiographic changes, such as marked ST elevation simulating acute myocardial infarction, have been reported in patients with hyperkalemia, especially in diabetic ketoacidosis accompanying renal failure [1–4]. Levine and associates reported four cases with severe hyperkalemia presenting ST elevation; the electrocardiographic changes returned to normal after the serum potassium level was lowered by hemodialysis [1]. They called the marked ST elevation and upward coving resembling acute myocardial infarction or pericarditis the "dialyzable currents of injury." Chou also presented a case with diabetes and ketoacidosis [4]. The marked diffuse ST elevation involved all 12 leads except aVR and V1, resembling monophasic action potential. In the literature, most cases with pseudoinfarction pattern of ECG were caused by hyperkalemia [1–4]. In the following two cases, we present monophasic action potential-like ECG caused by abnormal electrolyte levels other than myocardial infarction.

Case 1

An 86-year-old female patient had a history of chronic renal insufficiency for 4 years and developed an episode of acute loss of consciousness. She was brought to our emergency department. On presentation, she had a serum potassium level of greater than 6.5 mEq/L (normal range 3.6 to 5.2 mEq/L), calcium 7.6 mEq/L (normal range 8.4 to 10.2 mEq/L) and a blood PH value of 7.298. ECG showed diffuse RS-T segment elevation involving all leads except aVR, aVL and V1 (Fig. 1A). The morphology of QRST complexes resembled a monophasic action potential. She was transferred to intensive care unit with a diagnosis of hyperkalemia. After treatment with intravenous calcium gluconate, sodium bicarbonate and emergent hemodialysis, serum potassium level returned to normal one day later (4.5 mEq/L). Because there was no significant change in QRS-T complex, myocardial infraction was considered. However, serum cardiac enzymes had not increased significantly. Recheck of serum electrolytes showed still hypocalcemia (calcium 6.9 mEq/L). After further correction of hypocalcemia, the RS-T segment elevation returned to normal two days later (Fig. 1B). We concluded that this monophasic action potential-like ECG was caused by severe hyperkalemia combined with hypocalcemia, rather than myocardial infarction.

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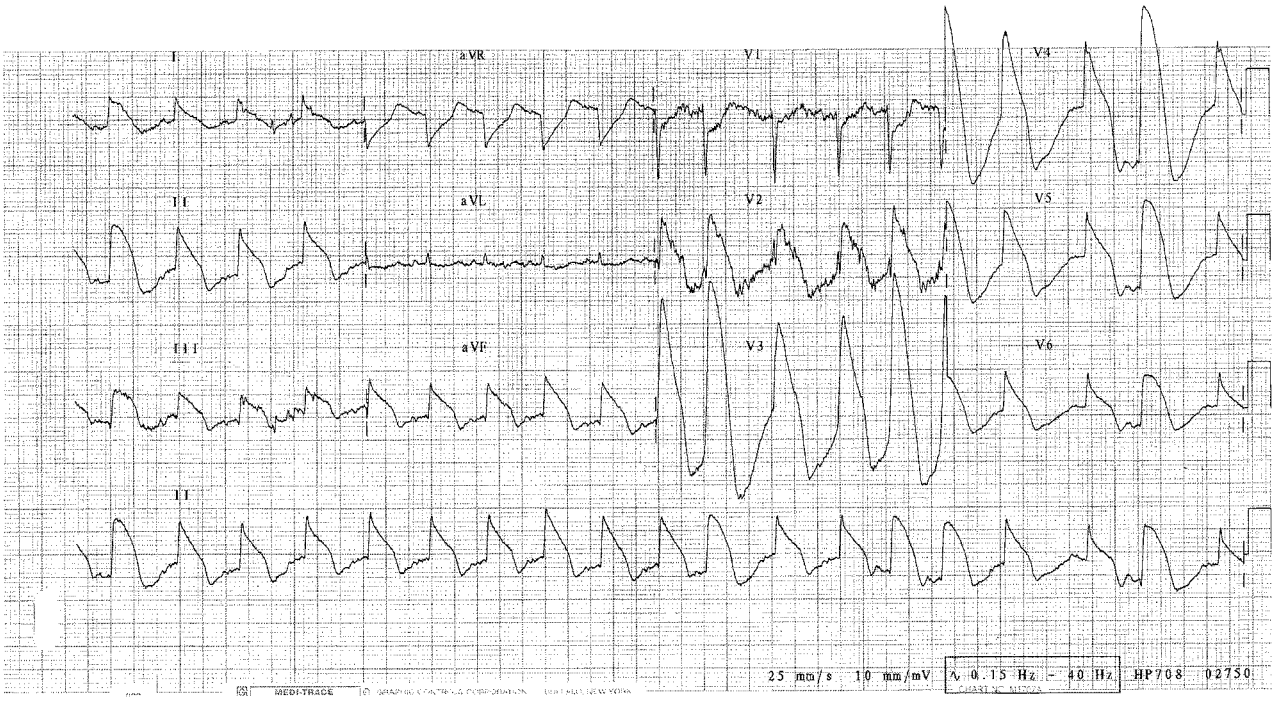
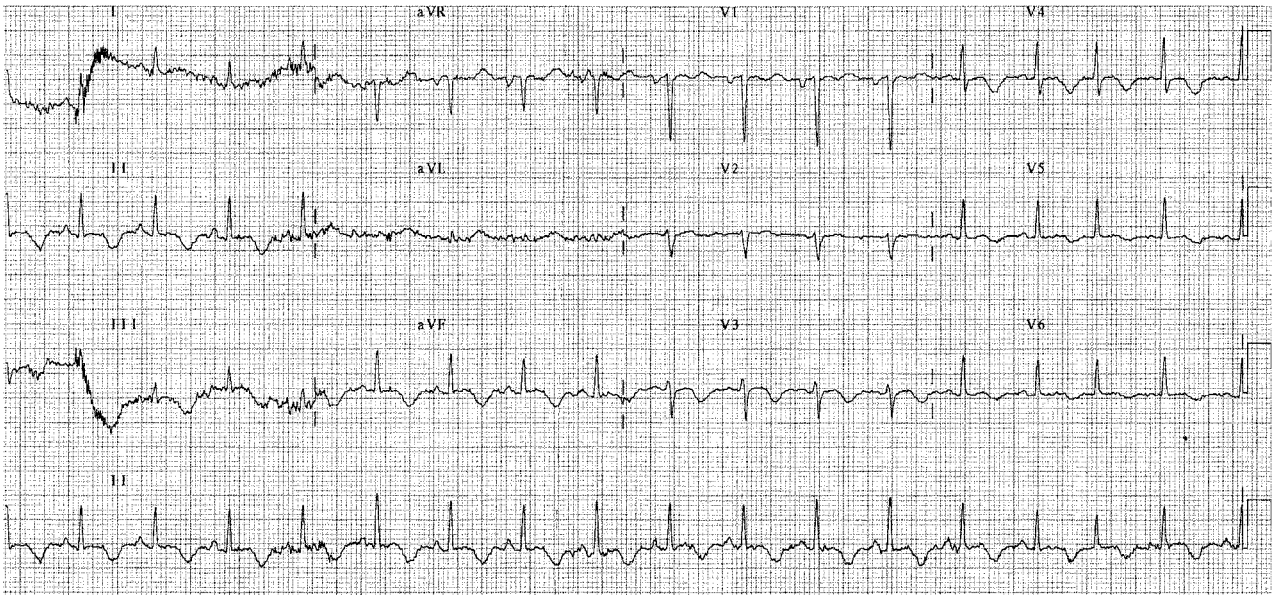
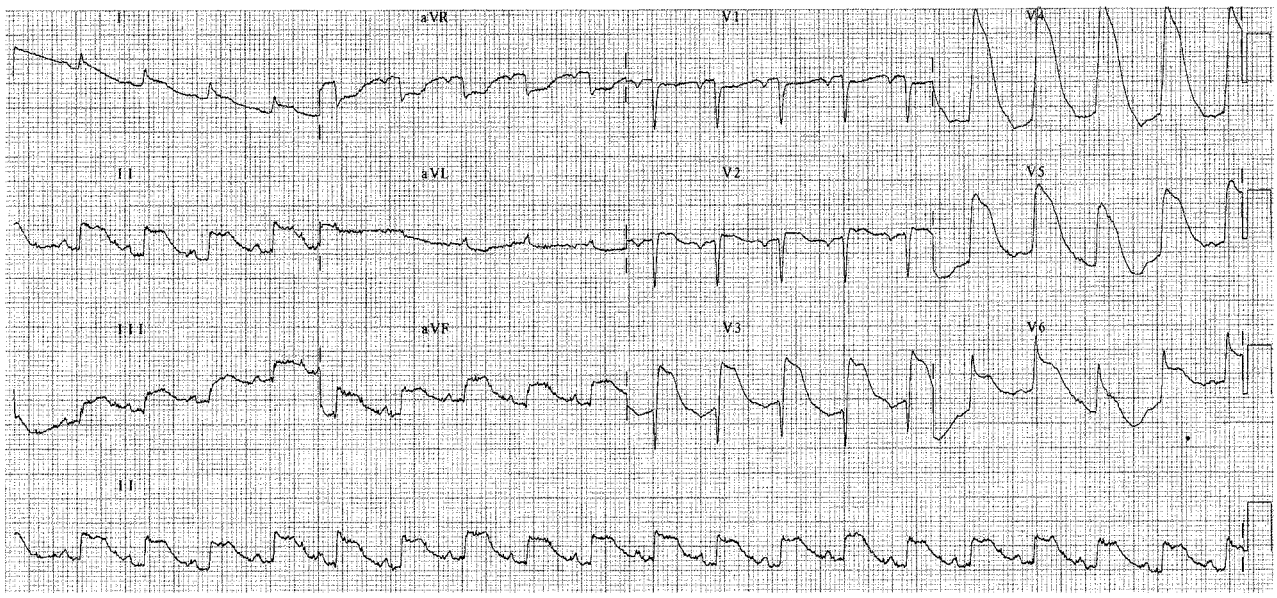


(A)

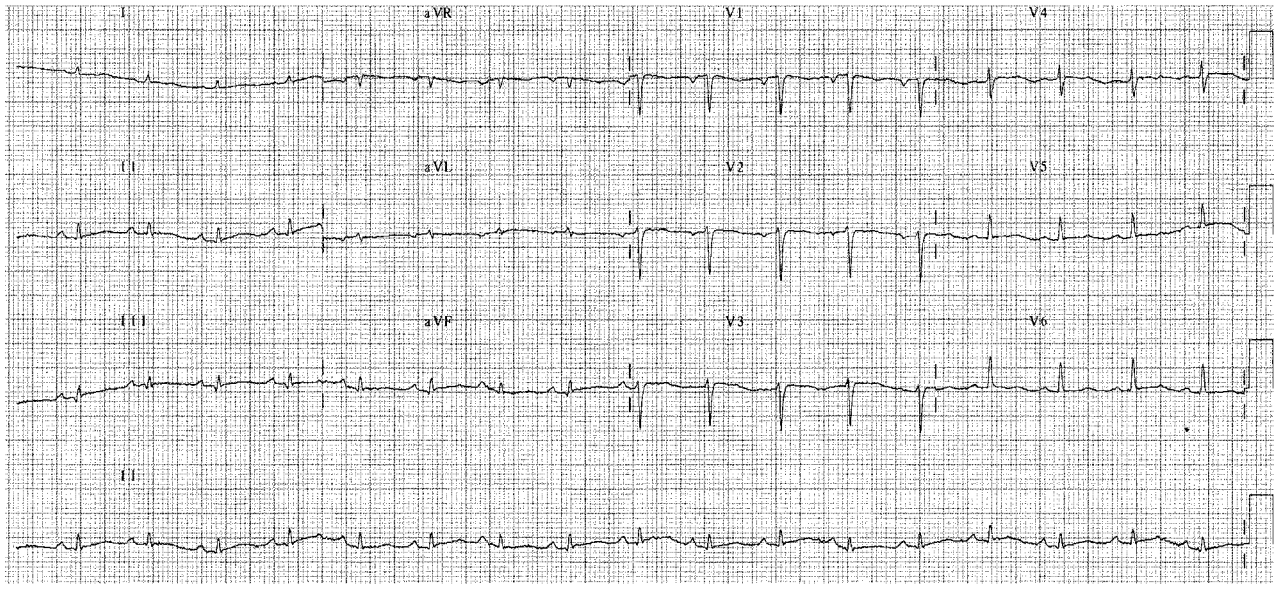


(B)

Fig. 1. *Monophasic action potential-like ECG in a patient with renal insufficiency and hyperkalemia (>6.5 mEq/L), hypocalcemia (7.6 mEq/L) (case 1). (A) Initial ECG on presentation to emergency department showed diffuse RS-T elevation in all leads except aVR, aVL and V1, which showed depressed. The morphology of the QRS-T complex resembled monophasic action potential. (B) Follow-up tracing 2 days later; the RS-T elevation returned to baseline, with T inversion in inferior leads V3-6 and prolonged QT interval after correction of electrolyte imbalance.*



(A)



(B)

Fig. 2. *Monophasic action potential-like ECG in a patient with subarachnoid hemorrhage and hypokalemia (2.6 mEq/L), hypocalcemia (7.8 mEq/L) (case 2). (A) Initial ECG recorded at intensive care unit showed diffuse RS-T elevation in all leads except aVR, aVL and V1. The QRS-T morphology was similar to monophasic action potential. (B) Three days later, after correction of electrolyte imbalance, the RS-T segment returned to normal.*

Case 2

A 74-year-old male patient had right hemiparesis after a cerebrovascular accident attack 9 years before. He was sent to our emergency department by his family after being found in an unconscious state at home. The patient was transferred to intensive care unit because of his acute respiratory distress. Brain CT scan showed subarachnoid hemorrhage. He was noticed to have wide QRS-T complex waves shown on the ECG monitor (Fig. 2A). This was misinterpreted as ventricular tachycardia by a neurologist. Then he was given intravenous xylocaine 100 mg followed by cardioversion with 100 joules. The

abnormal ECG morphologies persisted. After consultation with a cardiologist, diffuse RS-T segment elevation (resembling monophasic action potential) caused by abnormal electrolyte levels was considered a possibility. Serum electrolyte analysis revealed hypokalemia (K^+ 2.6 mEq/L) and hypocalcemia (Ca^{++} 7.8 mEq/L). The monophasic action potential-like ECG returned to normal 3 days later after correction of hypokalemia and hypocalcemia (Fig. 2B). We therefore concluded that this monophasic action potential-like ECG was caused by hypokalemia and hypocalcemia.

Discussion

Several diseases may present ST segment elevation simulating acute myocardial infarction (pseudoinfarction), and hyperkalemia is a common disease reported in the literature [1–4]. The possible mechanism was hypothesized as "dialyzable currents of injury" by Levine and coworkers in 1956 [1]. In previous reports, most patients with hyperkalemia presenting ST elevation in ECG had diabetes or renal insufficiency [1–4]. The diffuse ST change returned to normal after correction of potassium level. Our cases also had abnormal electrolyte levels, including hyperkalemia and hypocalcemia in case 1, hypokalemia and hypocalcemia in case 2. However, they had similar ECG presentation, like monophasic action potential, and the abnormality could be corrected by treating electrolyte disturbance and/or underlying diseases. We cannot be sure what were the mechanisms of abnormal ECG in two cases. We also cannot exclude the effects of cerebral hemorrhage on the RS-T change in the ECG (maybe both effects due to electrolyte disturbance and cerebral hemorrhage). However, we can confirm that the potassium level was low in case 2, completely different from previous reports.

It is difficult to understand why hypokalemia can also produce ST segment elevation like that produced by acute myocardial infarction. Madias et al. presented a hyperkalemia-like ECG in a patient with hypokalemia undergoing potassium supplement [5]. They found transient ST segment elevation simulating acute myocardial infarction (like hyperkalemia effect) during the course of potassium replacement in a patient with severe hypokalemia, even though the serum potassium level was still low. They raised a hypothesis of "relative hyperkalemia" to explain why hypokalemia induced ST segment elevation. That is, relative hyperkalemia due to change of the intracellular/extracellular potassium ratio resulted in hypokalemia related ST segment elevation.

Several reports have demonstrated that hypocalcemia can increase the effect of hyperkalemia on the ECG [3,6–8]. In our case 1, ST segment elevation did not return to baseline after correction of hyperkalemia, but became normal after further treatment of hypocalcemia. On the other hand, hypocalcemia by itself can also produce unusual QRS-T change simulating acute myocardial infarction; the true mechanism is still unknown [8]. Both cases 1 and 2 had hypocalcemia that may have contributed to the presence of monophasic action potential-like ECG.

In conclusion, we report two cases with an unusual monophasic action potential-like ECG. They had different underlying diseases other than myocardial infarction, and abnormal electrolyte levels other than hyperkalemia. The abnormality disappeared after correction of electrolytes and/or treating underlying diseases. The true mechanisms of monophasic action potential-like ECG are still unknown.

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