Electrocardiographic Manifestations of Hyperkalemia

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Hyperkalemia is one of the more common acute life-threatening metabolic emergencies seen in the emergency department. Early diagnosis and empiric treatment of hyperkalemia is dependent in many cases on the emergency physician's ability to recognize the electrocardiographic manifestations of hyperkalemia. The electrocardiographic manifestations commonly include peaked T-waves, widening of the QRS-complex, and other abnormalities of altered cardiac conduction. Peaked T-waves in the precordial leads are among the most common and the most frequently recognized findings on the electrocardiogram. Other "classic" electrocardiographic findings in patients with hyperkalemia include prolongation of the PR interval, flattening or absence of the P-wave, widening of the QRS complex, and a "sine-wave" appearance at severely elevated levels. A thorough knowledge of these findings is imperative for rapid diagnosis and treatment of hyperkalemia. (Am J Emerg Med 2000;18:721-729.

Hyperkalemia is one of the more common acute life-threatening metabolic emergencies seen in the emergency department (ED). Life-threatening hyperkalemia is most frequently diagnosed in patients with known chronic renal insufficiency or end-stage renal disease. However, significant hyperkalemia may also be found in patients with many other medical conditions, including diabetic ketoacidosis, adrenal insufficiency, acute digoxin toxicity, severe dehydration with resulting acute renal insufficiency, and in association with medications that affect kidney function.

Because of potential laboratory delays in obtaining serum potassium levels in patients, early diagnosis and empiric treatment of hyperkalemia is dependent in many cases on the emergency physician's ability to recognize the electrocardiographic manifestations of hyperkalemia. Peaked T-waves in the precordial leads are among the most common and the most frequently recognized findings on the electrocardiogram (ECG). Other ''classic'' electrocardiographic findings in patients with hyperkalemia include prolongation of the PR interval, flattening or absence of the P-wave, widening of the QRS complex, and a ''sine-wave'' appearance at severely elevated levels. A thorough knowledge of these findings is imperative for rapid diagnosis and treatment of hyperkalemia. Five cases are presented which show the classic electrocardiographic manifestations of hyperkalemia.

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CASE PRESENTATIONS

Case One

A 34-year-old man with a history of sickle cell anemia, hypertension, and end-stage renal disease presented to the ED complaining of generalized malaise and myalgias. He reported having missed his last dialysis session 2 days earlier. On examination, he appeared comfortable and alert. He was afebrile, pulse 90 beats/min, respiratory rate 26 breaths/min, pulse oximetry 94%, and blood pressure 165/90 mm Hg. His examination was notable only for bibasilar rales on lung examination. The patient was placed on a cardiac monitor and an ECG was obtained (Fig 1), showing normal sinus rhythm with a first-degree AV block, diffuse T-wave inversions, peaked T-waves, and slight widening of the QRS complex. Empiric treatment for hyperkalemia was initiated.

Serum chemistries obtained on the patient revealed a sodium level of 134 mEq/L, potassium 8.4 mEq/L, chloride 129 mEq/L, bicarbonate 18 mEq/L, blood urea nitrogen (BUN) 37 mg/dL, and creatinine 13.9 mg/dL. A repeat ECG was obtained after he had been treated with 20 mL of intravenous 10% calcium gluconate, 100 mEq of intravenous sodium bicarbonate, 10 units of intravenous insulin (with D50), and 50 grams of oral Kayexalate (Fig 2). The ECG shows normalization of the PR and QRS intervals, reversion of the inverted T-waves in the inferior leads, and most notably, loss of the peaked T-waves. The patient underwent emergent hemodialysis and had an uneventful hospital course. His ECG did not change from that shown in Figure 2 after hemodialysis.

Case Two

A 64-year-old woman with a history of diabetes, hypertension, and end-stage renal disease presented to the ED complaining of diffuse weakness for 24 to 36 hours. She was due for routine hemodialysis on the day of presentation, but she came to the ED instead because of the weakness. On examination, she appeared comfortable and alert. Vital signs showed a normal temperature, pulse 100 beats/min, respiratory rate 22 breaths/min, pulse oximetry 97%, blood pressure 163/107 mm Hg. A fingerstick glucose was 200. Her examination was otherwise unremarkable. An ECG was obtained (Fig 3), showing an irregular rhythm, absence of regular distinct P-waves, and a ventricular rate of approximately 75 beats/min. There were frequent pauses noted on cardiac monitor and ECG, as well as peaked T-waves, widening of the QRS complex, leftward axis, and left anterior fascicular block.

Serum chemistries obtained were notable for a sodium level of 136 mEq/L, potassium 8.0 mEq/L, chloride 90

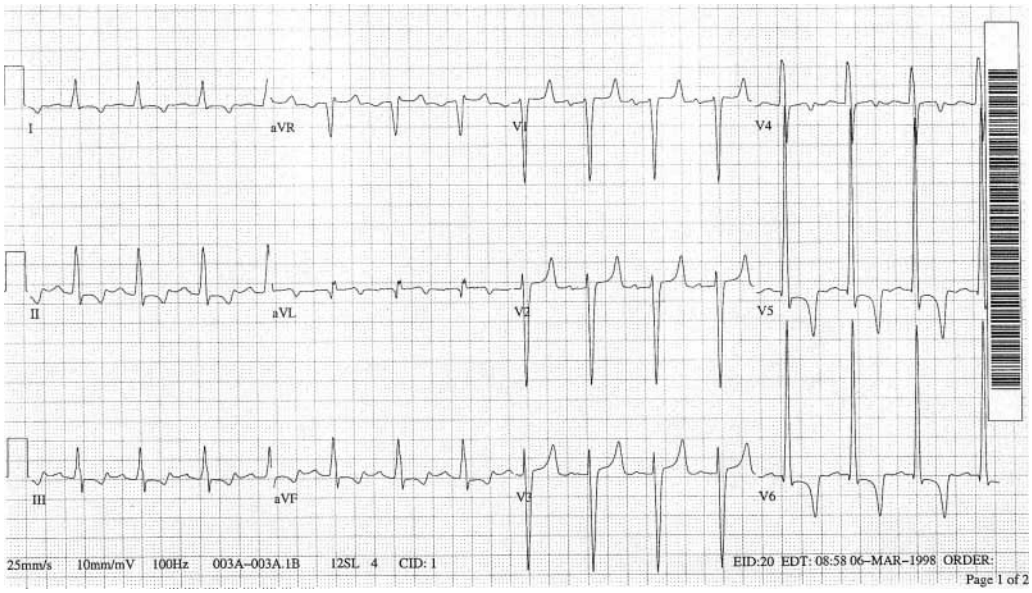

FIGURE 1. Case One. ECG showing NSR with prominent T waves in the right to mid precordial leads (V1 to V3). In addition, T wave inversions are seen in the inferolateral distribution, first-degree atrioventricular (AV) bock, and minimal widening of the QRS complex are seen.

mEq/L, bicarbonate 25 mEq/L, BUN 118 mg/dL, creatinine 18.3 mg/dL, and glucose 240 mg/dL. The patient was treated intravenously with 20 mL of 10% calcium gluconate, 100 mEq of sodium bicarbonate, and 10 units of insulin (glucose 50% solution). The patient was also treated with continuous albuterol nebulization and 50 grams of oral Kayexalate. Two hours after treatment was initiated, a repeat serum potassium level of 6.9 mEq/L was obtained. A repeat ECG was obtained (Fig 4), showing normal sinus rhythm, less ''peaking'' of the T-waves, narrowing of the QRS complex, normalization of the axis, and resolution of the fascicular block. The patient underwent emergent hemodialysis and did well. The ECG before hospital discharge (Fig 5) shows normal sinus rhythm with occasional premature atrial complexes and normal T-waves.

Case Three

A 62-year-old woman with a history of diabetes, hypertension, and congestive heart failure presented to the ED complaining of 3 days of persistent nausea, vomiting, malaise, and lightheadedness. She had no history of renal insufficiency. Her medications were lasix, verapamil, and clonidine. There had been no recent change in her medication regimen. On examination, she appeared comfortable and alert, but became lightheaded when sitting upright or standing. Vital signs included a normal temperature, pulse 50 beats/min, respiratory rate 24 breaths/min, pulse oximetry 96%, and blood pressure 110/70 mm Hg. A fingerstick glucose was 220. The remainder of her examination was notable only for clinical evidence of mild-moderate dehydra-

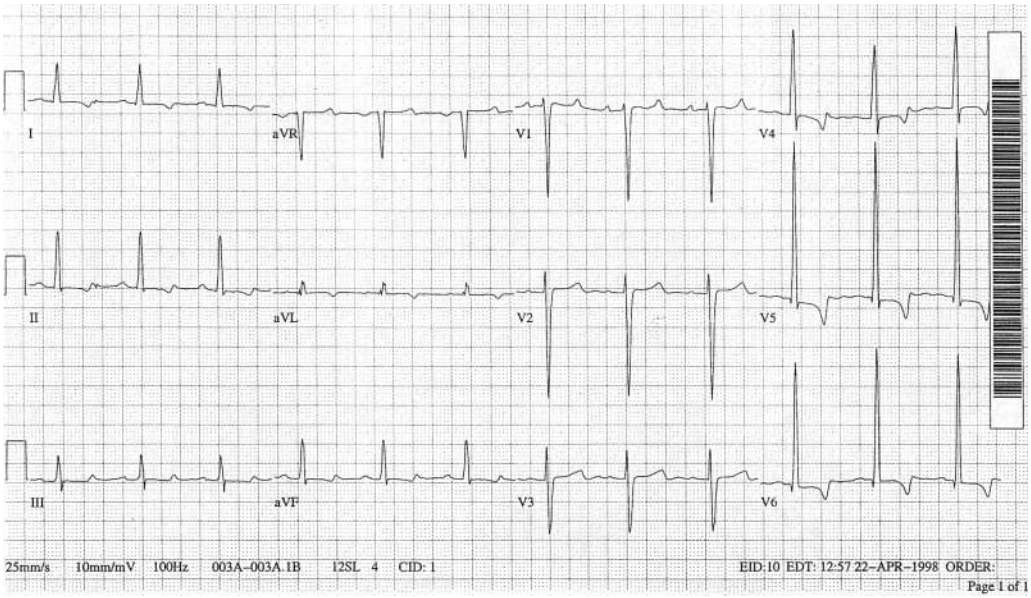

FIGURE 2. Case One. With therapy, the prominent T waves normalized as did the conduction abnormalities (AV block and QRS complex widening).

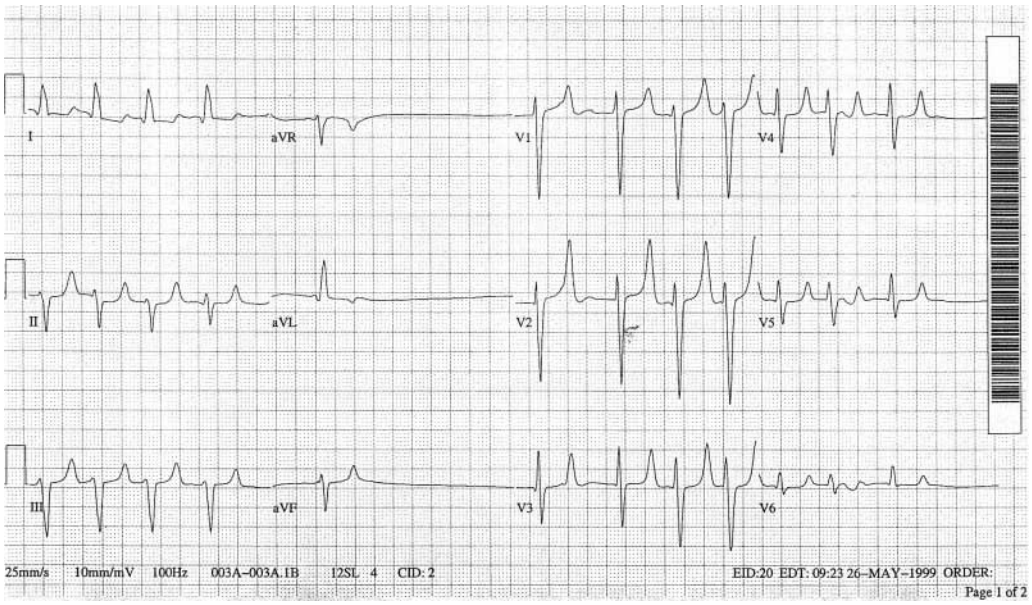

FIGURE 3. Case Two. ECG showing an irregular, narrow QRS complex rhythm and absence of regular distinct P-waves with a ventricular rate of approximately 75 beats/min. There were frequent pauses noted on cardiac monitor as seen on the this ECG. Peaked T-waves, widening of the QRS complex, leftward axis, and left anterior fascicular block (Q-wave in lead I and R-wave in lead III with left axis deviation) are also seen.

tion. Intravenous fluids were administered to the patient and she was placed on a cardiac monitor. An ECG was obtained (Fig 6), showing an atrial rate of 45 beats/min (very small P-waves are best seen in the rhythm strip, lead II), AV dissociation, a junctional escape rhythm with a rate of approximately 50 beats/min, and a capture beat (the seventh QRS complex on the rhythm strip). Note that the T-waves are, at most, minimally peaked, and the QRS duration is normal.

Serum chemistries obtained on the patient were notable for a sodium level of 137 mEq/L, potassium 8.1 mEq/L, chloride 129 mEq/L, bicarbonate 22 mEq/L, BUN 79 mg/dL, creatinine 5.0 mg/dL, and glucose 249 mg/dL. The patient was immediately treated intravenously with 10 mL of 10% calcium chloride solution, 10 units of regular insulin, and 50 mEq of sodium bicarbonate. She was also administered 50 grams of oral Kayexalate. A repeat ECG approximately 60 minutes after treatment was initiated (Fig 7) showed normal sinus rhythm (increased P-wave amplitude) with a first degree AV block. The T-waves are unchanged, and the QRS duration remains normal. The serum potassium as this time was 6.6 mEq/L. The patient's baseline ECG (after normalization of the serum potassium) is shown in Fig 8. The ECG shows normal sinus rhythm with a normal PR interval, and T-wave inversions in the lateral leads. Note that this patient developed ''pseudonormalization'' of the lateral inverted T-waves during the period of hyperkalemia. The remainder of the patient's hospital course was uneventful.

Case Four

A 29-year-old man with a history of chronic renal insufficiency (nondialysis) and hypertension presented to the

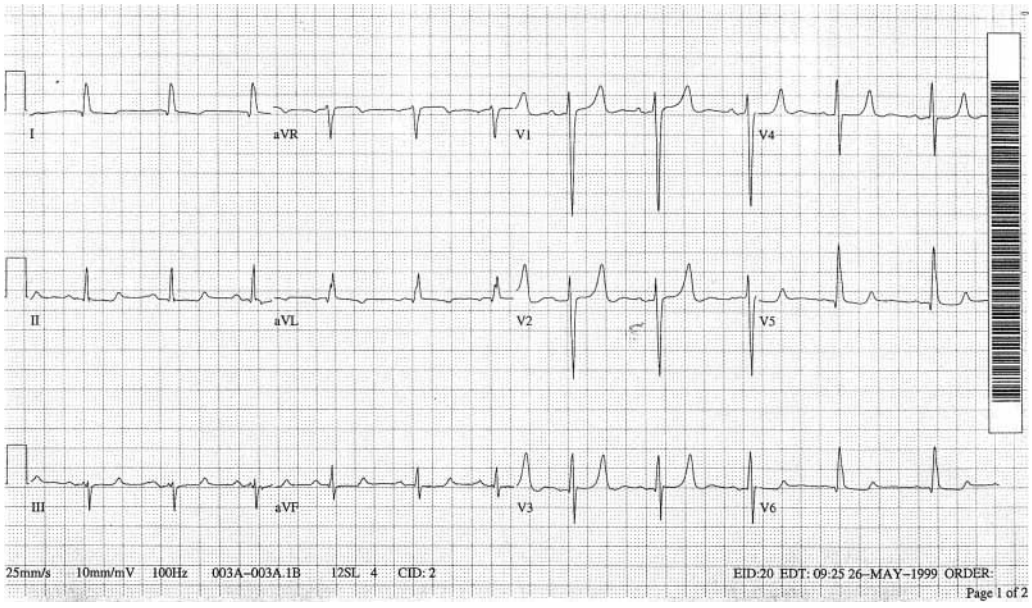

FIGURE 4. Case Two. With therapy, the rhythm normalizes to NSR while the T waves remain prominent.

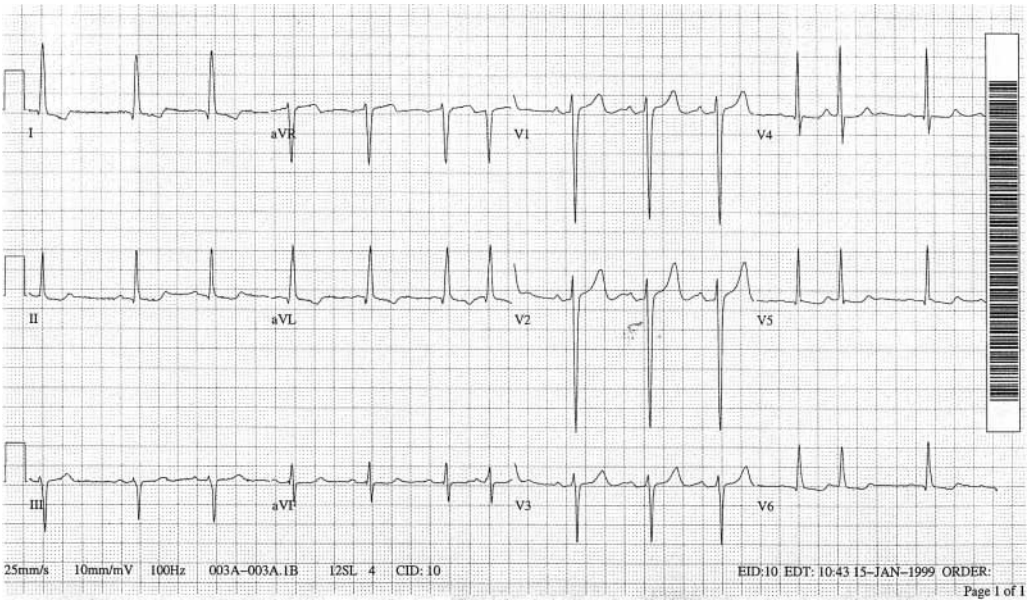

FIGURE 5. Case Two. Additional therapy produces normal T waves and further narrowing of the QRS complex.

ED with a 2-day history of worsening dyspnea, diffuse weakness, subjective fevers, and cough productive of yellow blood-tinged sputum. His only medications were enalapril and an over-the-counter cough suppressant. On arrival, the patient's temperature was 100.6, pulse 90 beats/min, respiratory rate 26 breaths/min, pulse oximetry 91%, and blood pressure 140/90 mm Hg. His examination was notable for dry mucous membranes and decreased breath sounds at the right lung base. The patient was given supplemental oxygen and placed on a cardiac monitor, which showed a wide-complex irregular rhythm with a rate in the 80s-90s. An immediate ECG was obtained (Fig 9) and showed a markedly wide-complex irregular ventricular rhythm without regular distinct atrial activity.

The patient was empirically treated intravenously with 20 mL of 10% calcium chloride solution, 150 mEq of sodium bicarbonate, 10 units of regular insulin (and D50), and 50 grams of oral Kayexalate. Serum chemistries obtained before treatment revealed a sodium of 140 mEq/L, potassium 7.6 mEq/L, chloride 97 mEq/L, bicarbonate 14 mEq/L, BUN 211 mEq/L, creatinine 37.5 mEq/L, and glucose 110 mg/dL. An arterial blood gas on supplemental oxygen showed pH 7.30, pO_2 96 mm Hg, and pCO_2 26 mm Hg. Chest radiograph showed a right lower lobe pneumonia. A repeat ECG was performed one hour later (Fig 10) and shows normal sinus rhythm with occasional premature atrial complexes. The QRS duration is markedly improved, though still slightly prolonged, and the T-waves are minimally peaked.

A Quinton catheter was placed and he was admitted to the intensive care unit for emergent hemodialysis. The patient required regular hemodialysis after discharge from the

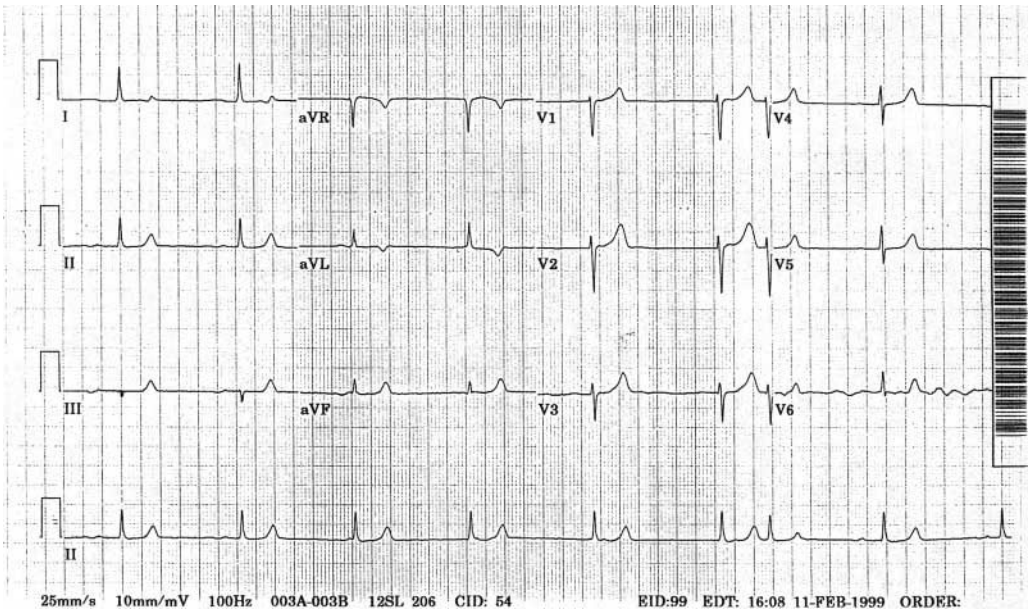

FIGURE 6. Case Three. ECG showing an atrial rate of 45 beats/min (Very small P-waves are best seen in the accompanying rhythm strip—lead II), AV dissociation, a junctional escape rhythm with a rate of approximately 50 beats/min, and a capture beat (the seventh QRS complex on the rhythm strip). Note that the T-waves are, at most, minimally peaked, and the QRS duration is normal.

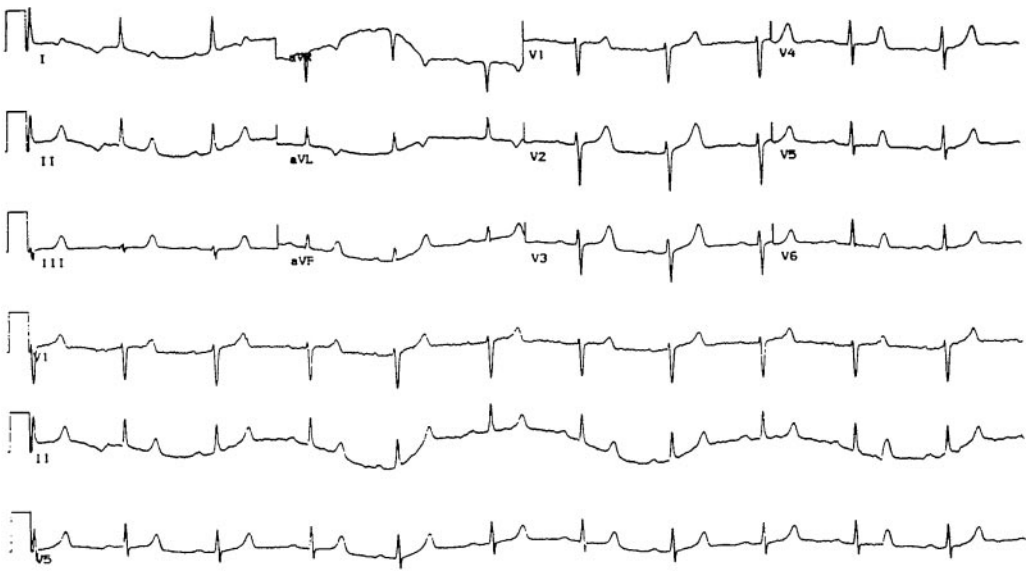

FIGURE 7. Case Three. Improvement in the marked abnormalities is seen—note the persistence of the first-degree AV block and prominent T-waves in the precordial leads.

hospital. His final predischarge ECG (Fig 11) showed little change from his second ECG (Fig 10).

Case Five

A 54-year-old woman presented to the ED with a 2-hour history of nausea, vomiting, and weakness. She had a past history of ischaemic heart disease, diabetes, and hypertension. Over the past week, the patient had consumed a daily average of 2,000 mg of ibuprofen for lower back pain as prescribed by her internist.

On ED arrival, the patient's temperature was normal, pulse 100 beats/min, respiratory rate 36 breaths/min, and blood pressure 100/88 mm Hg. Her examination was notable for dry mucous membranes and a lethargic mental status. The patient was given supplemental oxygen and placed on a cardiac monitor, which showed a wide-complex rhythm. An immediate ECG was obtained (Fig 12), showing a wide QRS-complex rhythm without distinct atrial activity.

The patient was empirically treated intravenously with 20 mL of 10% calcium chloride solution; additional therapy included intravenous sodium bicarbonate, regular insulin, and dextrose as well as oral Kayexalate. Serum chemistries obtained before treatment revealed a sodium of 128 mEq/L, potassium 7.9 mEq/L, chloride 96 mEq/L, bicarbonate 13 mEq/L, BUN 119 mEq/L, creatinine 8.7 mEq/L, and glucose 221 mg/dL.

With the initiation of the parenteral therapy, the QRS complex become more narrow coupled with an increase in rate and the appearance of atrial activity. The patient's mental improved. The nephrologist was contacted; immediate plans for hemodialysis were made while the patient was admitted to the intensive care unit. She was discharged from

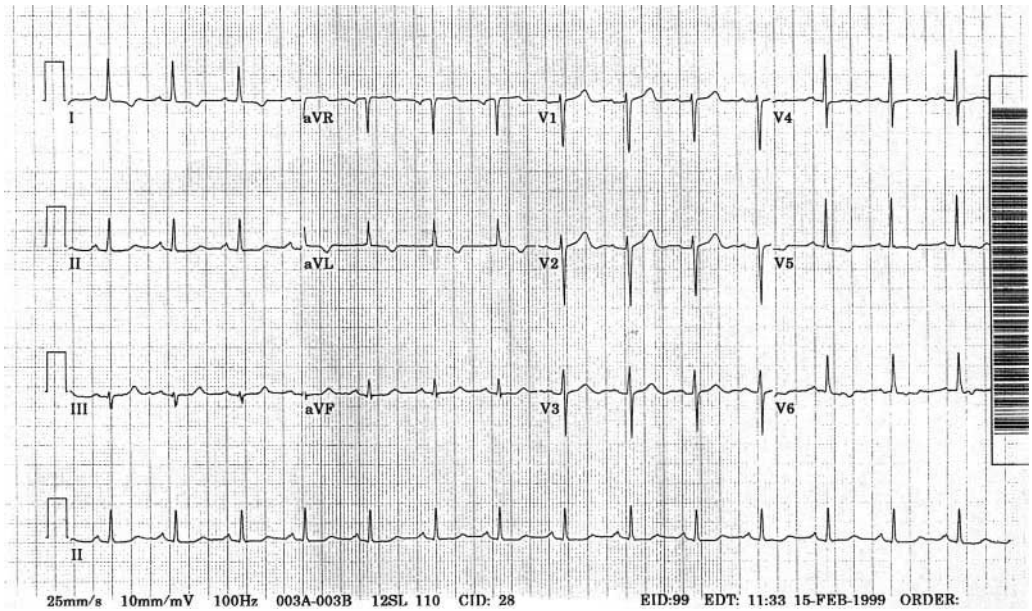

FIGURE 8. Case Three. The ECG has normalized.

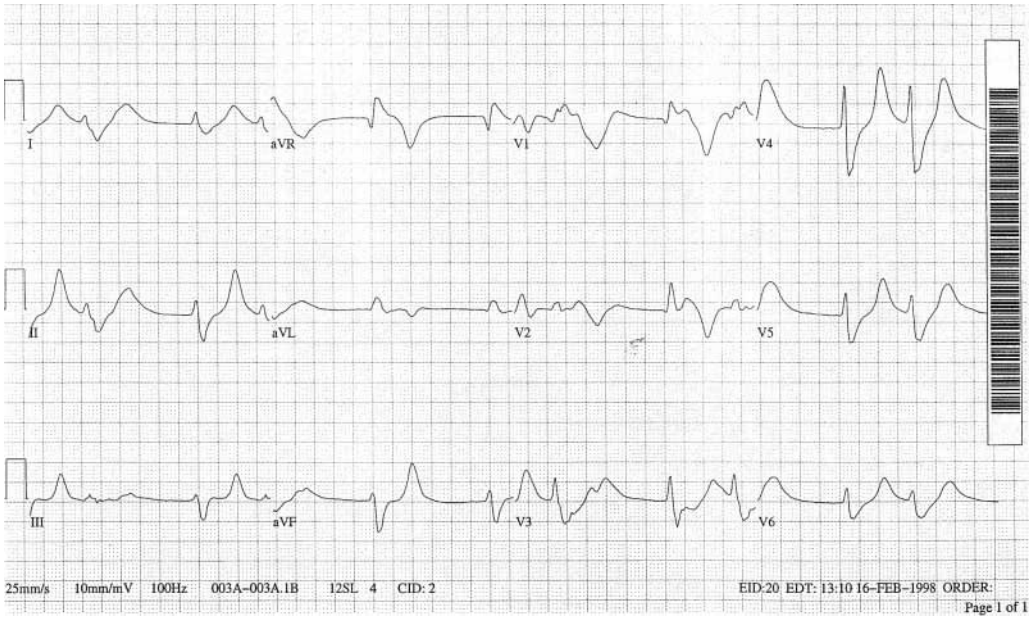

FIGURE 9. Case Four. The ECG showed a markedly wide-complex irregular ventricular rhythm without regular distinct atrial activity—the sinoventricular rhythm.

the hospital 11 days later with minimal renal dysfunction (creatinine 1.7 mEq/L) and the following diagnoses: advanced hyperkalemia and nonsteroidal agent-related renal failure.

DISCUSSION

To understand the electrocardiographic manifestations of hyperkalemia, a brief discussion of electropathophysiology is warranted. Mild levels of hyperkalemia are associated with acceleration of terminal repolarization, resulting in T-wave changes.[1] These T-wave changes are described as ''tenting'' or ''peaking'' of the T-wave (Figs 13 A and D). The ''peaked'' T-wave is generally considered the earliest sign of hyperkalemia.[1] The direction of the T-wave may also change. Cases one and three above show examples of this phenomenon. It is especially common for the normally inverted lateral T-waves of left ventricular hypertrophy to become upright, or ''pseudonormalized.''[2] Mild to moderate hyperkalemia causes depression of conduction between adjacent cardiac myocytes, resulting in progressive prolongation of the PR and QRS intervals as potassium levels rise. Atrial myocytes are most sensitive to hyperkalemia. As a result, P-wave amplitude becomes less prominent early in the course; later, the P-wave may disappear altogether, even in the presence of continued sinus node activity.[3] Some believe that the sinus impulses are conducted preferentially down internodal tracts directly to the AV junctional tissues without depolarizing the atria.[1] This ''sinoventricular'' conduction may still be detected by intracardiac electrocardiography.[3] At progressively more severe levels of hyperkalemia, further depression of sinoatrial and atrioventricular conduction is seen, resulting in the appearance of escape

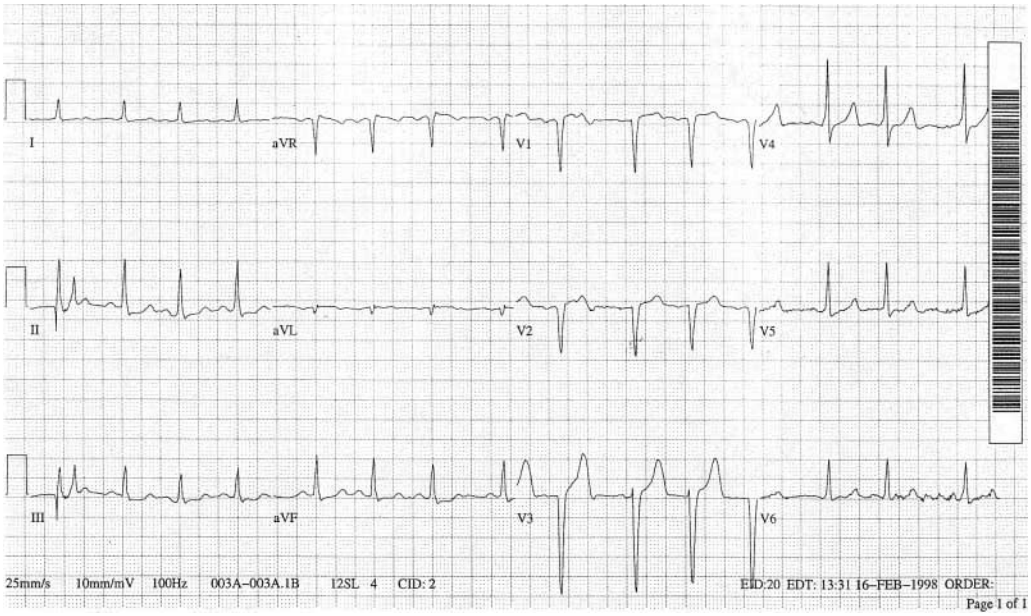

FIGURE 10. Case Four. A repeat ECG was performed 1 hour later, revealing normal sinus rhythm with occasional premature atrial complexes. The QRS duration is markedly improved, though still slightly prolonged, and the T-waves are minimally peaked.

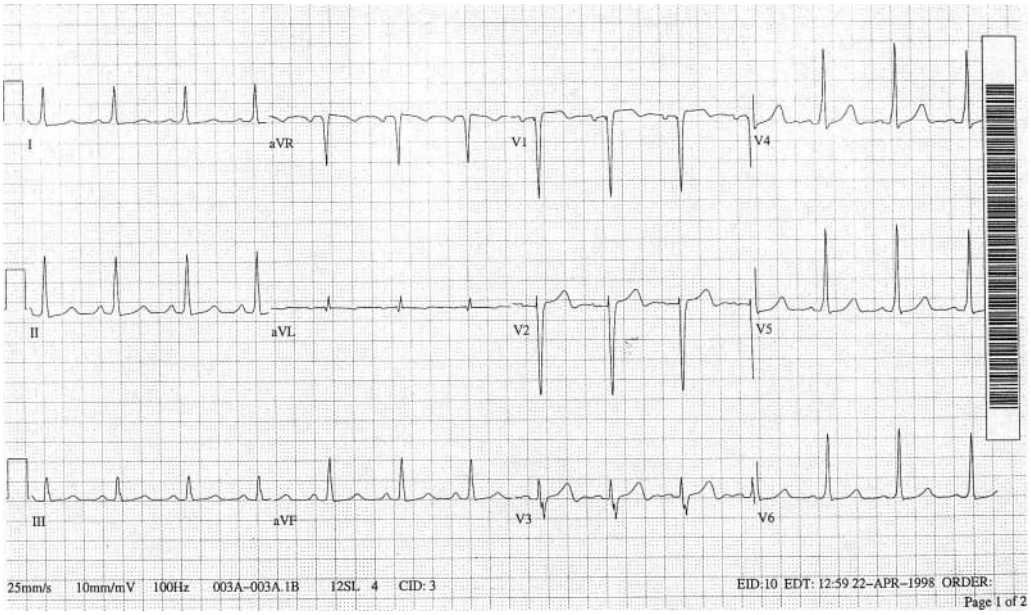

FIGURE 11. Case Four. ECG at hospital discharge revealing normal P-waves.

beats and escape rhythms.[3,4] The QRS complex continues to widen and may eventually blend with the T-wave, creating a ''sine-wave'' appearance to the ECG—producing the sinoventricular rhythm (Figs 13 B, C, and D). Progressive increases in the potassium level eventually result in ventricular fibrillation and asystole.[3]

The expected progression of electrocardiographic changes associated with hyperkalemia is well described in the medical literature (Table 1). It is important to note, however, that the relationship between potassium levels and electrocardiographic changes may vary between different patients. It is also important to realize that the ECG, in contrast with common medical belief, is not always a reliable test for mild to moderate hyperkalemia.[1,5-7] In fact, normal ECGs, ECGs with minimal abnormalities, and ECGs with ''atypical'' abnormalities may be obtained even in patients with severe hyperkalemia.[1,5-6] Case three above shows this point; given the relative absence of peaked T-waves and the normal QRS interval, hyperkalemia was not suspected before obtaining the serum potassium value. The result was a delay in appropriate treatment. In general, however, progressive electrocardiographic changes do occur with rising levels of hyperkalemia and involve all components of the ECG.[1]

Although the electrocardiographic manifestations noted earlier are fairly well known, many less known and less recognized electrocardiographic changes associated with hyperkalemia are also reported in the literature. Because of the depressed atrioventricular conduction, hyperkalemia

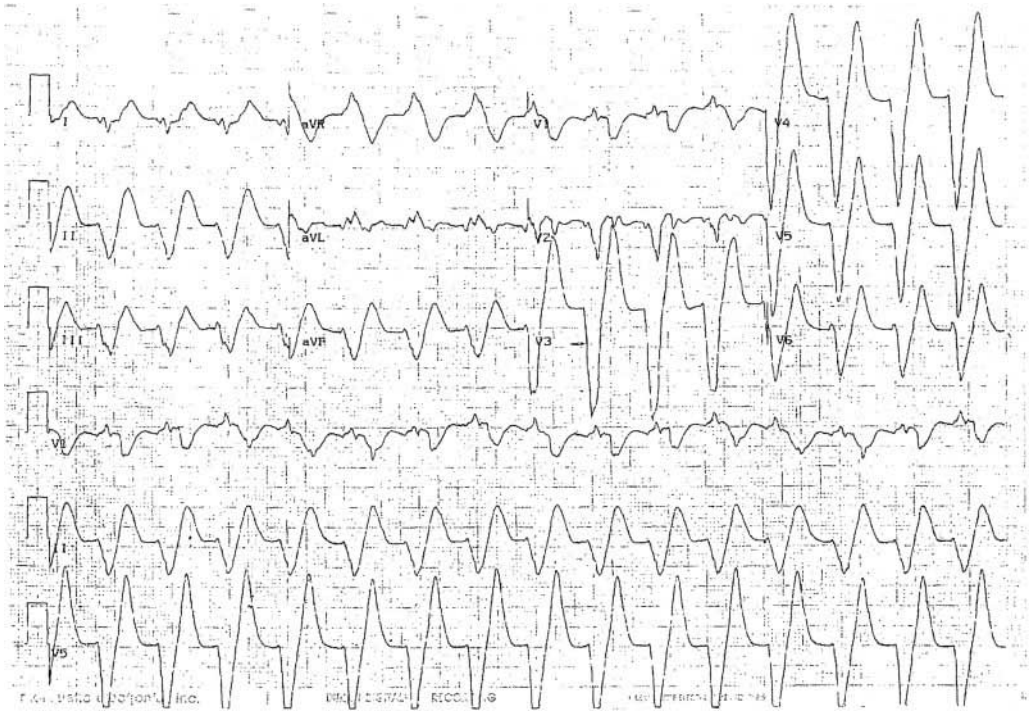

FIGURE 12. Case Five. ECG showing a wide QRS-complex rhythm at a rate of 105 beats/min. No evidence of atrial activity is seen. Furthermore, the QRS-complex, particularly in the inferior leads, has assumed a sine wave configuration, consistent with pronounced hyperkalemia and the sinoventricular rhythm.

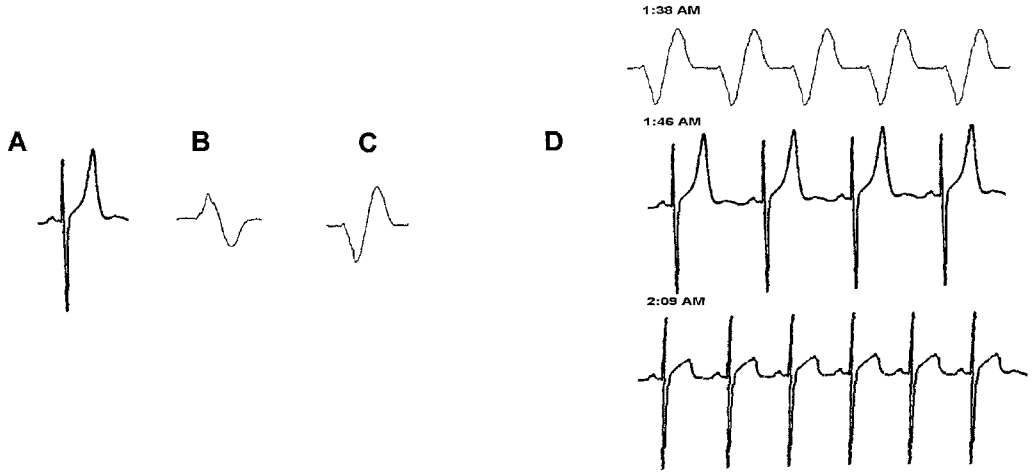

FIGURE 13. The ''classic'' electrocardiographic structures seen in the hyperkalemic patient. (A) The hyperacute or peaked T wave—note the tall, steep contour with symmetry. (B) The sine wave configuration. (C) The sine wave configuration. (D) A patient who had missed hemodialysis twice to attend a friend's wedding presented with weakness and nausea; the initial serum potassium value was 8.3 mEq/dL. The initial ECG revealed the sinoventricular rhythm which responded to therapy—at 1:38 AM, the patient exhibited the sinoventricular rhythm; at 1:46 AM, the QRS complex rapidly narrowed with the restoration of sinus rhythm and the persistence of peaked T waves after the administration of intravenous dextrose, insulin, and sodium bicarbonate as well as nebulized albuterol (calcium was also given); and, at 2:09 AM, the patient's ECG has entirely normalized. Note the normalization of the previously widened QRS complex and peaked T wave.

may result in complete heart block.[3] Severe hyperkalemia has also been reported to cause significantly depressed intraventricular conduction, resulting in a hemiblock, right bundle branch block, left bundle branch block, bifascicular block, or trifascicular block.[1,3,8-11] The intraventricular conduction delay may also cause a shift of the QRS complex axis.[12] Case two above shows both of these findings—a new fascicular block and QRS axis shift, both of which resolved after treatment. Interestingly, hyperkalemia seems to have a greater effect on depressing conduction through bypass tracts; consequently, the QRS complex in patients with Wolff-Parkinson-White syndrome may ''normalize'' (loss of delta wave) and the PR interval may have a normal duration.[13] However, other typical electrocardiographic changes of hyperkalemia (eg, peaked T-waves, and so on) are generally present in these patients.

Of particular importance, especially to emergency physicians, is recognition that hyperkalemia may also produce ECG changes that mimic acute myocardial infarction.[9,14-20] The ECG may show widespread ST-segment elevation; often, a more localized pattern that simulates an inferior infarction is observed.[14] Generally, no Q-wave development occurs; however, on rare occasions, Q-waves have been noted to occur transiently or evolve in the precordial leads.[15,21] ST-segment depression and T-wave inversion typical of myocardial ischemia has also been reported.[15,22] The changes mimicking infarction or ischemia resolve after treatment.

TABLE 1. Electrocardiographic Manifestations of Serum Hyperkalemia Relative to Serum Potassium Level

Serum Potassium Level	Expected ECG Abnormality
Mild hyperkalemia 5.5-6.5 mEq/L	Tall, tent-shaped (''peaked'') T-waves with narrow base, best seen in precordial leads
Moderate hyperkalemia 6.5-8.0 mEq/L	Peaked T-waves Prolonged PR interval Decreased amplitude of P-waves Widening of QRS complex
Severe hyperkalemia >8.0 mEq/L	Absence of P-wave Intraventricular blocks, fascicular blocks, bundle branch blocks, QRS axis shift Progressive widening of the QRS complex resulting in bizarre QRS morphology Eventual ''sine-wave'' pattern (sinoventricular rhythm), VF, asystole

CONCLUSION

A variety of electrocardiographic manifestations of hyperkalemia exist. The classic findings of peaked T-waves, prolongation of the PR-interval, absence of the P-wave, and QRS widening are common and well known to emergency physicians. However, severe hyperkalemia may occur in the presence of minimal or atypical ECG findings. Hyperkalemia may also mimic acute myocardial ischemia or infarction. Given the inherent delays in obtaining serum potassium results from the laboratory, a thorough knowledge of the electrocardiographic manifestations of hyperkalemia is imperative to ensure prompt treatment of this potentially life-threatening condition.

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