ECG Changes in Hyperkalemia

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A succinct review of hyperkalemia . . . its various causes, clinical manifestations and consequences, ECG findings, and treatment approaches.

A 58-year-old man who resides in a nursing home complains of fatigue, general weakness, and malaise. His history includes chronic renal insufficiency, stroke, cardiomyopathy, and hypothyroidism. His baseline creatinine level is 2.3 mg/dL; he has no history of dialysis. Laboratory data obtained in the morning at his nursing home indicated worsening renal failure. His physician sends him to the emergency department (ED) for further evaluation.

In the ambulance, the patient becomes bradycardic and hypotensive and complains of shortness of breath. He is given atropine, which somewhat relieves his symptoms.

On arrival in the ED, the patient is drowsy and slow to respond. His heart rate is 58 beats per minute and regular. His lungs are clear. Results of a neurologic examination are nonfocal. Bilateral lower extremity edema is noted. The remainder of the examination is unremarkable.

An ECG shows an accelerated junctional rhythm with marked widening of the QRS complex and peaked T waves (Figure 1). Initial laboratory results include potassium, 9.5 mEq/L; bicarbonate, 18 mEq/L; creatinine, 8.3 mg/dL; and blood urea nitrogen, 85 mg/dL. In the ED, the patient is given intravenous calcium gluconate, insulin, and glucose. A second ECG ordered 30 minutes later shows a return to sinus rhythm with a first-degree atrioventricular (AV) block, a nonspecific intraventricular block, shortened QRS duration, shortened QT interval, and a decrease in T-wave amplitude (Figure 2).

HYPERKALEMIA: AN OVERVIEW

Potassium irregularities are the most common electrolyte abnormalities in hospitalized patients.\textsuperscript{1-3} Although hypokalemia is more common, hyperkalemia is much more serious. Hyperkalemia occurs almost exclusively in patients with underlying renal disorders. In normal potassium homeostasis, intake varies and renal excretion generally adjusts to match. Ninety percent of potassium is excreted in the kidneys. Any acute or chronic kidney disease or dysfunction interrupts the delicate homeostasis and causes hyperkalemia.\textsuperscript{1,2}

Hyperkalemia is defined as a serum level of potassium greater than 5.5 mEq/L. It can be further broken down as follows:

- **Minimal** (potassium level, 5.5 to 6.5 mEq/L; associated with minor ECG changes).
- **Moderate** (potassium level, 6.6 to 8.0 mEq/L; peaked T waves are generally seen on ECG).
- **Severe** (potassium levels higher than 8 mEq/L or any level with wide QRS complexes, AV block, or ventricular dysrhythmias).\textsuperscript{2-5}

Evidence of hyperkalemia can be clinically apparent in several organ systems. The most serious are cardiac manifestations, but the GI and neuromuscular systems are also affected. Clinical signs and symptoms include malaise, generalized weakness, nausea and vomiting, muscle cramps, paralysis,
paresthesias, and decreased deep tendon reflexes. Cardiac manifestations include dysrhythmias, ventricular fibrillation, and asystole.²

**CAUSES OF HYPERKALEMIA**

These can be divided into 5 categories (Table 1). The most common cause of hyperkalemia seen in the ED is impaired excretion attributable to underlying renal dysfunction that is often the result of long-standing hypertension, diabetes, or cardiac disease.¹

<table>
<thead>
<tr>
<th>Impaired excretion</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Chronic renal failure</td>
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<td>Acute renal failure</td>
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<td>Hypoaldosteronism</td>
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<td>Potassium-sparing diuretics</td>
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<td>Other (advanced age, diabetes, interstitial nephritis, obstructive nephropathy, systemic lupus erythematosus, AIDS, NSAIDs)</td>
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tubular defects in excretion and hypoaldosteronism. The latter condition can include primary hyperaldosteronism (Addison disease) and secondary types (such as renal tubular acidosis, congenital adrenal hyperplasia, and medication-induced hyperaldosteronism). Increased intake. Potassium supplementation, salt substitutes, medications containing potassium, and massive transfusion may cause hyperkalemia.

Cellular shifts. Insulin deficiency, rhabdomyolysis, and acidosis may lead to hyperkalemia by causing a shift of potassium from the intracellular to the extracellular space.

Medications. A number of medications may impair renal function. NSAIDs may have this effect in a patient with known renal insufficiency. NSAIDs impair renin secretion and synthesis of vasodilatory prostaglandins, which results in a decreased glomerular filtration rate and impaired secretion of potassium.

Pseudohyperkalemia (fictitious hyperkalemia) occurs with hemolyzed specimens, muscle clenching distal to the tourniquet during a blood draw, leukocytosis, and thrombocytosis. Hemolysis from phlebotomy is common and should be suspected in asymptomatic patients with no other underlying cause.

**ECG FINDINGS IN HYPERKALEMIA**

Potassium has a significant effect on the myocardium and is a key determinant of the resting membrane potential of cells. The ECG is an important diagnostic tool in the early diagnosis of hyperkalemia as well as in the initiation of empiric treatment (Table 2). Peaked T waves, especially in the precordial leads, reflect acceleration of the terminal phase of ventricular repolarization and typically are the initial ECG change seen; they often suggest mild to moderate hyperkalemia. Moderately elevated potassium levels are associated with flattening of P waves, prolongation of the PR interval, and nodal rhythms. This represents a depression in conduction and change in sensitivity of atrial myocytes. As the severity of hyperkalemia increases, P waves disappear, the QRS complex assumes a bizarre shape, sine waves are seen, and eventually ventricular fibrillation or asystole occurs. Despite the correlation of certain serum levels of potassium with ECG changes, such changes vary

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<th>Severity</th>
<th>Findings</th>
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<td>Mild to moderate</td>
<td>Peaked, &quot;tented&quot; T waves, Flattening of P waves</td>
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<tr>
<td>Moderate*</td>
<td>Loss of P waves, Increased PR interval, Uniform widening of QRS complex, Nodal rhythm</td>
</tr>
<tr>
<td>Severe</td>
<td>Bizarre widening of QRS complex, Sine wave, Ventricular fibrillation, Asystole</td>
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*There is no clear correlation between levels of potassium and the likelihood of an arrhythmia; however, arrhythmia is more likely if the increase in potassium is rapid.
among patients and may also be affected by the acuity of the increase in serum levels.\textsuperscript{5} Other, less typical ECG changes are sometimes seen with hyperkalemia; these include complete heart block, QRS axis shift, and bundle-branch block.\textsuperscript{5,6}

**TREATMENT**

The urgency of treatment is determined by the patient's symptoms, underlying disorders, kidney function, hemodynamic status, and ECG findings. Important initial management considerations include identification of the cause and assessment of the rise in potassium levels.\textsuperscript{2,3}

A 4-pronged approach for patients with moderate to severe hyperkalemia is recommended:  
1. The initial step in the management of patients with ECG changes is administration of calcium gluconate to reduce membrane excitability. The duration of action is short (20 to 60 minutes), and repeated doses may be necessary.  
2. Short-term treatment with insulin and glucose moves the potassium from the extracellular to the intracellular space.  
3. Treatment with \(-\)agonists, such as albuterol aerosols, may also lower serum potassium levels for 2 hours or more by moving potassium to the intracellular space.  
4. Administration of an exchange resin, such as sodium polystyrene sulfonate, is effective for long-term reduction of serum potassium. Given orally or rectally, sodium polystyrene sulfonate removes 1 mEq of potassium per gram given; it begins to act in 1 to 2 hours.

Definitive treatment for hyperkalemia and renal failure consists of regular dialysis, either for a few days (if a specific cause can be identified and treated) or for the rest of the patient's life.

**OUTCOME OF THIS CASE**

The patient was admitted to the medical ICU, where he underwent hemodialysis in conjunction with administration of insulin and sodium polystyrene sulfonate. He was found to be hypothyroid and his dosage of thyroxine was adjusted. Within 24 hours, his potassium level decreased to 4.7 mEq/L and his creatinine level decreased to 4.5 mg/dL. Acute myocardial infarction was ruled out after normal results were seen on 3 tests of cardiac enzyme levels and serial ECGs. After discharge to a long-term-care facility, he was lost to follow-up.

No specific cause was found for this patient's acute renal failure, which was believed to be a result of deterioration from his chronic illnesses.

**References:**

REFERENCES:  

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