A 34-year-old man presents to the emergency department with progressive, generalized weakness and shortness of breath that began 2 weeks earlier. He has no history of cardiac disorders, and he denies chest pain, palpitations, and abdominal pain. He admits to recent methamphetamine use.

The patient is in moderate distress. Heart rate is 152 beats per minute and regular; respiration rate, 24 breaths per minute; blood pressure, 142/68 mm Hg. Pulse oximetry shows that oxygen saturation is 95% on room air. Lungs are clear. A 2/6 systolic murmur is audible. Abdomen is soft and nontender. Neurologic examination demonstrates mild diffuse symmetric weakness.

An ECG is obtained (A). Initial treatment includes oxygen, intravenous fluids, and lidocaine; the patient's condition does not change. After arterial blood gas measurement reveals significant metabolic acidosis (pH, 7.08; \(\text{PO}_2\), 217 mm Hg; \(\text{PCO}_2\), 22 mm Hg), sodium bicarbonate is administered. Fifteen minutes later, a second ECG is obtained (B).

Which of the following diagnoses is best supported by the ECGs and clinical findings?
A. Acute myocardial infarction.
B. Hyperkalemia.
C. Ventricular tachycardia.
D. Preexcitation syndrome.

**WHAT THE ECGs SHOW**

The first ECG reveals a regular wide-complex tachycardia of unclear origin (Figure 1). The differential diagnosis for this finding includes supraventricular tachycardia with aberrancy or bundle-branch block, pacemaker-mediated tachycardia, sodium channel blocker toxicity, hyperkalemia, preexcitation, and ventricular tachycardia. The potential causes of ventricular tachycardia are numerous; among them are primary cardiac disease; ischemia; and other primary noncardiac disorders, such as toxicologic insults and electrolyte abnormalities.

The second ECG (Figure 2), obtained after administration of bicarbonate, reveals slowing of the heart rate, narrowing of the QRS complex, and the presence of "tented," or peaked, T waves, primarily in the precordial leads. The findings on these 2 ECGs suggest hyperkalemia, B.

The patient's ECG response to bicarbonate makes primary cardiac disease and ischemia less likely. Although this response to bicarbonate might also be associated with a toxicologic insult (such as can cause ventricular tachycardia), the finding of peaked T waves points strongly to an elevated serum potassium level. **PATHOGENESIS OF HYPERKALEMIA**

Potassium, the predominant intracellular cation, plays an important role in maintenance of the potential across the cellular membrane, as well as in depolarization and repolarization of myocytes and neurons. As a result, changes in serum potassium level can have dramatic effects on cardiac cell conduction and, consequently, on the ECG.

The causes of hyperkalemia include acute and chronic renal failure, diabetic ketoacidosis, mineralocorticoid deficiency, type IV renal tubular acidosis, medications (angiotensin-converting enzyme inhibitors, potassium-sparing diuretics, lithium, NSAIDs, and b-adrenergic antagonists), acute digoxin toxicity, rhabdomyolysis, burns, crush injuries, and severe dehydration-- as well as any combination of the above. Methamphetamine use can cause renal failure. Spuriously elevated potassium levels can occur with hemolysis during phlebotomy. Pseudohyperkalemia results when potassium is released from platelets in the setting of thrombocytosis. **TYPICAL ECG FINDINGS**

The earliest ECG signs of hyperkalemia are "tented" T waves, which are classically described as tall, symmetrically narrow, and peaked (see Figure 2). However, in most patients, the ECG shows large-amplitude T waves rather than the classic tented T waves. In addition, the direction of the T wave may change. In particular, the inverted lateral T waves associated with left ventricular hypertrophy may pseudonormalize. These T-wave changes result from acceleration of the terminal repolarization of the myocytes and are often most pronounced in the precordial leads.
As potassium levels rise further, cardiac conduction between myocytes is suppressed. Atrial tissue is more sensitive to this effect than ventricular tissue. As a result, P-wave amplitude decreases and P waves flatten and may disappear altogether. The PR and QRS intervals may lengthen. Further elevations in potassium levels can lead to sinoatrial and atrioventricular conduction blocks with resulting escape beats and rhythms. Intraventricular conduction delays, including bundle-branch blocks and fascicular blocks, have been reported. Bypass tracts may be more sensitive to these effects; "normalization" and loss of the delta wave have been reported in patients with Wolff-Parkinson-White syndrome in whom hyperkalemia developed. In severe hyperkalemia, conduction delays affect all portions of the QRS complex (not just the terminal portion); this results in further widening and slurring of the QRS complex. This complex may ultimately blend into the T wave, which gives the ECG tracing a sine-wave appearance (see Figure 1). Without treatment, the rhythm can deteriorate into ventricular fibrillation or asystole. The ECG findings commonly associated with mild, moderate, and severe hyperkalemia are shown in the Table. However, keep in mind that the findings can vary significantly from patient to patient; some patients with severe hyperkalemia may have only minimal ECG changes. TREATMENT Early treatment of hyperkalemia can be lifesaving. Prompt diagnosis often depends on the recognition of suggestive ECG findings. The goals of treatment are as follows:

- To block the effects of elevated serum potassium levels on cellular membranes, particularly those in cardiac tissue.
- To increase the flow of potassium from extracellular to intracellular spaces.
- To remove excess potassium from the body.

Give patients with unstable dysrhythmias calcium chloride or calcium gluconate. (The former has 3 times more elemental calcium but can be sclerosing to peripheral veins.) Calcium cations block the effect of potassium in cardiac tissues and stabilize myocyte membranes immediately. However, the effect lasts only 20 to 40 minutes; thus, repeated dosing may be required. Some authorities recommend withholding calcium if concomitant digoxin toxicity is suspected, to avoid inducing dysrhythmias associated with digoxin toxicity. Recent animal research has raised questions about the necessity of this precaution, but conclusive data have yet to be published.

Treatment with sodium bicarbonate, glucose and insulin, or a β2-adrenergic agonist drives potassium ions into the cells and reduces extracellular serum levels. Alkalosis induced by bicarbonate administration increases the exchange of extracellular potassium ions for intracellular hydrogen ions; the duration of action of bicarbonate is approximately 2 hours. Sodium bicarbonate has the most profound effect in patients who are acidic. Insulin (with glucose to prevent iatrogenic hypoglycemia) drives potassium into the cells and has a duration of action of 4 to 6 hours. Treatment with nebulized β2-agonists can similarly drive potassium into the intracellular spaces, an effect that also lasts approximately 2 hours. Because the effects of all these treatments are transitory, repeated administration may be necessary in severe cases, pending definitive therapy.

Removal of excess potassium from body stores is effected primarily through the use of exchange resins, such as sodium polystyrene sulfonate, or hemodialysis. Hemodialysis is recommended for all patients with hyperkalemia in whom the ECG shows abnormal QRS complexes. OUTCOME OF THIS CASE

Hyperkalemia was confirmed by laboratory studies that revealed a potassium level of 8.1 mEq/L. Methamphetamine-induced acute renal failure was diagnosed, and the patient was treated with calcium chloride, additional bicarbonate, and insulin/glucose therapy. He was hospitalized and underwent hemodialysis. A repeated ECG, obtained the day after admission, revealed a return to sinus rhythm, with a normal QRS interval and resolution of the peaked T waves (Figure 3). After 5 days, the patient's renal function returned to normal and he recovered uneventfully.

References:


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