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CASE REPORTS

# FIELD PRESENTATION OF HYPERKALEMIA AND CARDIAC ARREST

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## ABSTRACT

Unresponsive patients with no family or friends can be particularly challenging for emergency medical services (EMS) providers, especially if they rapidly deteriorate or arrest on scene. Although the "Hs & Ts" of cardiac arrest, including hyperkalemia, are taught in Advanced Cardiac Life Support (ACLS), they are an infrequent and easily overlooked cause of cardiac arrest. Hyperkalemia may not even be considered, especially in patients not on dialysis. However, recognition is crucial as specific medications are needed, and the standard approach to cardiac arrest will not work and may, in fact, be harmful. Fortunately, there are specific and readily identifiable electrocardiogram (ECG) changes preceding the arrest. We present a case illustrating these changes and highlight several visual and physical clues on patient assessment, all of which easily could be overlooked in a hurried evaluation.

## CASE REPORT

Police were called to do a welfare check on an 83-year-old male who had not been seen for a week. He was unresponsive, and the fire department and EMS were called to assist. The firefighters removed him from the house before EMS arrived. EMS providers found a disheveled and chronically ill-appearing elderly patient lying on the grass. He was unresponsive, lived alone, and there was no additional medical history available. Unresponsive to all stimuli, he was taking shallow breaths and had weak central and peripheral pulses. EMS providers immediately moved him to the ambulance. There was a considerable amount of debris and emesis suctioned from his airway, and bag-valve-mask (BVM) ventilations were started. His clothing was removed revealing a Foley urinary catheter with minimal urine in the bag and leakage around the catheter at the tip of the penis. A large, firm "mass" was noted in the suprapubic region. Initial vital signs included a pulse of 74, blood pressure of 148/100 mmHg, respirations of 24, and an undetectable pulse oximetry reading. A 12-lead ECG showed a sinus rhythm with a first-degree atrial-ventricular (AV) block and a wide QRS complex made up of a

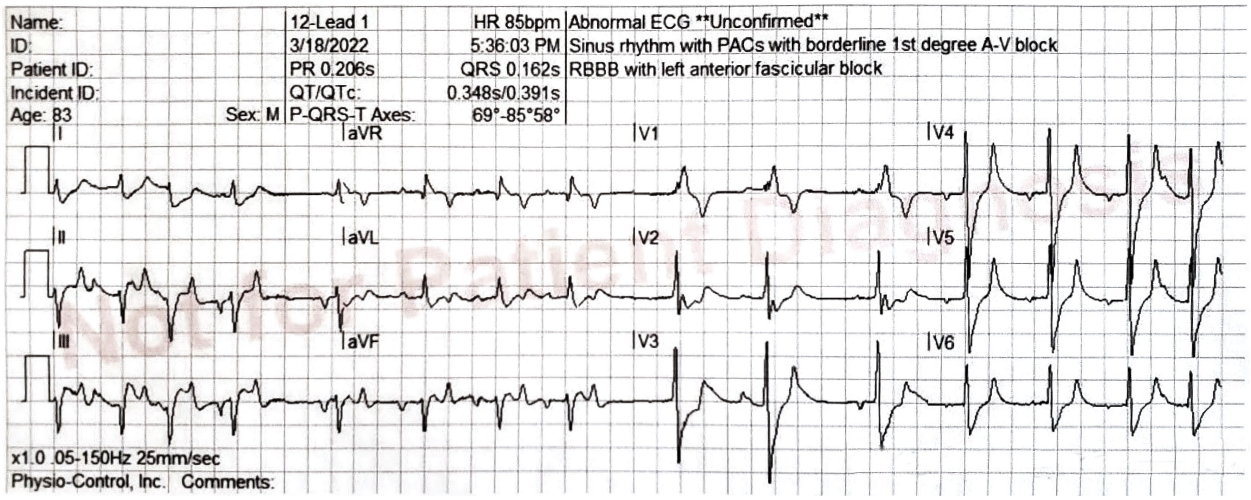


*Figure 1.* 12 Lead ECG showing sinus rhythm with 1st degree AV block, RBBB and LAFB with peaked T waves.

right bundle branch block (RBBB) and a left anterior fascicular block (LAFB) with peaked T waves (Figure 1).

He was intubated without sedation or paralysis, and an intraosseous line was placed. At this point, he became markedly hypotensive (74/30). Fluids were started, and push-dose epinephrine of 20 micrograms (mcg) was quickly administered. Despite this, the patient lost pulses. Two minutes of cardiopulmonary resuscitation (CPR) with chest compressions was performed resulting in return of spontaneous circulation (ROSC). He remained hypotensive and was given an additional push dose of 20 mcg of epinephrine and a fluid bolus.

Meanwhile, the QRS complexes on the monitor had started to widen even more with a loss of the P wave (Figure 2). Given the absence of urine in the Foley bag, leakage around the catheter, and the suprapubic mass, post-obstructive renal failure with resultant hyperkalemia was suspected. One gram of calcium chloride was administered with some narrowing of the QRS on the monitor observed with a heart rate of 110 (Figure 3). Despite this, the patient rearrested. Compressions resumed, and 50 milliequivalents (mEq) of sodium bicarbonate was administered. He received a total of three shocks for ventricular tachycardia (VT) before arriving at the emergency department (ED) where ROSC was again obtained. In the ED he was found to have a potassium of 8.8 mEq/L and a creatinine >12 mg/dL confirming the suspicions of the prehospital providers. He was given 10 units of insulin and 50 g of dextrose with an emergent consult to nephrology for dialysis. A new Foley urinary catheter was also placed and drained 2.5L of urine. He was transferred to the intensive care unit (ICU) where he underwent emergent dialysis. Unfortunately, his neurological outcome was poor, and he was made "comfort measures" before ultimately dying.

## DISCUSSION

Potassium is taken into the body and stored (98%) in cells from where it is released into the blood. Most potassium is ingested in the form of potassium-rich foods like spinach, in salt substitutes, or supplements. Outside of an intentional overdose, this is unlikely to cause significant hyperkalemia. Ninety percent of potassium is eliminated in the urine

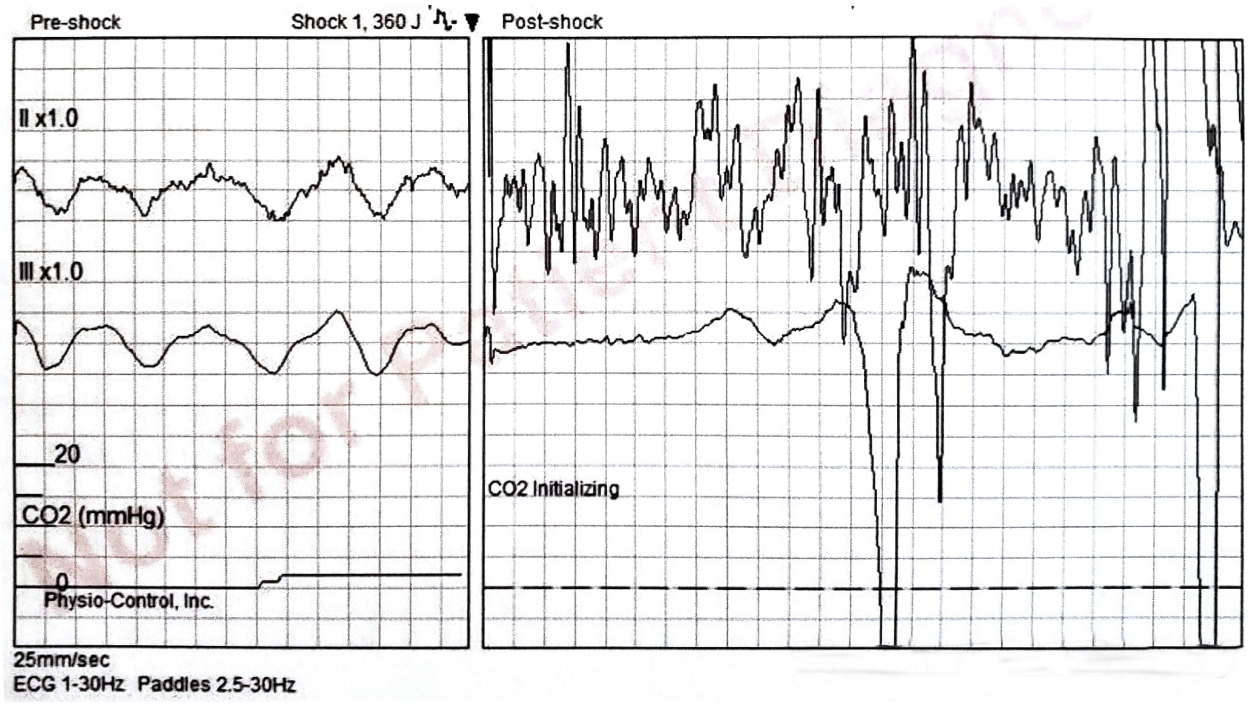


*Figure 2*. Rhythm showing sine wave in pre-shock section and prior to calcium administration.

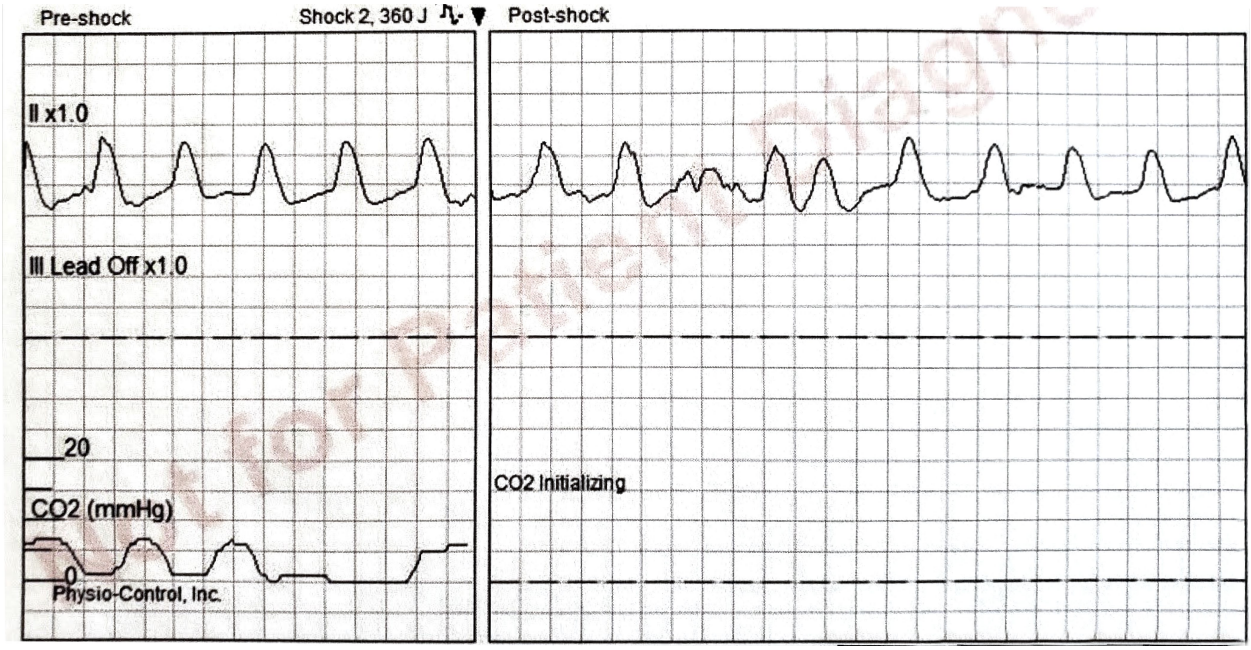


*Figure 3*: Post calcium administration showing narrowing of QRS complex as compared to figure 2.

with the kidneys playing a crucial role in its regulation. The remaining 10% is eliminated or lost in the gastrointestinal (GI) tract which is why severe diarrhea can lead to hypokalemia. Given the role they play in potassium regulation, it is not surprising that problems arise when the kidneys are not working correctly. This could be from damage to the kidney itself, from compromised flow in (blood) or out (urine) of the kidneys.

Our patient was elderly, apparently not well cared for, and likely had some amount of underlying, baseline, kidney dysfunction or chronic kidney disease (CKD) to begin with.

He appeared dehydrated which would have reduced flow to the kidneys, decreasing the elimination of potassium. It is also possible that he had been lying and not moving very much for some time. If he had been on a hard surface, it would have caused tissue and muscle breakdown with the release of myoglobin and creatine kinase, both of which are toxic to the kidneys. The "crushed" tissue also releases potassium from the damaged cells, increasing the potassium load on the damaged kidneys. Finally, the potassium, along with other waste products, needs to be eliminated in the urine. The clogged Foley catheter prevented this from taking place.

It is the amount of potassium that is circulating in the blood that causes the ill-effects such as muscle weakness and cardiac arrest. Hyperkalemia acts by prolonging phase 2 of the cardiac action potential keeping slow calcium channels open longer. This alters the electrical depolarization of the heart muscle and is reflected in the ECG. There is a progression of defined changes seen with rising serum levels: sharp, narrow-based, peaked T waves are generally seen with a potassium of 5.5-6.5 mEq/L, loss of P waves at levels of 6.5-7.5 mEq/L, wide QRS durations at 7.0-8.0 mEq/L, and a sine wave with levels between 8.0-10 mEq/L. From here, it progresses to asystole (Mattu et al., 2000). The widened QRS complex reflects the altered electrical activity in the heart and coincides with impaired pump function and a decrease in cardiac output. This, in turn, leads to a drop in measured blood pressure and a change in the patient's level of consciousness from the reduced blood flow to the brain. Note, however, that there is individual variability with people who have chronic kidney disease, generally running and tolerating higher potassium levels without any ECG changes.

Calcium should be administered immediately upon suspicion of hyperkalemia, especially as the QRS complexes begin to widen. Calcium chloride and calcium gluconate work to antagonize the effects of hyperkalemia and stabilize the myocardium (Parham et al., 2006). Calcium chloride contains three times the elemental calcium. However, calcium gluconate causes less irritation at the IV site and is preferred (Long et al., 2018). The onset of action is rapid but short-lived with membrane stabilization lasting only about 30-60 minutes and doing nothing to affect the amount of circulating potassium. The dosing of calcium chloride ranges from 1-2 g, and repeat dosing should be considered every 5-10 minutes during cardiac arrest due to suspected hyperkalemia (Mushinyakh et al., 2012). This recognition is crucial because standard ACLS treatment with epinephrine and amiodarone will not be effective. However, routine administration of calcium in pre-hospital cardiac arrest is also not recommended and has been called into question by a recent Danish study, stopped early due to concerns of harm (Vallentin et al., 2021).

While sodium bicarbonate is not routinely used in the treatment of cardiac arrest, in this case, it is crucial, as it is a useful aid in the treatment of hyperkalemia. It has weak actions on potassium by increasing the serum pH. Cells will then shift hydrogen ions into the serum to normalize the pH, and, in turn, move some potassium into the cells to maintain the electrical balance (Burnell et al., 1956). Insulin can also shift potassium and can be used to treat hyperkalemia. Insulin acts on the Na-K-ATPase pumps of skeletal muscles shifting potassium out of the serum and into cells, thus reducing circulating potassium in the blood. Administration of 10 units of insulin can lower serum potassium by 1 mEq/L (Harel & Kamel, 2016). Dextrose should be administered with insulin for patients whose blood glucose levels are below 250 mg/dL. This will allow for more potassium to be pushed into the cells. Other adjuncts, such as continuous albuterol or

loop diuretics such as furosemide, can also be considered to reduce circulating potassium. Finally, flushing the body with IV fluids and increasing the clearance of potassium through the urine is crucial, especially when combined with a diuretic. However, this requires a free flow of urine, and any obstruction to the flow of urine, such as a blocked, Foley urinary catheter can interfere. If severe, this will lead to renal failure and a rise in serum potassium. Other causes of post-renal (obstructive) failure can also be seen with prostate or bladder issues such as benign prostatic hyperplasia (enlarged prostate), prostatitis, prostate or bladder cancer, or with a neurogenic (non-contracting) bladder, as well as urethritis or even pregnancy, all of which interfere with the outflow of urine (Klahr, 1983). Clearing the obstruction normally corrects the renal failure. In our patient, replacement of the malfunctioning Foley catheter would empty his bladder. However, this could not be done until arrival in the ED.

Another notable feature of this patient's initial 12 lead ECG is the trifascicular block due to his hyperkalemia. The classic definition of the trifascicular block involves a right bundle branch block (RBBB), an AV block and either a left anterior fascicular block (LAFB) or a left posterior fascicular block (LPFB). To be considered a true trifascicular block the AV block needs to be high-grade. A first-degree block is generally in the AV node itself and does not involve the His-Purkinje system (Kusumoto et al., 2019). A patient who presents with syncope, chest pain, and lightheadedness should be taken to a center capable of providing advanced cardiac care. Patients who present with a trifascicular block (3rd degree AV block, RBBB, and LAFB or LPFB) will require a pacemaker as the risk of deteriorating to a lethal arrhythmia is high (Brignole et al., 2013). Patients who present with syncope with a bifascicular block (RBBB and LAFB or LPFB) who have had other causes of syncope evaluated should be observed on telemetry for blocks over a 24-48-hour period (Brignole et al., 2018). These patients may require pacemaker placement and an electrophysiology consult.

## CONCLUSION

This case illustrates the importance of critical thinking and synthesizing all available evidence while caring for a patient. This was not a dialysis patient and the cause of his arrest might easily have been missed. Undressing this patient was crucial to discovering the nondraining, Foley catheter and the distended bladder which suggested urinary retention and potential renal failure. His overall appearance suggested poor self-care, possible medication noncompliance, and likely dehydration further exacerbating any preexisting chronic renal issues. In addition, it was unclear how long he may have been lying in his home with the potential for further kidney injury from rhabdomyolysis and release of creatine kinase and potassium from tissue breakdown. Taken together these are highly suggestive of hyperkalemia. It also illustrates the importance of early monitoring and ECG acquisition allowing for the recognition of the signs of hyperkalemia that herald impending deterioration and cardiac arrest. When the ECG findings were combined with the physical assessment, this identified his problem, allowing for the correct treatment using calcium and sodium bicarbonate along with intravenous fluids. This recognition was crucial because standard ACLS treatment with epinephrine and amiodarone would have been ineffective. Rapid administration of calcium stabilizes the cardiac membrane by reversing some of the immediate effects of the hyperkalemia. Sodium bicarbonate, insulin and dextrose, as well as albuterol aerosols, shift the potassium

from the circulation into the cells, while IV fluids and diuretics help flush some of the potassium out through the kidneys. All, however, require the problem to be recognized.

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