



CASE REPORT

AGGRESSIVE HYPERKALEMIA TREATMENT IN THE PREHOSPITAL SETTING: A CASE REPORT

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ABSTRACT

This is a case of profoundly unstable hyperkalemia encountered in the American prehospital 911 setting, with subsequent aggressive treatment and stabilization. Although hyperkalemia may be present in the prehospital setting, it is difficult to diagnose without laboratory testing or a reliable history of illness. It is frequently encountered in the prehospital setting, especially in communities with high levels of comorbidities and multiple dialysis centers. However, in the spectrum of hyperkalemia presentations, many of these patients are either early in their disease presentation or found when already in cardiac arrest. Early identification and rapid, aggressive treatment are paramount to decreasing morbidity and mortality in these patient populations. This case shows how aggressive prehospital critical care can have a significant impact on patient outcomes.

BACKGROUND

This case was encountered by a suburban fire department-based Emergency Medical Service (EMS) serving a population of approximately 50,000 residents in the United States with a call volume of 3,000-4,000 calls per year. The response area is home to a suburban community hospital without dialysis capabilities and is located near a metro area with a Level I tertiary care center approximately 25 minutes away. This agency provides advanced life support level service, with paramedics being present on all ambulances, as well as some of the fire suppression apparatus. Daily staffing includes three ambulances, three engine companies, one truck company, one Battalion Chief, and one Paramedic Shift Supervisor. New Paramedics go through a field-training process with a Paramedic preceptor, with periodic reviews and ultimate clearing by the department's Physician Medical Directors. Expansive "Patient Care Guidelines" are in place, with Paramedics in this system given the latitude to deviate from these guidelines if the patient's condition warrants.

CASE REPORT

EMS was dispatched to a skilled nursing facility for an elderly male patient for difficulty breathing. The response to this call included an ambulance, engine company, and Paramedic Shift Supervisor. All three paramedics responding to the call had at least three or more years of experience providing advanced life support care. Nursing staff on location noted the patient to have a sudden change in responsiveness, being unable to arouse him during normally scheduled rounds. His reported baseline mental status was alert, oriented, and able to carry on normal conversations. Nursing staff also noted an increased difficulty breathing, but no additional information on chronic medical history was made available to responding personnel.

Upon physical exam, the patient was responsive to verbal stimuli but had confused conversation and difficulty speaking. Lung sounds were diminished, crackles were noted in the left and right lung bases, and no unilateral neurological deficits were observed. The skin was pale, cool, and dry, with no obvious signs of trauma. The patient was noted to be slightly hypotensive with a blood pressure of 88/50 and notably hypoxic on room air with an oxygen saturation of 81% on initial contact. To immediately address this initial presentation, the patient was placed on supplemental oxygen at 15 liters per minute by a non-rebreather mask. This intervention resolved the hypoxia and slightly improved his mental status. Capillary blood glucose was 166 mg/dL, and the patient was afebrile. The nursing staff noted the patient had labs drawn the previous evening and was found to have a newly elevated serum potassium level of 6.9 mEq/L. The lab report also revealed hypocalcemia.

With the finding of the lab report, combined with the patient's critical condition, aggressive management was started. Paramedics on location decided to stabilize the patient prior to transport, as it appeared to be the safer option for the patient due to the patient's vital signs and altered mental status. The cardiac monitor revealed wide complex tachycardia, with a 12-lead ECG showing a wide QRS complex concerning for hyperkalemic instability. Initial treatment included 1 gram of Calcium Gluconate IV and 100 mEq Sodium Bicarbonate IV. The patient became more somnolent during this time, with a decreased respiratory drive. Due to the progressively deteriorating respiratory status combined with a projected poor clinical course, the decision was made to intubate the patient.

The patient was pre-oxygenated with oxygen at 15 liters per minute via non-rebreather mask, combined with passive oxygenation at 15 liters per minute via nasal cannula. Pre-induction capnography was noted to be 17 mmHg with normal plateau waveform. A push dose of 30 mcg of epinephrine IV was given prior to induction to optimize blood pressure to greater than 100 systolic blood pressure. Following SpO₂ optimization to greater than 94%, 30 mg of etomidate IV and 160 mg of rocuronium IV were administered for induction and paralysis, respectively. The patient was intubated with video laryngoscopy on the first attempt. Following confirmation of tube placement, three combined doses of 2.5 mg Albuterol sulfate were administered in-line via nebulizer simultaneously, which ran continuously throughout transport. After these three doses were completed, three DuoNeb (ipratropium bromide and albuterol sulfate) treatments were started simultaneously, which ran continuously for the remainder of transport. DuoNeb was chosen as the crew had expended its supply of albuterol.

Following intubation, the patient was hypotensive again. An additional 30 mcg of Epinephrine IV was given. Norepinephrine infusion was also started for sustained blood pressure control, which adequately stabilized blood pressure. Paramedics on scene decided to limit crystalloid fluid administration to limit fluid overload in the presence of presumed new acute renal failure in the setting of hyperkalemia and lung physical exam findings consistent with volume overload.

Following the stabilization of the patient, emergent transport was started to a tertiary care facility capable of providing emergent dialysis. During transport, the crew was directed to hyperventilate the patient to maintain capnography near 17 mmHg to limit worsening metabolic acidosis. The patient also received an additional 1 gram of Calcium Gluconate IV and 50 mEq Sodium Bicarbonate IV during transport. Pt was sedated with Ketamine and Fentanyl was given for analgesia during transport.

The emergency department was given advanced notice to have staff and resources available to continue treatment and potentially arrange emergent dialysis. Ultimately, the patient remained critical throughout transport, and care was turned over to emergency department staff with the patient in critical condition.

DISCUSSION

This case is an excellent representation of a departure from the mantra of “load and go,” which has been present within EMS for generations. Many times, especially in trauma, it is appropriate to initiate early and rapid transport to definitive care; sometimes, it may be necessary to depart from this mantra. Remaining on a scene through initiating and continued patient care for resuscitations has shown improved outcomes, especially in out-of-hospital cardiac arrests (Grunau et al., 2020). The focus is placed on performing essential interventions and procedures that lead to decreased morbidity and less so on packaging a patient for transport, which may impair or delay best practices, including high-quality CPR. In this case, the crews were concerned about lethal arrhythmia, respiratory failure, and hypotension if stabilization did not occur before transport.

Hyperkalemia is well-supported in literature with cardiac sequelae. Kovesdy (2016) described a heightened risk of mortality associated with hyperkalemia in all patient populations, including those with normal kidney function. Early rises in potassium concentration are attributed to lowering the cardiac membrane potential, often resulting in the classic ‘peaked’ t-waves. Larger rises in potassium concentration have been demonstrated to result in a lengthening of the PR interval and widening of the QRS complex. This often occurs prior to the patient progressing into a ‘sine wave,’ often associated with cardiac arrest following shortly thereafter (Montford & Linas, 2017). The patient’s initial ECG showed a wide QRS complex tachycardia, commonly associated with severe hyperkalemia. As such, the decision was made to treat this patient aggressively on scene to ensure safer and rapid transport. Prehospital rhythm strips were not available from this patient; however, Figure 1 shows the patient’s ECG that was obtained in the emergency department following interventions by EMS. The ECG shows a wide complex tachycardia with nonspecific ST segment changes. These ECG findings show a narrower QRS complex than the ECG initially acquired by EMS. The QRS segment reportedly narrowed with additional hyperkalemia treatments in the emergency department.

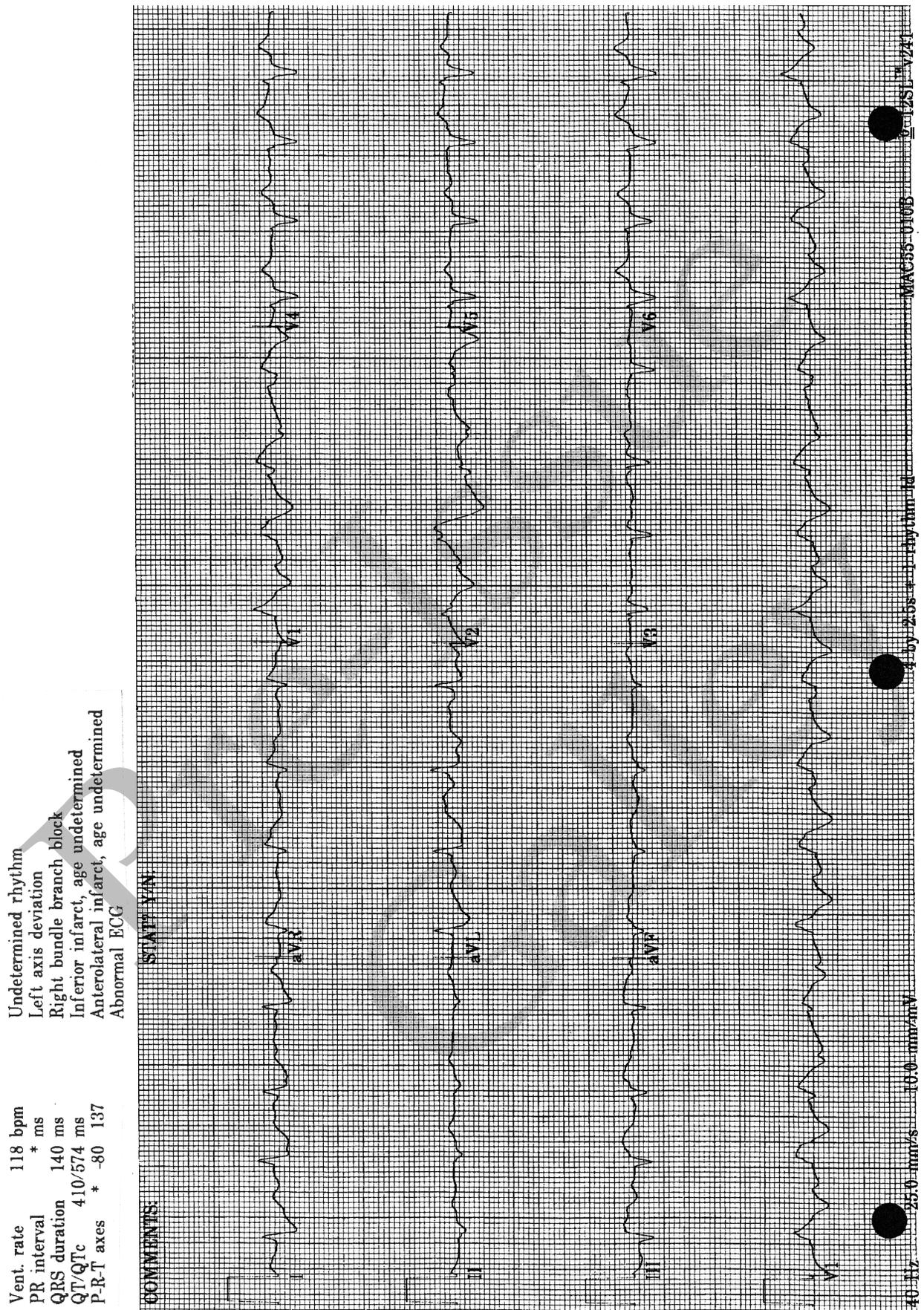


Figure 1. ECG obtained in the emergency department following interventions by EMS

In determining the ventilatory rate, this decision was made based on knowledge of the patient's physiologic status. As a homeostatic mechanism, brainstem chemoreceptors will affect large changes in minute volume based on small changes to pH and PaCO₂ in the blood and cerebro-spinal fluid (Jung et al., 2019). Given this was a presumed metabolic acidosis, steps were taken to prevent an accumulation of carbon dioxide, further worsening the patient's acidosis. The pH will decrease by 0.08 for every 10 mmHg increase in PaCO₂ (Berend et al., 2014). While the crew, in this case, did not have the benefit of arterial blood gas values to guide decision-making, end-tidal capnography was utilized to guide the ventilatory rate. The decision was made to titrate the ventilatory rate to maintain the pre-intubation end-tidal capnography reading to continue respiratory compensation for the patient's metabolic acidosis. The crew did not have access to a ventilator, so a provider was tasked with titrating manual ventilations to maintain this end-tidal capnography level. This provider was tasked with providing ventilation up to 30 cycles per minute.

Hyperkalemic cardiac arrest is also associated with poor neurologic outcomes. Choi, et.al. (2020) explored trending serum potassium levels in cardiac arrest in relation to neurologic outcomes. The study found 1.6% of patients in the hyperkalemia group were found to have a positive neurologic outcome (Choi et al., 2020). By aggressively treating this condition early in the field, we can attempt to prevent poor long-term neurologic outcomes. Hyperkalemic emergencies are treated with intravenous calcium infusions to decrease cardiac excitability. Insulin is the most common agent to assist in moving potassium into the intracellular space, albeit not commonly found in EMS systems. Beta-agonists, such as Albuterol, and Sodium Bicarbonate are commonly utilized in the management of hyperkalemia to assist in treating this acidosis. Hemodialysis, although invasive, is the ultimate tool for rapid elimination of potassium in these patients. Montford, et.al. (2017) defines 'hyperkalemic emergency' as a serum potassium greater than 6 mEq/L. They further suggest these patients be treated with agents capable of rapidly shifting potassium into the cells and using emergent dialysis to decrease the serum potassium level and the long-term complications of this condition (Montford & Linas, 2017). Paramedics on scene for this case elected to transport this patient to a facility capable of providing emergent dialysis, which was located 25 minutes away at the tertiary care center.

CONCLUSION

After the patient was transported to the emergency department, past medical history was obtained, including diabetes mellitus, coronary artery disease, chronic obstructive pulmonary disease, and recently diagnosed mild chronic kidney disease. Upon arrival, a lab test confirmed hyperkalemia with a level of 7.0 mEq/L and the patient received additional doses of calcium gluconate, sodium bicarbonate, and was given intravenous insulin and a dextrose infusion, as well as an isotonic crystalloid bolus. The patient's ECG reportedly narrowed following interventions with potassium lowering to 5.8 mEq/L but remained hypotensive despite vasoactive agents and broad-spectrum antibiotics for undifferentiated shock. The patient was continued on multiple vasoactive infusions and was found to have worsening kidney function based on laboratory findings. He was admitted to the intensive care unit, where his status did not improve. During hospitalization, next-of-kin opted for comfort measures, and the patient was shortly after declared deceased with no found cause of hypotension.

This is a case of aggressive treatment of profound hyperkalemia in the 9-1-1 prehospital setting. This case illustrates how aggressive and critical treatment and transport in the 9-1-1 setting could potentially have a profound impact on reducing morbidity and mortality in this patient population. Prehospital treatments led to temporary stabilization and improvement of the patient's ECG while they were in a critical state. Without instant laboratory testing, paramedics determined, based on previous labs, ECG findings, and the patient's vital signs, that hyperkalemia was a treatable diagnosis that was contributing to the patient's ill status. Although unknown to what extent, there were likely multiple processes contributing to the patient's sudden decline, including worsening kidney function and hyperkalemia. This case also shows the benefit of training paramedics to act as practitioners and make sound clinical decisions in the field. Paramedics, in this case, relied on clinical judgment and expanded from defined patient care guidelines, when necessary, in order to aggressively treat this patient and limit the cardiac effects of the patient's hyperkalemic state.

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