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# Compartment Syndrome and Rhabdomyolysis Presenting with the Rare Pseudo-Infarction Pattern of Hyperkalemia

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**Abstract** The rapid identification and treatment of patients with ST-segment elevation myocardial infarction (STEMI) is a priority for emergency department providers. Occasionally conditions other than acute coronary syndrome (ACS) can mimic the presence of ST-elevation on electrocardiogram (ECG), making the accurate identification of these patients challenging. We present a case in which severe metabolic derangements resulted in a rare pseudo-STEMI pattern on ECG. A 26 year old male was found at home by family after a reported fall. A pre-hospital ECG conducted by EMS personnel indicated STEMI and the cardiac catheterization team was activated prior to patient arrival. Before undergoing PCI, laboratory values revealed severe hyperkalemia and rhabdomyolysis. Treatment of the patient's hyperkalemia resulted in resolution of ECG abnormalities. The patient later developed acute compartment syndrome and underwent emergent fasciotomy. This case demonstrates the rare pseudo-infarction pattern of hyperkalemia and underscores the importance of clinical context in the initial evaluation of the emergency patient.

***Keywords:*** *hyperkalemia, Psuedo-Infarction, Emergency Medicine, compartment syndrome, rhabdomyolysis, myocardial infarction*

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## 1. Background

The rapid identification and treatment of patients with ST-segment elevation myocardial infarction (STEMI) is a priority for emergency department providers. Occasionally conditions other than acute coronary syndrome can mimic the presence of ST-elevation on electrocardiogram (ECG), making the accurate identification of these patients challenging. We present a case in which severe metabolic derangements resulted in a characteristic STEMI pattern on ECG.

## 2. Case Report

A 26-year-old male with a history of intravenous drug use (IVDU) presented to the emergency department via Emergency Medical Services (EMS) in full spinal immobilization after being "found down" at home by a family member. As per the EMS report, the patient had fallen in the bathroom and lost consciousness. Upon waking, he felt short of breath with occasional chest pain. Further discussion with the patient in the absence of family members revealed that he had relapsed with intravenous drug use the night prior, injecting two bags of heroin and subsequently "passing out" on the bathroom floor. The patient estimated that he was unconscious for at least six hours. Pre-hospital providers performed a 12-lead ECG, which showed concern for acute septal STEMI. A pre-hospital STEMI alert was called and paramedics administered 324mg of aspirin.

On arrival to the emergency department, the patient was alert, awake, and oriented. He complained of chest pain and shortness of breath as well as pain and weakness in his left upper and lower extremities. Vital signs recorded on arrival demonstrated tachycardia with a heart rate of 108 beats per minute, hypotension with blood pressure of 87/54 mmHg, respirations of 16 breaths per minute, and pulse oximetry of 93%. Physical exam was significant for facial bruising and abrasions as well as minimal tenderness and swelling of the patient's left wrist. The patient moved all extremities equally and had no motor or sensory deficits. He had no abdominal or chest wall tenderness as well as no spinous process tenderness. A 12-lead ECG was performed on ED arrival, which revealed septal and lateral ST elevations with reciprocal depressions (Figure 1). Although the cardiac catheterization lab was activated for primary percutaneous intervention (PCI)

based on the pre-hospital ECG findings, the patient underwent computed tomography (CT) scanning of the head and cervical spine prior to catheterization in order to evaluate for traumatic injuries given his fall and complaints of extremity weakness. The interventional cardiology team was in agreement.

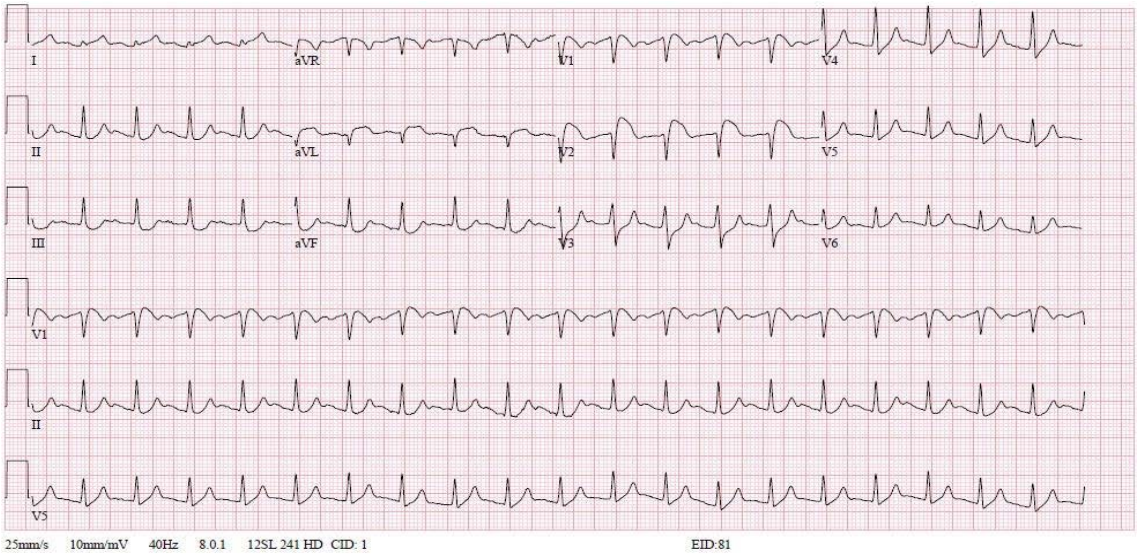


**Figure 1.** Electrocardiogram on presentation to Emergency Department

CT imaging was negative. Before the patient could be taken for cardiac catheterization, laboratory results returned. Initial laboratory tests demonstrated acute renal failure with a serum creatinine of 4.50 mg/dl (reference range 0.60-1.20 mg/dl) and hyperkalemia with serum potassium of 8.5 mmol/L (reference range 3.5-5.0 mmol/L). Additionally, the patient was found to be in rhabdomyolysis with a serum creatine kinase (CK) of 128690 units/L (reference range 0-200 units/L) along with a lactic acidosis of 5.2 mmol/L (reference range 0.5-2.2 mmol/L) (Table 1). Initial cardiac troponin was elevated at 0.53 ng/ml. Urine toxicology was positive for opiates, however negative for other tested illicit substances, including cocaine, amphetamines, benzodiazepines, ethanol, and phencyclidine. Planned cardiac catheterization was canceled, as it was thought his electrocardiographic changes were unlikely to be due to coronary artery disease.

**Table 1. Metabolic Characteristics During Inpatient Stay**

| | Admission | Post-Op Day 1 | Post-Op Day 3 | Post-Op Day 5 | Post-Op Day 7 |
|---|---|---|---|---|---|
| **Serum Creatinine** | 4.5 | 4.2 | 4.1 | 6.6 | 5.4 |
| **Serum Potassium** | 8.5 | 5.4 | 4.1 | 4.2 | 4 |
| **Anion gap** | 26 | 12 | 10 | 11 | 15 |
| **Creatine Kinase** | 128690 | 36430 | 16728 | 12742 | 3419 |
| **White Blood Count** | 46.9 | 16.3 | 15 | 25.9 | 22.5 |
| **Lactic Acid** | 5.2 | 2.3 | 1.9 | - | - |

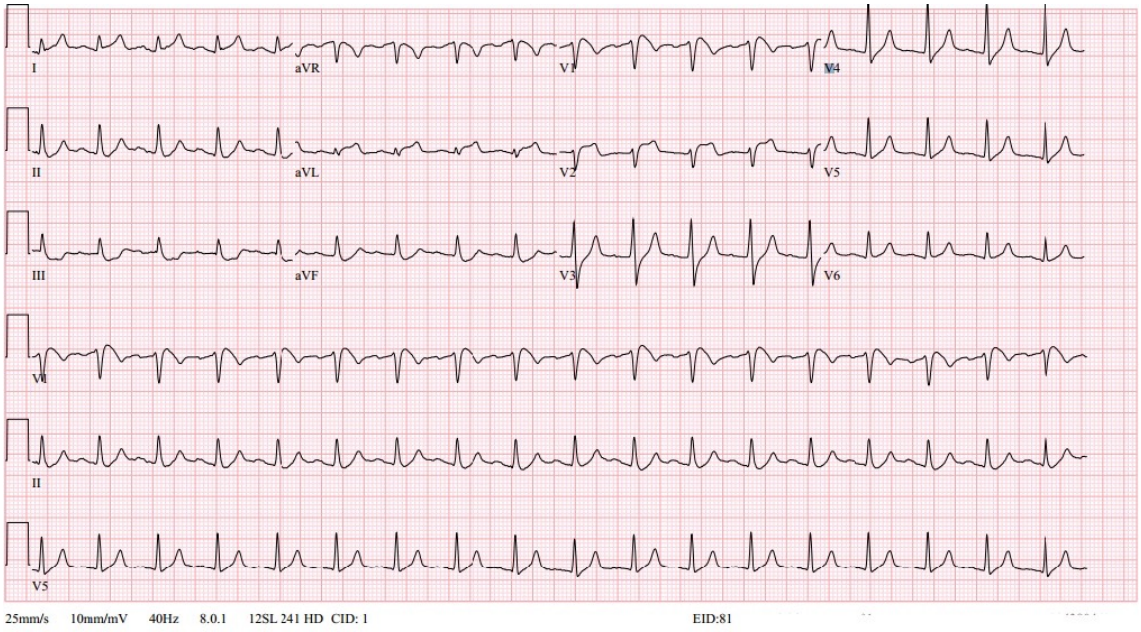


**Figure 2.** Electrocardiogram approximately 2 hours following hyperkalemia treatment

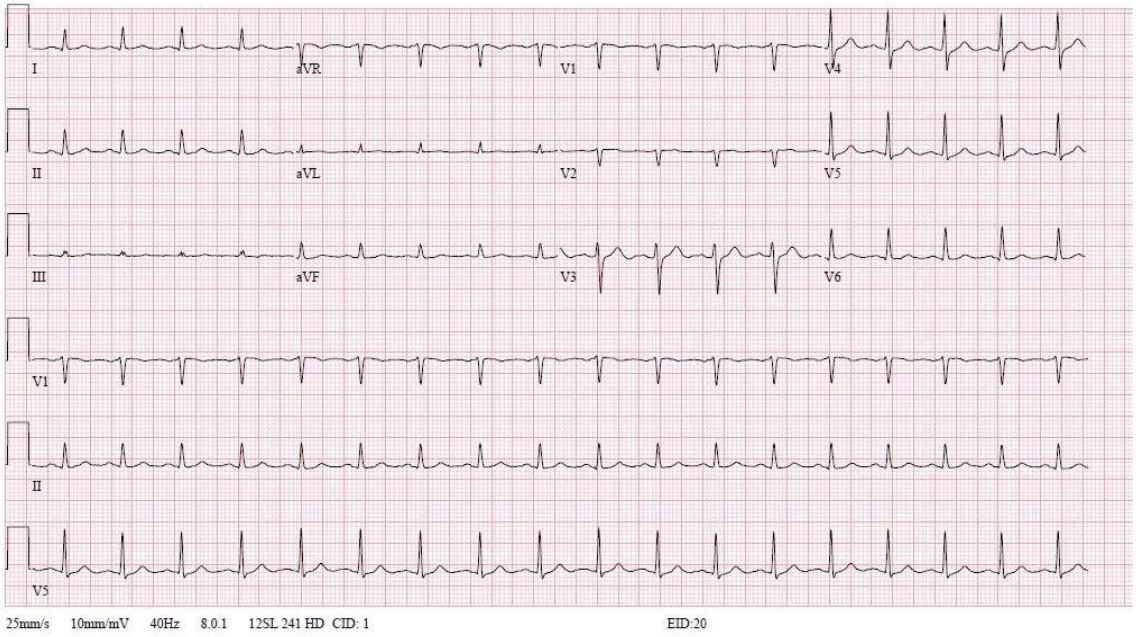


**Figure 3.** Electrocardiogram approximately 12 hours following hyperkalemia treatment

ED staff administered calcium gluconate, insulin, and dextrose for myocardial stabilization in the setting of the patient's severe hyperkalemia and commenced fluid resuscitation with 0.9% normal saline solution. Repeat ECGs demonstrated remarkable resolution of ischemic changes (Figure 2, Figure 3). Echocardiogram revealed no focal wall motion abnormalities.

Approximately 45 minutes after arrival, the patient began to complain of increasing pain in the LUE and LLE. Exam revealed sensory deficits in the ulnar distribution of the left upper extremity, as well as pain and pallor of the left upper and left lower extremities. The patient's left hand was markedly swollen with non-blanchable palmar erythema. Compartment pressures were obtained using a Stryker intra-compartmental pressure monitor system (Table 2). The patient was taken emergently to the operating room for fasciotomy, which revealed bulging sub-fascial compartments in both extremities as well as extensive necrotic tissue in the lower extremity.

**Table 2. Upper And Lower Extremity Compartment Pressures**

| | Compartment Pressure |
|---|---|
| Life Upper Extremity | |
| Dorsal Compartment | 24 mmHg |
| Hypothenar Compartment | 34 mmHg |
| Thenar Compartment | 40 mmHg |
| Life Lower Extremity | |
| Superfical Posterior Compartment | 24 mmHg |
| Deep Posterior Compartment | 29 mmHg |
| Anterior Compartment | 87 mmHg |
| Lateral Compartment | 71 mmHg |

The patient was admitted to the intensive care unit and immediately commenced continuous veno-venous hemofiltration, with progressive improvement of his severe hyperkalemia and elevated CK. Over the next week his renal function improved and continuous dialysis was transitioned to intermittent dialysis. He was eventually discharged to long term rehabilitation for extensive physical therapy and continued dialysis.

## 3. Discussion

Intravenous and opioid drug abuse represent significant public health crises. In the U.S., it is estimated that 425,000 individuals (0.17% of individuals aged 12 or older) inject heroin, cocaine, methamphetamine, or other stimulants [1]. Opioid drug abuse can lead to altered sensorium, including mental and respiratory depression, and result in significant morbidity and mortality. In 2010, opioids accounted for 75.2% of the 38,329 drug overdose deaths in the United States [2]. In addition to those injuries and deaths associated directly with overdose, the consequences of opioid drug abuse are diverse.

Previous case reports have demonstrated the potential for heroin abuse to result in rhabdomyolysis and compartment syndrome [3-10]. Hyperkalemia, a frequent consequence of rhabdomyolysis, is well-known for its various ECG manifestations. Few case reports, however, have documented "pseudo-infarction," or hyperkalemia-induced ECG changes consistent with STEMI [11-18]. Additionally, this appears to be the first incidence to be reported that resulted in compartment syndrome.

Potassium is a predominantly intracellular cation. It plays a pivotal role in the regulation of resting myocardium membrane potential. Aberrations in extracellular potassium concentration lead to disruption of the normal electrical gradient, which can manifest with a broad array of abnormalities on ECG. Classically, the most common ECG changes seen with hyperkalemia are tall "peaked" t waves with eventual widening of the QRS complex. While not always present, these changes typically proportionally with the serum potassium concentration [19]. Hyperkalemia-induced ST-segment elevation may either be a primary repolarization abnormality or an artifact caused by the merging of the QRS segment with the T wave.

Compartment syndrome occurs when an initial injury leads to a rise in intracompartmental pressure within a closed muscle compartment surrounded by fascia and

bone [20]. High pressure leads initially to venule and vein compression, thus further increasing intracompartmental pressure and exacerbating the injury. Eventually, rising compartmental pressure leads to arteriolar compression, causing muscle ischemia and necrosis. Definitive therapy requires emergent fasciotomy to relieve compartment pressure and prevent progression of muscle death [21]. Measuring compartment pressures can assist in diagnosis, however a definitive diagnostic pressure threshold has not been established. Normal compartment pressures range from 0-8 mmHg. Various sources suggest emergent fasciotomy at compartment pressures ranging from 30 to 50 mmHg [22,23]. In our patient, lower extremity compartment pressures far exceeded even the most conservative guidelines. The patient's upper extremity pressures, when taken in context with sensory deficits elicited on physical exam, demonstrated a clear indication for surgery.

## 4. Conclusions

This case effectively highlights the diagnostic dilemma faced by emergency physicians and consulting teams when patients initially present to the ED. Emergency physicians are under pressure to rapidly diagnose and act on ischemic ECGs, specifically STEMIs, often leaving little time for in-depth patient evaluation. The many and varied ECG abnormalities that can be caused by hyperkalemia present a challenge in the interpretation of the ECG of the undifferentiated patient. This case demonstrates the rare pseudo-infarction pattern of hyperkalemia and underscores the importance of clinical context in the initial evaluation of the emergency patient. Additionally, potentially devastating complications of intravenous drug use are well illustrated by this case.

Compartment syndrome and rhabdomyolysis represent rare but catastrophic sequelae of opioid abuse and its induced alteration of consciousness. Fasciotomy and fluid and electrolyte management are cornerstones in the successful management of such cases. Early recognition and effective management of these life- and limb-threatening processes can lead to better outcomes for patients.

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## Consent

Written informed consent for publication of their clinical details and clinical images was obtained from the patient. A copy of the consent form is available for review by the Editor of this journal.

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