

Severe Hyperkalemia: Electrocardiographic Tips for Early Recognition Based on a Case Report

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Abstract

Background: Rapid correction of severe hyperkalemia is mandatory to survival due to its induction of fatal cardiac arrhythmias. The electrocardiogram serves as the diagnostic tool that can provide insight into such fatal arrhythmias. We present two relevant alterations seen in an 84-year-old female patient with previous anterior myocardial infarction, angioplasty of the circumflex coronary artery, left ventricular ejection fraction of 35%, hypertension, dyslipidemia, paroxysmal atrial fibrillation, and diabetes. **The Case:** The patient had 4-days with asthenia, adynamia and dyspnea. Lung auscultation showed bilateral base rales with cardiomegaly and interstitial edema identified on chest x-ray. Lab work revealed severe metabolic acidosis, increased plasma urea, creatinine, and severe hyperkalemia (7.9 mEq/liter) considered secondary to acute renal failure. Treatment was initiated with 0.9% sodium chloride, bicarbonate, ASA diuretics and polarizing solution (insulin), resulting in a reduction of hyperkalemia to 6.1 mEq/liter. The patient suffered a cardiorespiratory arrest with recovery and needed intubation and dopamine for hemodynamic support but died 15 hours after admission. **Conclusion:** The electrocardiographic findings characteristic of severe hyperkalemia were: (i) regular rhythm (cycle length 920-950 ms) without discernible P-waves, which may have a junctional or ventricular origin and less probably could be a manifestation of sinoventricular conduction (preferential conduction from the sinus node to the AV node through specialized tracts without activation of the atrial cardiomyocytes), and (ii) sine wave morphology (markedly wide QRS, absence of ST-segment and broadly based T-waves). These electrocardiographic features, typical of hyperkalemia exceeding 7.0 mEq/liter, are harbingers of malignant arrhythmias and should prompt immediate therapy.

Introduction

Hyperkalemia is a potentially lethal electrolyte disturbance which needs urgent care as it could lead to fatal cardiac arrhythmias. The kidneys (with the capacity to handle high amounts of potassium) play the most important role in the maintenance of normal concentrations of this electrolyte,¹ and consequently renal failure is one of the most prevalent causes for this disorder.

The 12-lead electrocardiogram constitutes a valuable tool for rapid diagnosis and shows a wide spectrum of alterations:² (i) peaked high-amplitude and narrow-based T-waves (usually later replaced by broad-based T-waves and widening of the QRS), (ii) decreased amplitude or absence of the P-wave, (iii) conduction disturbances, (iv) ST-segment elevation, (v) absence of ST-segment and merging with the wide QRS, (vi) pulseless electrical activity, (vii) non-shockable ventricular tachycardia and (viii) ventricular fibrillation.

A case of severe hyperkalemia and typical electrocardiographic changes constitutes the subject of our presentation.

The Case

An 84-year-old woman presented to the emergency room with 96-hour evolution of asthenia, adynamia, and dyspnea. She had

Highlights:

- Critical importance of early electrocardiographic recognition in severe hyperkalemia through a patient case.
- Urgency of treatment for hyperkalemia exceeding 7.0 mEq/liter to prevent fatal arrhythmias.
- Challenges in managing complex cases with delayed presentation, underscoring the need for rapid diagnostic and therapeutic interventions.

prior history of an anterior myocardial infarction treated with angioplasty of the circumflex artery, severe depression of the left ventricular systolic function (ejection fraction 35%), hypothyroidism, chronic obstructive pulmonary disease, arterial hypertension, paroxysmal atrial fibrillation, dyslipidemia, and non-insulin dependent diabetes mellitus type 2. The patient was on inhaled budesonide, bisoprolol, amiodarone, enalapril, aspirin, rosuvastatin and metformin. Anticoagulation was not indicated because of frailty and low compliance. The patient lived by herself and her income was limited. Her mother (deceased) had suffered hypertension and she ignored the medical history of her father.

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Her vital signs on admission: Blood pressure 100/60 mmHg, heart rate 50 per minute, oxygen saturation 87%, and temperature 36°C. Auscultation of the heart and lung fields revealed normal sounds and absence of murmurs, with bilateral lung base rales. There was lividity in the lower limbs.

The patient was awake and disoriented in person, place, time and situation. She had no fever or chills. Abdominal palpation revealed soft and mildly tender abdomen, with no guarding or peritoneal signs.

An anteroposterior chest X-ray showed no evidence of acute lung disease (*Figure 1*).

Lab work was performed including cell counts, metabolic and urinary profiles, and cardiac biomarkers. Renal panel showed markedly elevated urea (228 mg/dl) and creatinine (3.75 mg/dl). There was severe hyperkalemia (7 mEq/liter) and severe metabolic acidosis, with a pH of 7.11 and bicarbonate of 11 mmol/liter (Table). Hematologic investigations (cell counts, INR) were normal, as well as CPK, while ultra-sensitive troponin was elevated (81.3 ng/liter for a normal value <19 ng/liter). Initial urinalysis was not suggestive of urinary tract infection. The patient was diagnosed with hyperkalemia (7.9 mEq/liter) secondary to acute renal failure. The 12-lead ECG showed regular rhythm with a cycle length of 920-950 ms without discernible P-waves, marked widening of the QRS complex (200 ms) and absence of ST-segment (*Figure 2*). In the emergency room, IV insulin was started at 10 units/hour and IV dextrose (5% in 500 ml for 8 hours) to prevent hypoglycemia. Treatment was complemented with 0.9% sodium chloride, bicarbonate and ASA diuretics.

Two hours after hospital admission, she suffered a cardiorespiratory arrest which required advanced resuscitation maneuvers followed by rapid sequence intubation for mechanical respiratory assistance with O₂ at 50% and hemodynamic support with vasoactive drugs (dopamine). Three hours later, a lab work showed K⁺ of 6.1 mEq/liter, and initial normalization of the 12-lead ECG (*Figure 3*).

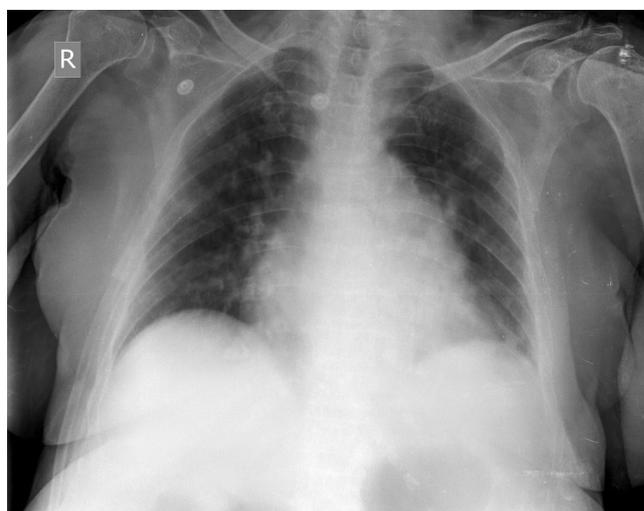
The patient's decompensation and persistent hypotension progressed gradually despite the increase in the dose of dopamine and the addition of noradrenaline. The marked hypotension and progressive decompensation precluded dialysis, and she died 15 hours post-admission.

Discussion

Sir Humphrey Davy was the first to isolate potassium (K⁺) by electrolyzing plant ashes "soaked in pots of water" (the term "pots" gives origin to the name of the electrolyte), as Weiss et al. point out in an interesting review.³ The largest amount of potassium in our bodies (98%) is intracellular, with a significant gradient in relation with the extracellular space. An increase in the serum concentration of potassium (above 5.5 mEq/liter) leads to electrophysiological disturbances mainly characterized by shortening

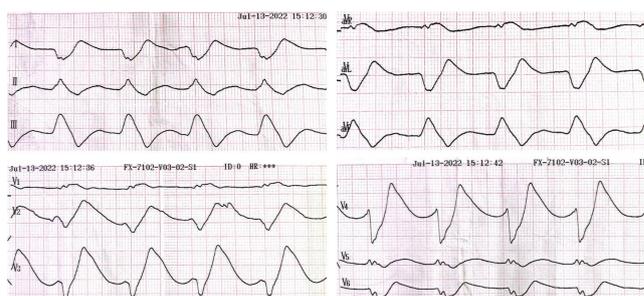
of the action potential and slow conduction. With increasing values, the electrocardiographic manifestations aggravate and may trigger malignant arrhythmias and death.⁴

Figure 1. Antero-Posterior Chest X-Ray Showing Cardiomegaly and Features of Interstitial Pulmonary Edema.



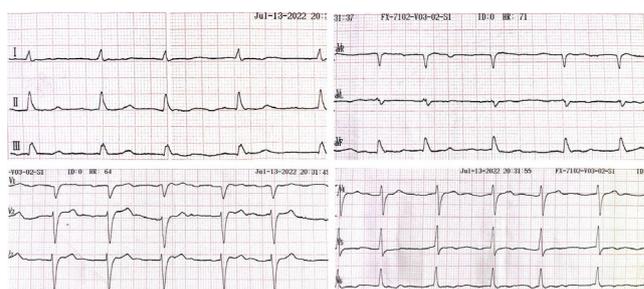
Legend: There are signs of interstitial pulmonary edema including poorly defined large pulmonary vessels, appearance of septal lines, interlobar septal thickening and diffuse reticular pattern associated with cardiomegaly.

Figure 2. Initial 12-Lead ECG Indicative of Severe Hyperkalemia.



Legend: The figure shows a regular rhythm with the typical sine-wave pattern: extremely wide QRS, absence of ST-segment and broadly based T-waves.

Figure 3. Follow-up 12-Lead ECG Showing Normalization Post-Treatment.



Legend: With a potassium level of 6.1 mEq/liter and atrial fibrillation, the QRS complex is narrow and the ST-segment reappears with normal voltage of the T-wave.

The 12-lead ECG is a readily available tool and helps establishing the potential risks better than the mere potassium level, which in fact should be considered in conjunction with the rate of increase and the duration of the elevated values.⁴ In a study from a single institution,⁵ the electrocardiograms recorded within one hour of potassium determination in 188 patients with concentration of the electrolyte >6.5 mEq/L were reviewed and the correlation between the electrocardiographic abnormalities and the occurrence of adverse events (symptomatic bradycardia, ventricular tachycardia or fibrillation, necessity of resuscitation maneuvers or death) was analyzed. The authors found adverse events in 15% and all occurred before calcium administration and all but one prior to interventions to lower potassium. Three electrocardiographic alterations were associated (alone or in combination) with adverse events: wide QRS (relative risk [RR] 4.74), bradycardia <50 beats/min (RR 12.29) and junctional rhythm (RR 7.46). Despite the need for a rapid intervention, the diagnosis may be challenging, especially in the presence of the so-called “dialyzable current of injury”, as some electrocardiographic changes seen in hyperkalemia may resemble pericarditis, STEMI (ST-elevation myocardial infarction)⁶ or type I Brugada pattern.⁷

Two Signs are Characteristic of Severe Hyperkalemia

1) **Junctional Rhythm:** The *regular* rhythm ([Figure 2](#)) with a cycle length of 920-950 ms may have a junctional or ventricular origin as P waves were not discernible. In the context of hyperkalemia could also be an example of “sinoventricular conduction”, in which the rhythm originates in the sinus node but – as a result of the alteration in transmembrane atrial potential-P-waves are not discernible because the impulse is supposed to be transmitted to the AV node through internodal tracts without activation of the cardiomyocytes.⁸⁻¹⁰ This would have been definitively proved in case the P-wave appeared at a similar rate once the electrolyte disturbance had been solved, but in this patient we may only mention the existence of the phenomenon as P-waves cannot be seen (in [Figure 3](#) the underlying rhythm was atrial fibrillation once the potassium level had been lowered).

2) **Sine-Wave Morphology:** Our case, with a potassium level of 7.9 mEq/liter, shows the typical sine-wave morphology: (i) markedly wide QRS due to reduced influx of sodium and slower

rise of the action potential,² and (ii) absence of ST-segment and broadly based T-wave (which differs from the “tenting” T-waves of high amplitude and narrow base seen in less pronounced hyperkalemia). Early recognition of this pattern may result in saving the life of a patient, as it correlates with hyperkalemia exceeding 7.0 mEq/liter.

In conclusion, electrocardiographic patterns present in this patient are characteristic of severe hyperkalemia and must prompt measures to lower potassium concentration and prevent death.

Summary – Accelerating Translation

La hiperpotasemia se define por una concentración de potasio en sangre mayor de 5,5 miliequivalentes por litro. Este trastorno puede tener diferentes causas, pero dado que los riñones tienen un rol primordial en su eliminación, la causa más común es la insuficiencia renal.

El tratamiento de la hiperpotasemia severa (cuando la concentración supera los 7 miliequivalentes por litro) es una urgencia ya que de lo contrario puede ocurrir un paro cardíaco.

En esta presentación se describe el caso de una paciente de 84 años que sufría de enfermedad coronaria (inclusive había padecido un infarto de miocardio), era hipertensa, tenía aumento de los lípidos en sangre, enfermedad pulmonar obstructiva crónica (EPOC), fibrilación auricular (una arritmia relativamente común en personas de edad avanzada) y era diabética.

Llegó a la sala de Guardia con 4 días de evolución de un cuadro de disnea (falta de aire) y cansancio. Estaba desorientada y, si bien en el examen físico no había hallazgos específicos, la radiografía de tórax mostraba agrandamiento del corazón e infiltrados pulmonares que eran claramente anormales. El electrocardiograma resultó de gran valor ya que había signos que no solo son característicos de la hiperpotasemia, sino que se asocian con un aumento del potasio superior a 7 miliequivalentes por litro.

Se iniciaron de inmediato todas las medidas que contribuyen a reducir el potasio, pero el cuadro generalizado de la paciente, con enfermedad multiorgánica, y el hecho de que la consulta se hubiera demorado 4 días desde el comienzo de los síntomas, impidieron una evolución favorable y lamentablemente falleció a las 15 horas del ingreso.

Este caso es importante porque muestra en el electrocardiograma dos de los hallazgos que multiplican el riesgo de muerte en presencia de potasio aumentado. El pronto reconocimiento de esos trastornos electrocardiográficos permite un tratamiento que muchas veces resulta efectivo.

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