

Hyperkalemia Mimicking Anteroseptal Myocardial Infarction: a Rare Feature that Confuses Clinicians

Raka Aldy Nugraha¹, Auliya Husen¹, Hary Sakti Muliawan², Dian Zamroni²

Abstract

Background: Hyperkalemia often results in cardiac emergencies associated with fatal cardiac arrhythmias. However, the presence of ST-segment elevation in hyperkalemia is rare and could potentially subject the patients to unnecessary risk of intervention. Most commonly, ST elevation in hyperkalemia presents in a down-sloping fashion compared to the typical convex or upsloping pattern in myocardial infarction. However, in some cases, the ST elevation morphology can be very identical and difficult to distinguish. Herein, we describe a hyperkalemic patient presenting with non-ischemic ST segment elevation that resolved spontaneously following therapy.

Case Illustration: A 77-year-old, bedridden, inarticulate woman was admitted to the emergency department with acute dyspnea perceived in the last 1.5 hours before admission. The patient's past clinical history includes craniotomy for subdural hematoma, poorly controlled hypertension, hypertensive heart disease, rheumatoid arthritis, and dementia, and was under candesartan, amlodipine, nebivolol, spironolactone, and atorvastatin treatment. The 12-lead electrocardiography (ECG) recording showed a wide QRS complex with left bundle branch block pattern, slow atrial fibrillation with total atrioventricular block, ST-segment elevation, Q wave in anteroseptal leads, and peaked T wave. The pattern of ST elevation was indistinguishable from that of myocardial infarction which necessitated further laboratory confirmation. Laboratory results showed severe hyperkalemia (K⁺ 7.93 mmol/L) and normal troponin level (45.0 ng/L). The patient was given serial insulin-based therapy and calcium gluconate immediately. The follow-up ECG pictured normal sinus rhythm with no sign of bundle branch block, resolution of ST-segment elevation, and reduction in T wave amplitude. However, the reduction in potassium level was not significant and the patient also experienced an acute kidney injury. The patient was transferred to the intensive care unit and was prepared for hemodialysis.

Conclusion: ST-segment elevation is a rare feature of hyperkalemia that could mislead the patient's treatment. Thorough ECG evaluation is the key to narrowing down the differential diagnosis. Every deviant feature should not be interpreted separately. Laboratory tests could help confirm the diagnosis, particularly in patients with atypical presentation, and could help avoid unnecessary risk of intervention.

(Indonesian J Cardiol. 2023;44:28-32)

¹ Universitas Indonesia Hospital, Depok, West Java, Indonesia.

² Department of Cardiology and Vascular Medicine, Universitas Indonesia Hospital, Depok, West Java, Indonesia.

Correspondence:

Raka Aldy Nugraha,

Department of General Medicine,
Universitas Indonesia Hospital,
Universitas Indonesia, 16424,
Pondok Cina, Beji, Depok,
Indonesia.

Email: rakaaldynugraha@hotmail.com

Keywords: Hyperkalemia, non-ischemic ST elevation, arrhythmia.

Introduction

Hyperkalemia is a condition characterized by an increase in the plasma potassium level. There is no international agreement on the definition and classification of hyperkalemia, however, according to the European Resuscitation Council, hyperkalemia is defined as a plasma potassium level of >5.5 mmol/L, while the term severe hyperkalemia is applied when the potassium level is >6.5 mmol/L.¹ For years, potassium has been shown to have a pivotal role in cardiac electrophysiology and any deviation from its normal level could manifest as electrocardiographic (ECG) abnormalities. In patients with mild hyperkalemia, the ECG may show the presence of tall, peaked, narrow-based T waves in precordial leads and evidence of fascicular blocks. Patients with moderate hyperkalemia may present with p-wave abnormalities, sinus arrest, atrioventricular block, and ST segment abnormalities, most commonly ST depression. Moreover, atypical bundle branch block, intraventricular conduction delay, idioventricular rhythm, ventricular tachycardia, ventricular fibrillation, sine wave appearance, and asystole could be found in patients with severe hyperkalemia.²⁻⁴ Occasionally, patients with hyperkalemia could also present with ST elevation on their ECG recording.⁵⁻⁹ Most commonly, the ST elevation in hyperkalemia has a down-sloping characteristic, however, it could also rarely be identical to that of acute myocardial infarction (MI) which has a plateau or upsloping pattern.⁴ Hence, even when it is infrequent, this finding is very important as it could mislead our diagnosis and treatment and subsequently subject the patients to the risk of unnecessary intervention.⁵ Here we describe a case of a severely hyperkalemic patient presenting with ST elevation that mimicked anteroseptal MI.

Case illustration

A 77-year-old, bedridden, inarticulate woman was admitted to our emergency room by her family. The patient was reported to experience dyspnea 1.5 hours before admission. Their patient was not fully conscious, but the family was not sure whether there was an acute episode of altered mental status as the patient had never been fully aware due to her illness. Other complaints

were difficult to identify as the patient was inarticulate. The patient's medical history included craniotomy for subdural hematoma, uncontrolled hypertension, hypertensive heart disease, rheumatoid arthritis, and dementia. The patient was under routine medications which included candesartan, amlodipine, nebivolol, spironolactone, and atorvastatin. On the primary survey it was noted that the patient was not fully conscious (Glasgow Comma Scale [GCS] E3M5V3), dyspneic (respiratory rate 26 breaths/min) with evidence of oxygen desaturation (oxygen saturation [SpO₂] range 84%-93%), hypertensive (blood pressure 155/80 mmHg), with episodes of bradycardia (heart rate range 31-99 bpm). Other important findings included bilateral pulmonary rales and arrhythmia. The bedside monitor showed a very dynamic rhythm consisting of accelerated junctional rhythm alternating with slow atrial fibrillation (AF), Wenckebach-type atrioventricular (AV) block, and accelerated idioventricular rhythm (AIVR).

Twelve-lead electrocardiography (ECG) showed wide QRS complex with left bundle branch block (LBBB) pattern, slow AF with total AV block; tall, peaked, narrow-based T waves; upsloping ST segment elevation in V1-V4, and deep Q waves in V1-V2 (**Figura.1**). The presence of ST elevation in anteroseptal leads was confusing as to whether an ST-elevation myocardial infarction (STEMI) protocol should be activated or not. We instead performed some confirmatory studies before activating the STEMI protocol. Laboratory studies demonstrated that the patient was severely hyperkalemic (K⁺ level 7.93 mEq/L) with normal troponin levels (**Table 1**). Hence, the diagnosis of severe hyperkalemia was established, and the possibility of STEMI was excluded. The patient was prescribed three doses of insulin-based therapy given in 3-hour intervals and calcium gluconate. A follow-up ECG was performed after three hours which showed normal sinus rhythm, no signs of bundle branch block and AV block, resolution of ST-segment elevation, and reduction in T wave amplitude. A follow-up electrolyte study showed the patient was still severely hyperkalemic (K⁺ level 7.11 mEq/L). The patient was transferred to the intensive care unit for urgent hemodialysis. However, due to our limited resources related to the COVID-19 pandemic, the hemodialysis was postponed, and insulin-based treatment was maintained with the same regimen. On the following day, another electrolyte study was

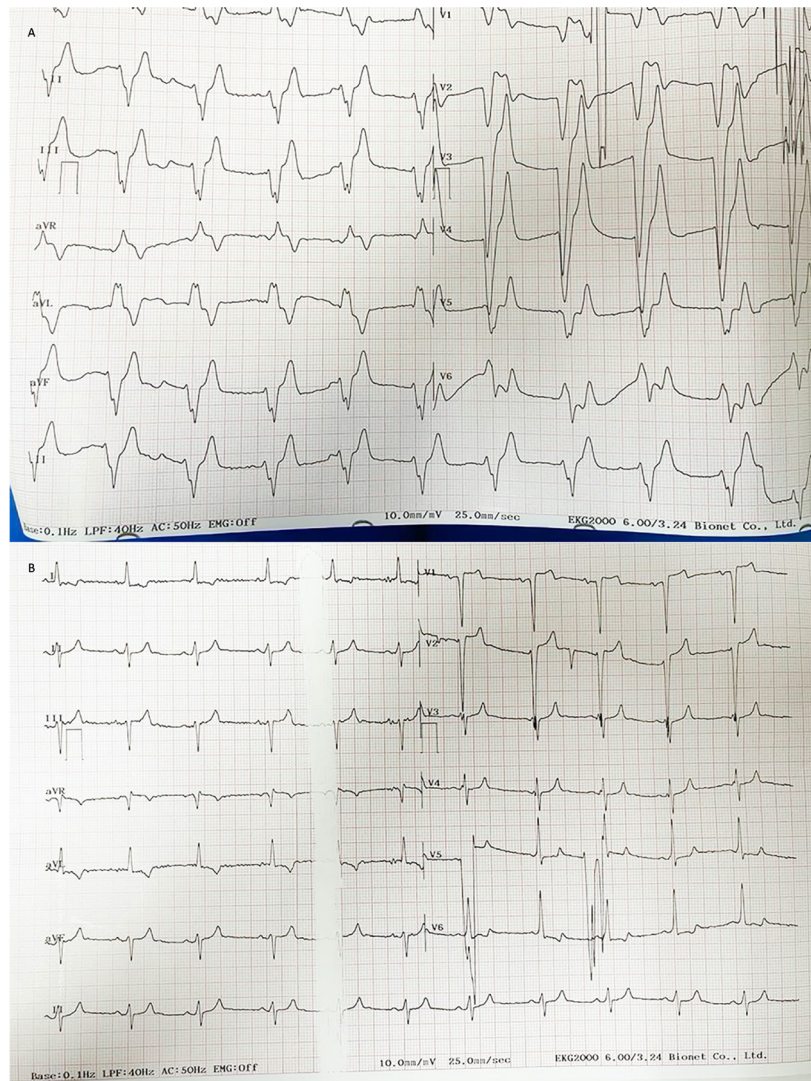


Figure 1. Electrocardiographic findings. A) Initial ECG showing wide QRS complex with left bundle branch block pattern, slow atrial fibrillation with total atrioventricular block, ST segment elevation in anteroseptal leads (V1-V4), Q waves in V1-V2, and peaked T wave. B) Follow-up ECG showing normal sinus rhythm with no sign of bundle branch block, resolution of ST segment elevation, and reduction in T wave amplitude. C) Follow-up ECG on the second admission showing relatively similar patterns to the previous ECG

undertaken showing that the potassium level was reversed back to normal (K^+ 4.94 mEq/L). The patient was discharged on day 10 of hospitalization.

Discussion

The role of potassium in cardiac electrophysiology is pivotal. Physiologically, the K^+ level is strictly regulated between its normal range of 3.5 to 5.0 mmol/L to keep the negative resting membrane potential (E_m).

This negative E_m helps in stabilizing atrial and ventricular myocytes during diastole, thus preventing the generation of spontaneous action potentials that manifest as premature contraction.¹⁰ Because of its role in maintaining normal cardiac electrophysiology, any deviation in potassium levels outside its normal range could manifest as ECG abnormalities.

Hyperkalemia is a common electrolyte abnormality with protean manifestations and is often progressive in nature.⁶ Hyperkalemia has long been known as the cause

of variable arrhythmias. ECG changes in hyperkalemia usually depend on the severity and rapidity of the onset and could potentially be life-threatening.² During systemic hyperkalemia, APD shortens and conduction velocity is altered as K^+ equilibrium potential (E_k) becomes less negative and therefore, prolonging the effective refractory period.¹⁰ ECG changes in hyperkalemia are progressive, following the increase in potassium levels. Initially, in patients with potassium levels between 5.5-6.0 mEq/L, the first encounter is usually typical tall, peaked, symmetrical narrow-based T waves, and sometimes evidence of fascicular block could also present.¹¹ As the potassium level continues to increase, cardiac conduction velocity decreases. The atrial myocytes are generally more sensitive to hyperkalemia compared to ventricular cells.² Therefore, changes in p-wave morphology are usually seen earlier. Patients may present with p wave widening and decreased p wave amplitude, followed by p wave disappearance.² In patients with severe hyperkalemia (K^+ levels >7.5 mEq/L), wide QRS complex with atypical bundle branch block appearance, idioventricular rhythm, ventricular tachycardia, ventricular fibrillation, sine wave appearance, and cardiac arrest may be found.^{2,12}

Although rare, the presence of ST-segment elevation has been discussed in hyperkalemia.¹³ The mechanism of ST elevation in hyperkalemia remains poorly understood, however, it is thought that APD shortening in phase three repolarization induces elevation of the ST segment in ECG.^{5,6} However, the APD shortening, along with resting membrane potential depolarization, causes both diastolic and systolic injury currents that would manifest not only as ST elevation but also as TQ depression. For this reason, ST elevation in hyperkalemia can sometimes look profound due to its combination with TQ depression.¹⁰ In general, ST elevation in hyperkalemia presents in the downsloping pattern.⁴ However, the ST elevation can be either upsloping or plateau creating a pseudo-infarction pattern. It has been noted that in 80% of hyperkalemia-induced pseudo-infarction, the ST elevation involves anteroseptal leads and aVR with the presence of Q waves in V1-V2. Some isolated inferior and anterolateral pseudo-infarctions have also been described. These findings were encountered in patients with mean potassium levels of 8.1 mEq/L and mostly found in the setting of diabetic ketoacidosis and renal insufficiency. The

pseudo-infarction pattern usually resolves following hyperkalemia correction.^{5,6,8,9,11,13} These findings are similar to what we encountered in our patients. Our patient presented with a pseudo-infarction pattern in anteroseptal leads that resolved spontaneously following therapy.

Our finding adds new evidence to the literature showing that myocardial infarction is not the only pathology causing ST elevation. Even when the characteristics can be somewhat convincing, thorough clinical and ECG evaluation must be undertaken. Activating the STEMI code without consideration of the non-ischemic cause of ST-segment elevation could subject our patient to the risk of unnecessary intervention and delay administration of the appropriate treatment.⁶ In a resource-capable setting, confirmation of myocardial infarction through cardiac marker evaluation should be performed in patients with multiple ECG findings, particularly in patients with atypical clinical presentation. On the other hand, in limited facilities, consultation with a cardiologist should always be done early and, in the setting of hyperkalemia, any ECG characteristics of hyperkalemia, particularly with confirmation of potassium level, should navigate the physician to immediately prescribe corrective treatment while assessing for the possibility of concomitant STEMI and bleeding risk. While doing so, serial ECG should be performed to evaluate for any resolution in ST elevation.

Conclusion

ST-segment elevation is a rare feature of hyperkalemia that could mislead the patient's treatment. Thorough ECG evaluation is the key to narrowing down the differential diagnosis. Every deviant feature should not be interpreted separately. Laboratory studies could help confirm the diagnosis, particularly in patients with atypical presentation, and could help avoid unnecessary risks of intervention.

Conflict of Interest

The authors declared no conflict of interest.

Funding

We confirm that we did not receive any grant from

any agencies, both in the profit and non-profit sectors.

References

1. 1. Truhlář A, Deakin CD, Soar J, Khalifa GEA, Alfonzo A, Bierens JJLM, et al. European Resuscitation Council Guidelines for Resuscitation 2015. *Resuscitation* [Internet]. 2015 Oct;95:148–201. Available from: <https://linkinghub.elsevier.com/retrieve/pii/S0300957215003299>
2. Campese VM, Adenuga G. Electrophysiological and clinical consequences of hyperkalemia. *Kidney Int Suppl* [Internet]. 2016 Apr;6(1):16–9. Available from: <https://linkinghub.elsevier.com/retrieve/pii/S2157171616000046>
3. Diercks DB, Shumaik GM, Harrigan RA, Brady WJ, Chan TC. Electrocardiographic manifestations: electrolyte abnormalities. *J Emerg Med* [Internet]. 2004 Aug;27(2):153–60. Available from: <https://linkinghub.elsevier.com/retrieve/pii/S0736467904001416>
4. Wang K, Asinger RW, Marriott HJL. ST-Segment Elevation in Conditions Other Than Acute Myocardial Infarction. *N Engl J Med* [Internet]. 2003 Nov 27;349(22):2128–35. Available from: <http://www.nejm.org/doi/abs/10.1056/NEJMra022580>
5. Heckle M, Agarwal M, Alsafwah S. ST Elevations in the Setting of Hyperkalemia. *JAMA Intern Med* [Internet]. 2018 Jan 1;178(1):133. Available from: <http://archinte.jamanetwork.com/article.aspx?doi=10.1001/jamainternmed.2017.6329>
6. Peerbhai S, Masha L, DaSilva-DeAbreu A, Dhoble A. Hyperkalemia masked by pseudo-stemi infarct pattern and cardiac arrest. *Int J Emerg Med* [Internet]. 2017 Dec 26;10(1):3. Available from: <https://intjem.biomedcentral.com/articles/10.1186/s12245-017-0132-0>
7. Ziakas A, Basagiannis C, Stiliadis I. Pseudoinfarction pattern in a patient with hyperkalemia, diabetic ketoacidosis and normal coronary vessels: a case report. *J Med Case Rep* [Internet]. 2010 Dec 26;4(1):115. Available from: <https://jmedicalcasereports.biomedcentral.com/articles/10.1186/1752-1947-4-115>
8. Wray J, Yoo MJ, Bridwell RE, Tannenbaum L, Henderson J. ST-Segment Elevation in the Setting of Diabetic Ketoacidosis: Is It Acute Coronary Syndrome? *Cureus* [Internet]. 2020 Mar 25; Available from: <https://www.cureus.com/articles/29251-st-segment-elevation-in-the-setting-of-diabetic-ketoacidosis-is-it-acute-coronary-syndrome>
9. Sims DB, Sperling LS. ST-Segment Elevation Resulting From Hyperkalemia. *Circulation* [Internet]. 2005 May 17;111(19). Available from: <https://www.ahajournals.org/doi/10.1161/01.CIR.0000165127.41028.D1>
10. Weiss JN, Qu Z, Shivkumar K. Electrophysiology of Hypokalemia and Hyperkalemia. *Circ Arrhythmia Electrophysiol* [Internet]. 2017 Mar;10(3). Available from: <https://www.ahajournals.org/doi/10.1161/CIRCEP.116.004667>
11. Bellazzini MA, Meyer T. Pseudo-Myocardial Infarction in Diabetic Ketoacidosis with Hyperkalemia. *J Emerg Med* [Internet]. 2010 Oct;39(4):e139–41. Available from: <https://linkinghub.elsevier.com/retrieve/pii/S0736467907003551>
12. Slovis C. ABC of clinical electrocardiography: Conditions not primarily affecting the heart. *BMJ* [Internet]. 2002 Jun 1;324(7349):1320–3. Available from: <https://www.bmj.com/lookup/doi/10.1136/bmj.324.7349.1320>
13. Pothiwala S. Hyperkalemia induced pseudo-myocardial infarction in septic shock. *J Postgrad Med* [Internet]. 2014;60(3):338. Available from: <http://www.jpgmonline.com/text.asp?2014/60/3/338/138828>.