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Case Report

Hyperkalemia Diagnosis and Management for a Total AV Block Patient with a Pseudo-STEMI Infarction Pattern: A Case Report

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ABSTRACT

Background: Hyperkalemia is an electrolytic disorder that is often encountered in hospitals and may be life-threatening. The ECG abnormalities that occur vary from tall T to deadly arrhythmia. ST segment elevation is rarely encountered and may be confused for the alternative diagnosis of myocardial infarction. **Case Summary:** The patient is a 53 year-old male with total AV block with an ECG pseudo-STEMI infarction pattern. Initial diagnosis was acute myocardial infarction; it was then known that the patient suffered from chronic kidney disease with hyperkalemia. The patient recovered after obtaining hyperkalemia therapy. **Conclusion:** In summary, hyperkalemia manifestation on ECG depends on potassium levels and comorbidities. Manifestation as ST segment elevation and total AV block are rare case, prompt diagnosis and right treatment may reduce the mortality.

Highlights:

1. A surprising finding in which manifestation of hyperkalemia depends on potassium levels and comorbidities.
2. The manifestation as ST elevation and total AV block are considered rare cases, which is why this article is very interesting to read.

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Introduction

Hyperkalemia is a condition that is potentially life-threatening and may cause arrhythmia, usually when the serum potassium level exceeds 8 mmol/L. This often occurs for patients with reduced kidney excretion, among users of heart failure medications, or when intracellular potassium leaks into intercellular space [1]. There are no clinical symptoms or distinct signs from physical inspection, and therefore diagnosis is often missed.[2]

Disorder manifestation on electrocardiography (ECG) depends on potassium levels and comorbidities. ECG may indicate symmetrical peaking of T wave, QRS widening, AV block, shortening of PR segment, decreasing to disappearing P wave amplitude, disappearing sinoatrial conduction (classic sinewave), and even asystole [3]. ECG manifestation as ST segment elevation may occur, although rarely discovered, and thus may be confused for myocardial infarction [4]. This paper involves the presentation of a hyperkalemia patient with an initial diagnosis of ST elevation myocardial infarction (STEMI) and discusses its pathophysiology and management.

Case Presentation

The patient is a 53 year-old male with complaints of epigastric pain accompanied by sudden breathlessness and continuous pain that did not resolve with rest. The patient also complained of queasiness and vomiting. The patient admitted of having last urinated approximately 12 hours before being admitted to the hospital, with a small volume. In the week prior, the patient was hospitalized with diagnosis of heart failure. The patient appeared weak and fully conscious, with blood pressure 130/90 mmHg, pulse 37 x/minute, breathing frequency 24 x/minute with O2 saturation of 97%, and axillar temperature of 36.1°C. Pressure pain was indicated in the epigastrium area. Rhonchi or wheezing were not indicated and extremities were warm.

Results of laboratory examination showed a reduction in hemoglobin (10 mg/dL) and an increase in leukocytes (13.400/µL) and thrombocytes (633.000/µL), increase in potassium levels (7.53 mmol/dL), increase in urea (159 mg/dL) and creatinine (6.31 mg/dL), and estimated glomerular filtration rate (eGFR) of 11.49 mL/min/1,73 m2, while troponin examination was negative. ECG showed a total AV block with a ventricular rate of 33x/minute, ST elevation for V2, V3, V4, and hyperacute T for II, III, aVF recordings (Figure 1a).

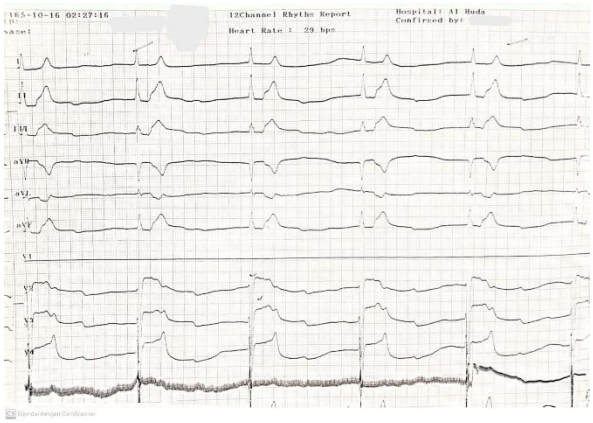

Figure 1a. ECG imaging at the ICU

Therapy for acute coronary syndrome (ACS) had been given in the emergency room (ER). Then, after laboratory results were found for hyperkalemia, furosemide injection of 40 mg, dopamine drip 5 mcg/kg BW/minute, and NaCl IV 0.9% 2000cc/24 hours were administered, and ACS therapy was terminated. (Figure 1b).

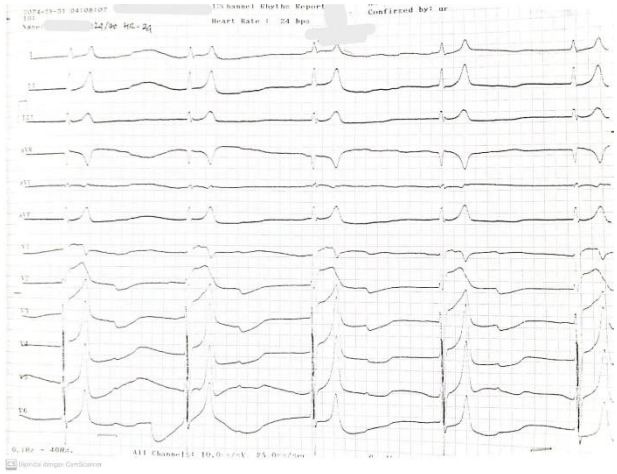

Figure 1b. ECG II total AV block, anteroseptal ST elevation, and tall T

On the second day of treatment, complaints decreased, hemodynamics were stable, and urine production was 1850 cc/24 hours. Therapies of Ca gluconate 10% 10 mL injection, dextrose 40% + actrapid 2 IU repeated for 3 cycles were added, and furosemide was continued with a dose of 3x20 mg. Potassium levels decreased to 5.98 mmol/L. On the third day of treatment, ECG showed improvement, with pulse 75 bpm, PR interval normal, ST segment isoelectric, and T waves normal (Figure 1c).

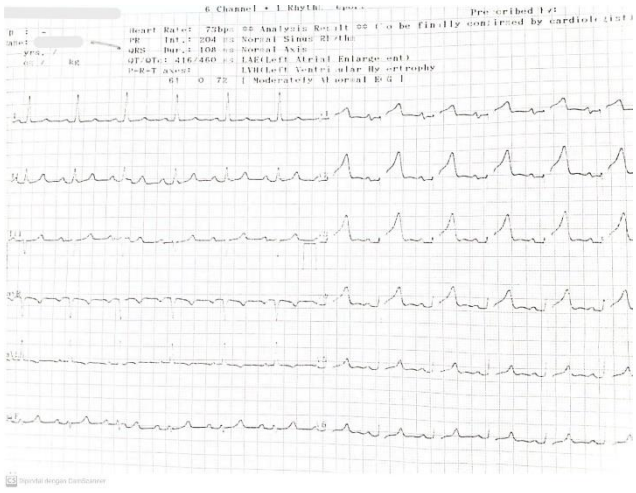

Figure 1c. ECG III sinus rhythm 75xm, normal axis, no ST-T changes

On the fourth day, potassium levels were still high (6.09 mg/dL), and nebulized salbutamol 2.5 mg was administered. Up to the seventh day of treatment, average daily urine production was 2000 cc and potassium levels were still above normal (5.77 mg/dL), with an increase in urea (167.5 mg/dL) and creatinine (7.64). The patient was sent home with an oral therapy of furosemide and sodium bicarbonate.

Results of echocardiography examination for the patient showed the presence of concentric hypertrophic left ventricle, diastolic dysfunction, mild dilatation of the left atrium, and mild mitral and

tricuspid regurgitation, and regional heart wall motion abnormalities were not indicated.

Discussion

Most patients of hyperkalemia do not suffer from distinct signs and symptoms, and thus the diagnosis is often missed by physician. Laboratory results confirm suspicions, as initial hints of electrolyte disorder suspicions are usually obtained from ECG imaging. Unfortunately, the ability of physicians to predict the presence of hyperkalemia only based on ECG is low, sensitivity of 34-43%, and specificity of 85-86%. For hyperkalemia of greater than 6.5 mmol/L, the sensitivity of examinations is higher, between 55-62%.[5,6]

Acute hyperkalemia with manifestation resembling acute STEMI on ECG imaging is a case that is rarely discovered. The ECG imaging pattern that occurs for 80% of reported cases comprise anteroseptal pseudo-infarctions, and patterns of inferior and anterolateral infarctions have also been reported.[4,7] Supporting examinations such as through tools of echocardiography become very useful in supporting diagnostics in this situation. Findings in echocardiography, as regional heart wall motion abnormalities, support the diagnosis for myocardial ischemia.[8]

The increase of potassium concentration in extracellular compartments, such as in interstitials due to myocardial infarctions or systemic hyperkalemia due to disruption of kidney functions, will reduce the concentration gradient of potassium and its potential (E_K) along the sarcolemma. Although the difference in potassium [K+] concentration reduces the locomotive force of potassium ions (because the E_m-E_K difference becomes smaller), the potassium channel has an allosteric property, and thus conductance increases instead. Therefore, the duration of repolarization (phase 2-3) decreased and conduction velocity increased (millivolt/second) (Figure 2).[3,9]

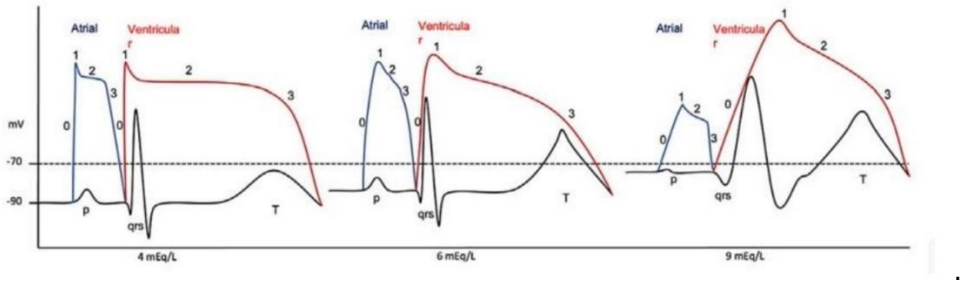

Figure 2. Membrane potential of ventricle and surface ECG on the change from normal condition to hyperkalemia

The decrease in E_m reduces the number of Na+ channels that could be activated, causing phase 0 to fall. The falling of phase 0 shows a widening of QRS duration. The increase in conduction velocity causes the period of phase 2 to shorten, the slope of phase 3 to become steeper, and the duration of action potential to decrease, shortening the QT interval. The increased conduction velocity, involving more myocytes in a short time, will result in an image of an increased ST segment on the surface ECG. Even so, the increase in the ST segment due to local hyperkalemia is more often observed in conditions of coronary occlusion. Systemic hyperkalemia also has the possibility of similar imaging, such as ST-T segment elevation, tall T, and Q-T interval shortening, resulting in a pseudo-infarction for patients of acute hyperkalemia.[3,9]

The reduction of E_m in the diastolic phase actually shows an image of T-Q depression due to relative depolarization, but because the ECG machine records with filtration eliminating the downward baseline shift, the image is displayed by increasing the upward shift of the ST segment. Thus, almost half of the ST elevation imaging is actually the role of T-Q depression that cannot be visualized by machines.[3,9]

Potassium homeostasis is preserved by balancing extra-/intracellular potassium concentration. The plasma K^+ concentration is tightly adjusted between 3.5 and 4.5 mmol/L, with 95% of the total potassium in the body in intracellular space, and only 2% being extracellular. Total levels of potassium in the body are mostly regulated by the kidney; only 5-10% of the potassium is excreted through feces. Potassium excretion by the kidneys is very much determined by the rate of potassium filtration in the glomerulus basal membrane and the level of secretion and reabsorption at the distal nephron tubule.[10] The risk of hyperkalemia increases twofold for kidney disorders with eGFR <15 mL/minute. The reduction of potassium excretion by chronic kidney disorders may still be compensated by an increase of the excretion function from the intestines.[10,11]

Treatment for hyperkalemia may be divided into acute therapy and long-term therapy. The objective of acute therapy is to stabilize the cardiomyocyte membrane in preventing arrhythmia, to shift potassium into cells, and to increase the elimination of potassium from the body [12]. The goal of acute therapy of hyperkalemia is to prevent electrophysiologic effect on the heart to reduce the risk of arrhythmias. Insulin and beta-agonist therapy with the working mechanism of shifting potassium to intracellular space is only temporary in nature, and the same is also true for administration of Ca-gluconate that works to stabilize the myocardial membrane, which only has an effect for 30 minutes.

Adjunctive salbutamol nebulizers can be given to help shift potassium into cells.[12,13]

A high plasma creatinine serum illustrates a bad rate of glomerulus filtration, but if urine output is still present, the function of potassium excretion from distal tubules still occurs [10]. Patients who respond toward the administration of furosemide, as an inhibitor of Na+/K+/Cl- co-transporter, can excrete more potassium from the kidneys. Furosemide works on the cell walls of the intraluminal ascending limb of the loop of Henle if excreted by the proximal tubules, and thus its potential depends on the amount of luminal excretion [14]. However, if anuria has already occurred, hemodialysis becomes the final approach for the disposal of potassium from the body. The combination of low-dose furosemide (5 mg/hour) and low-dose dopamine (5 mcg/kg/minute) as continuous IV for kidney failure patients has a synergistic effect for the improvement of kidney function.[15]

Conclusion

Hyperkalemia has the potential to cause death, but it is easy to be treated. Hyperkalemia needs to receive urgent therapy when exceeding 6.5 mmol/L and changes occur in the ECG. Manifestations resembling acute STEMI and total AV block in ECG imaging are rare cases, but may be encountered. Hyperkalemia is often related to kidney dysfunction, and the right treatment may prevent fatal arrhythmia of the heart. The aggressiveness of treatment for hyperkalemia depends on absolute serum concentration and evidence of cardiotoxicity. Frequent reevaluation of serum potassium is essential to monitor response of treatment and rebound rise in serum potassium.

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