Doi: 10.17944/mkutfd.307015

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2017; 8(29): 56-59

OLGU SUNUMU/CASE REPORT

ELECTROCARDIOGRAPHIC MANIFESTATIONS OF SEVERE HYPERKALEMIA: A CASE REPORT

Ağır Hiperkaleminin Elektrokardiyografik Bulguları: Olgu Sunumu

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ABSTRACT

Hyperkalemia is a life-threatening metabolic emergency. Delay in the treatment of hyperkalemia may lead to life-threatening consequences, even death. In this article, we presented a case of severe hyperkalemia. A 65-year-old woman was admitted to Emergency room complaints of dizziness and weakness. She was on hemodialysis (HD) three times weekly. Her heart rate was 125 per minuteand blood pressure was 80/60 mmHg. A wide QRS-complex rhythm was seen in electrocardiography. She was diagnosed as hyperkalemia. Meanwhile she was waiting for HD; she was treated with sodium bicarbonate, regular insulin, and salbutamol nebulization. Within 40 minutes, HD was performed. After HD session, she fully recovered and discharged from the hospital. In conclusion, although the severity of hyperkalemia is not correlated with ECG findings, in the presence of ECG findings, it is crucial to be evaluated these findings by emergent physicians and treated in a timely manner for this potentially life-threatening condition.

Key words: Hyperkalemia, electrocardiography, emergent treatment.

ÖZET

Hiperkalemi hayatı tehdit eden metabolik acillerden biridir. Hiperkalemi tedavisindeki gecikme hayatı tehdit eden sonuçlara hatta ölüme bile neden olabilir. Bu yazıda, biz ağır hiperkalemili bir olguyu sunduk. 65 yaşında kadın hasta acil servise güçsüzlük ve baş dönmesi şikayetiyle başvurdu. Hasta haftada 3 gün hemodiyalize (HD) girmekteydi. Kalp hızı dakikada 125 ve kan basıncı 80/60 mmhg saptandı. Elektrokardiyografide (EKG) geniş QRS kompleksli ritim görüldü. Hastaya hiperkalemi tanısı konuldu. Hasta HD için beklerken, hastaya sodyum bikarbonat, kristalize insülin ve salbutamol nebul uygulandı. 40 dakika içinde, HD tedavisi uygulandı. HD seansından sonra, hasta tamamen iyileşti ve taburcu edildi. Sonuç olarak, hiperkalemi şiddeti mutlaka EKG bulguları ile korele olmamasına rağmen, EKG bulgularının varlığında, acil serviste çalışan hekimler tarafından bu yaşamı tehdit eden durumun zamanında tanınıp, tedavi edilmesi kritik öneme sahiptir.

Anahtar kelimeler: Hiperkalemi, elektrokardiyografi, acil tedavi.

Gönderme tarihi / Received: 23.05.2016 **Kabul tarihi / Accepted:** 17.08.2016

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INTRODUCTION

Hyperkalemia is a life-threatening metabolic emergency that commonly occurs in patients with chronic kidney disease (CKD) and in patients taking certain medicines such as potassium-sparing diuretics and/or renin angiotensin aldosterone system blockers (1-3). Delay in the treatment of hyperkalemia in emergency rooms may lead to life-threatening consequences, even deaths. Thus, early diagnosis and treatment of hyperkalemia is critical. The ability of emergent physicians is vital to evaluate the electrocardiographic evidence of hyperkalemia. In this article, we presented a case of severe hyperkalemia due to CKD, and its electrocardiographic manifestations and emergent treatment.

CASE

A 65-year-old woman was admitted to Emergency Room complaints of nausea, vomiting, dizziness and weakness for 4 hours. She had a history of CKD and hypertension for 3 years and she undergone hemodialysis (HD) three times a week. She was on treatment with amlodipine 10 mg and prazocin 4 mg daily for hypertension. Her last HD session was 2 days ago for 4 hours. In her physical examination; she was in normal mental status, body temperature was 36.9°C, heart rate was 125 per minute, 3/6 pan systolic murmur was heard at the apex, respiratory rate was 22 per minute, and blood pressure was 80/60 mmHg. Fingertip oximeter showed saturation of 90%. Oxygen was given 4 liter per minute and wide QRS complexes were detected in cardiac monitoring. A wide QRS-complex rhythm without distinct atrial activity was seen in electrocardiography (ECG) (Figure 1). She was diagnosed as hyperkalemia due to CKD and was treated with 10 ml of 10% calcium gluconate solution at slow intravenous infusion. In blood gas analysis; her results were as follow; sodium 128 mEq/L, potassium 9.9 mEq/L and bicarbonate 11mEq/L. Emergent HD was planned, however, while she was waiting for HD, she was treated with 60 mEq of sodium bicarbonate, regular insulin (6 U/100 cc of 20% dextrose over 30 min), and 2.5 mg salbutamol nebulization. Pre-treatment laboratory results revealed as follow; Blood urea nitrogen 119 mg/dL, creatinine 7.7 mg/dL, and glucose 121 mg/dL. After 30 minutes she was re-evaluated; blood pressure was 95/70 mmHg, heart rate was 105 per minute, K level was decreased to 8.8 mEq/L and the ECG was repeated (Figure 2). Second ECG revealed narrowing QRS complex and increased atrial activity. In approximately 40 minutes, HD was performed. After HD session, she fully recovered and discharged from the hospital with the recommendation of HD treatment thrice weekly.

Figure 1. The ECG showed a markedly wide-complex irregular ventricular rhythm without regular distinct atrial activity—the sinoventricular rhythm. K: 9.9 mEq/L.

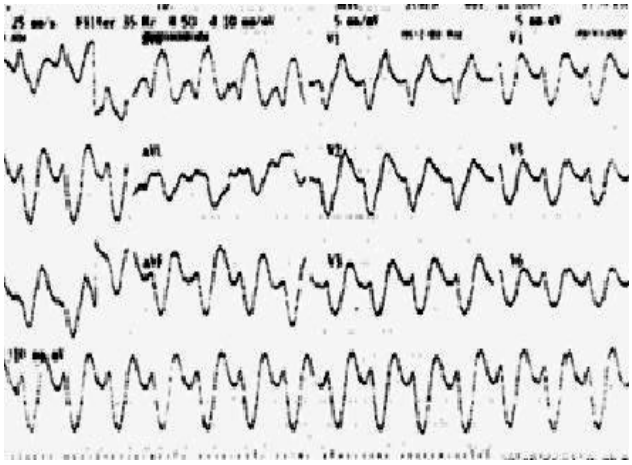
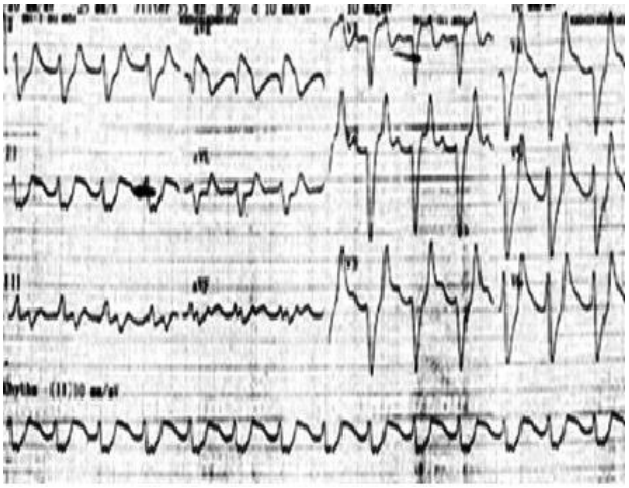


Figure 2. The ECG showing a wide QRS complex and rhythm at a rate of 105 beats per min.



No evidence of atrial activity was seen. Furthermore, the QRS-complex, particularly in the inferior leads, has assumed a sine wave configuration, consistent with pronounced hyperkalemia and the sinoventricular rhythm. K: 8.8 mEq/l.

DISCUSSION

Most serious clinical signs of hyperkalemia are cardiac symptoms and ECG changes associated with hyperkalemia are well documented. The electrocardiographic manifestations of hyperkalemia include the peaked T waves, progressive prolongation of the PR intervals and widened QRS complexes, and decreased amplitude and/or eventual loss of the P waves followed by ventricular fibrillation or asystole (4). An ECG is not always a reliable indicator for the severity of hyperkalemia because hyperkalemia may occur without significant ECG changes (5, 6). However, it would be necessary to start treatment for hyperkalemia if ECG changes were detected. Strict cardiac monitoring, correction and monitoring of electrolyte disturbance are required. Treatment of symptomatic patient includes three phases; firstly, membrane stabilization of cardiac tissue, secondly, intracellular shift of potassium, thirdly, removal or excretion of potassium from the body. Hemodialysis is the definitive management of severe hyperkalemia (7). In our case, we started to treatment based on the ECG findings and blood gas analysis. In the treatment of hyperkalemia, it is essential to start treatment earlier, because waiting for the results of biochemical test to begin the treatment may cause fatal complication. We treated our patient by medical and HD without any complication.

Cardiac conduction between myocytes may be suppressed due to higher levels of serum potassium. P wave and PR interval prolongation may be seen before the QRS interval prolongation. These changes generally occur when potassium levels exceed 6.5 mEq/L (8). When serum levels reached to two times above normal, sinoatrial and atrioventricular conduction was suppressed resulting in sinoatrial and atrioventricular blocks and escape beats. Intraventricular conduction blocks including delay, bundle branch block, and fascicular have been reported. This finding will be a pre-terminal event unless treatment is initiated immediately. The fatal event can be either asystole, as there is a complete block in ventricular conduction, or ventricular fibrillation (8). Although the above ECG progression is descriptive of the classic presentation of hyperkalemia, but metabolic alterations such as alkalosis, hypernatremia, or hypercalcemia can antagonize the trans membrane effects of hyperkalemia and result

in the blunting of the ECG changes associated with elevated potassium levels (8).

Treatment of life-threatening hyperkalemia focuses on blocking the effects on myocyte trans membrane potential and cardiac conduction with calcium infusion as well as decreasing extracellular potassium levels: sodium bicarbonate, magnesium, beta-2 adrenergic agonists, and the combination of glucose and insulin all drive potassium intracellular and lower the extracellular serum potassium level. Response to the treatment is often prompt with visualization noted on the ECG or electrocardiographic monitor. Finally, excess body potassium can be removed with the use of sodium polystyrene sulfonate or HD. In our case we used all of these treatment alternatives. Fortunately, we were able to treat our patient without any complications.

In conclusion, although the severity of hyperkalemia is not necessarily correlated with ECG findings and determining of serum potassium level takes time, in the presence of ECG findings, it is crucial to be evaluated these findings by emergent physicians accurately and treated in a timely manner for this potentially life-threatening condition.

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