Severe Hyperkalemia without Electrocardiographic Changes

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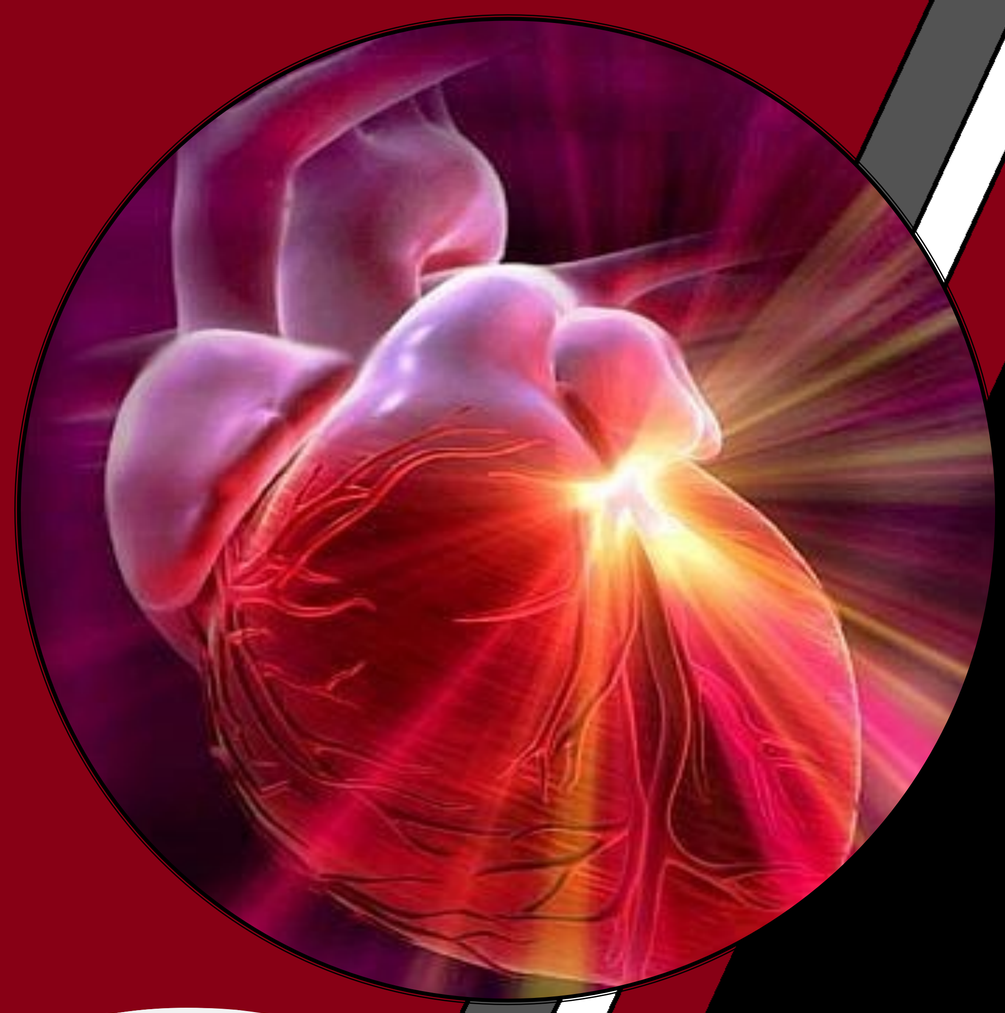
Introduction

Potassium plays an important role in maintaining the electrical potential across the cellular membrane,

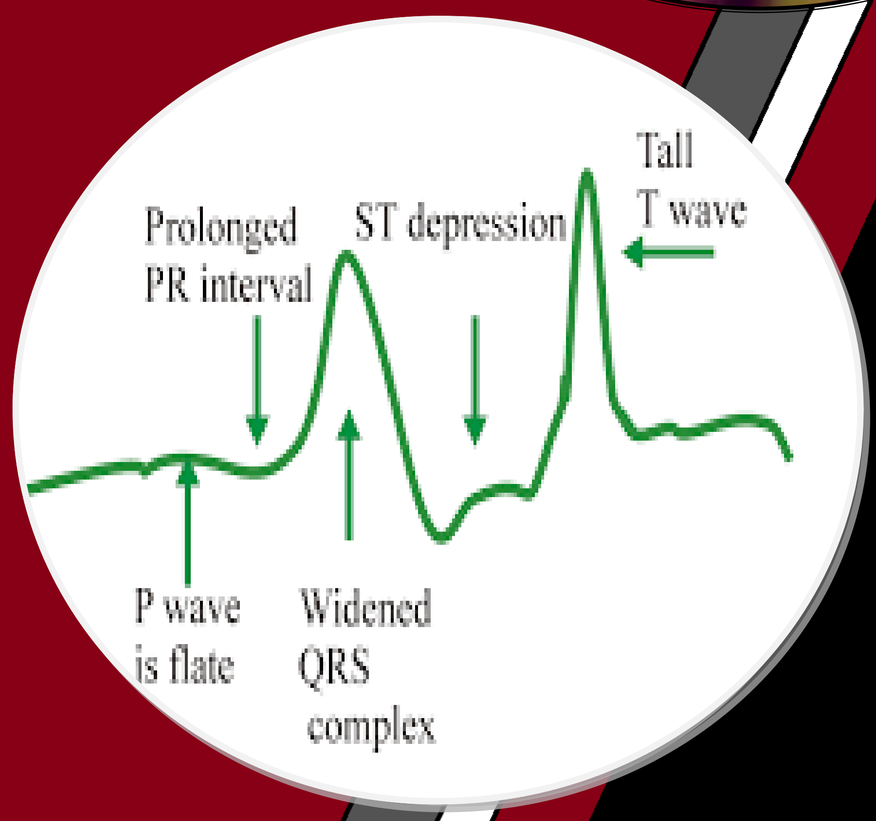
as well as in depolarization and repolarization of the myocytes.These electrophysiological effects are reflected as some predictable ECG manifestations(1, 2).

The recognition of severe hyperkalemia's electrocardiographic manifestations is very important. We describe here a case of CRF patient with minimal ECG changes(3).



Case Report

A 73-year-old man was admitted to the emergency service with generalized pain, bad health, nausea and vertigo; with a week history of mild chest pain. His medical history included chronic renal failure (from 8 years ago) and hypertension (from 20 years ago) and type 2 diabetes mellitus. Usual medications at the time of admission included: aspirin, triamterene, amlodipine and atorvastatin. The patient's history of smoking was 45 pack/ year. His physical examination was notable for mild weakness and decreased sensitivity in the calf. Vital signs showed a blood pressure of 190/140 mmHg and arterial blood gas (ABG) analysis revealed PH 7.25 that indicates acidosis. The admission ECG revealed heart rate 80 /min ,ST elevation in inferior leads (I ,II ,avF) and ST depression in avL.(Fig.1) .The patient hospitalized in CCU unit with diagnosis of acute coronary syndrome (ACS) and got treatment with anti-anginas and anti-platelets. His renal and urinary tract's sonography showed normal kidney dimensions and no sign of hydronephrosis was shown. The serum potassium was 9 mEq/L and other investigations revealed hemoglobin mg/dl, sodium 135 mEq/L, calcium 9.1 mg/dl, keratinize 2.3 mg/dl, urea 84 mg/dl, and troponin 467 ng/ml. Considering pseudohyperkalemia; the serum potassium was rechecked. Repeated serum potassium confirmed the same level. Therefore he immediately treated with calcium gluconate, k. oxalate and sorbitol with the diagnosis of hyperkalemia and high potassium diet was limited for him. His serum potassium level gradually returned to normal level. Finally, with hospital treatments, the patient was discharged from hospital with creatinine 1.5 mg/dl and stable potassium level of 3.5 mmol/L. The important point in this case is normal ECG in the presence of severe hyperkalemia. (No defused tall and tent T wave was seen.)



Discussion

Potassium is the major intracellular cation (150mmol/L), the extracellular concentration being about 4mmol/L. This creates a large concentration gradient across the cell membrane which is important for maintaining the resting membrane potential. The most prominent effects of hyperkalemia are on the myocardium. Decrease in resting membrane potential decreases myocardial cell conduction velocity and increases the rate of depolarization(4) Hyperkalemia is a common and potentially life threating electrolyte disorder in the hospital setting. (5).Among main causes of hyperkalemia are increased intake and impaired excretion, renal impairment aldosterone deficiency, drug action on renal excretion (Heparin, NSAID agents, etc.) alteration in intra/extra cellular potassium distribution (acidosis, severe insulin deficiency (diabetic ketosis), cell lysis, etc.)(3, 6).Errors in potassium measurement can cause Pseudo Hyperkalemia ,where serum potassium is falsely elevated .Usually ,these are recognized either by the laboratory or the clinicians(7). In experimental settings, hyperkalemia has been associated with a defined series of electrocardiogram (ECG) abnormalities(8).Peaked T waves in the precordial leads are among the most common and the most frequently recognized findings on ECG.A tall peaked T wave with shortened QT interval is the first change seen on the ECG in a patient with hyperkalemia. This is followed by progressive prolongation of the PR interval and QRS duration. The P wave may disappear, and ultimately the QRS widens further to a "sine wave". Ventricular fibrillation or standstill is the most severe consequences. Metabolic alterations such as alkalosis, hypernatremia, or hyperkalemia can antagonize the Trans membrane effects of hyperkalemia and result in unremarkable ECG changes .

Conclusion

Our case confirms that diagnostic ECG changes do not always accompany severe hyperkalemia. Therefore, clinicians should be careful that ECG may look normal in the presence of severe hyperkalemia.

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