Case Report

Electrographic Alterations Induced by Hyperkalaemia Simulating Acute Myocardial Infarction

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Abstract. In general, severe hyperkalaemia produces classic electrocardiographic manifestations including tenting of T waves, widening of the QRS complex, loss of P waves, and eventually, sine waves and asystole.

This report concerns a patient with chronic renal failure on maintenance haemodialysis who developed a severe hyperkalaemia associated with chest pain, manifested electrocardiographically by elevation of the S-T segment resembling acute myocardial infarction. After haemodialysis, serum potassium decreased and the electrocardiogram returned to normal. We review the literature and discuss the possible physiology of this electrocardiographic alteration.

Key words: Hyperkalaemia; Haemodialysis

Introduction

Potassium represents the major intracellular cation, and membrane polarization is critically dependent on the relationship between extracellular and intracellular potassium concentrations [1–3]. Relatively small changes in the concentration of extracellular potassium may be associated with extensive morbidity and death [4–6]. The kidney plays a major role in the regulation of potassium balance [7,8].

Acute hyperkalaemia has been reported in a variety of clinical situations, underlying which is an inability to excrete urinary potassium because of renal failure and/or adrenal hormonal deficiency [7–10]. Elevations of extracellular potassium concentration decrease the transmembrane potential and reduce the duration of the cardiac action potential [11–14]. These changes cause the classic electrocardiographic manifestations of hyperkalaemia, including tenting of T waves, widening of the QRS complex, loss of P waves, and eventually, sine waves and asystole [15–17].

In a few exceptional instances of severe potassium intoxication, elevation rather than depression of the RS-T segments may be produced [18–20]. The resultant 'currents of injury' may seem much more suggestive of acute myocardial infarction or pericarditis than extracellular potassium elevation. The present report describes a patient on haemodialysis, who developed a severe hyperkalaemia with electrocardiographic events suggesting acute myocardial infarction. Treatment with haemodialysis was followed by a return of both serum potassium and the electrocardiogram to normal.

Case Report

The patient was a 51-year-old woman with chronic renal failure related to bilateral cortical necrosis secondary to streptococcal tonsilitis; she had been on maintenance haemodialysis for the last 3 years. Her medical background included moderate arterial hypertension 10 years ago, uraemic pericarditis 2 years ago, and atypical thoracic pain during the past year. Medications consisted of aluminium hydroxide and diltiazem.

She was admitted to our hospital in December 1986 suffering from severe chest pain at rest, accompanied by sweating and vomiting for at least 20 min. Physical examination was normal, with blood pressure 150/85 mmHg,
Fig. 1. Electrocardiogram obtained on admission when the serum potassium was 8.2 mEq/l. Note the typical alterations of hyperkalaemia (peaking of T waves, widening of QRS), in V1 and V2 the elevation of S-T segment, and in leads 3 and aVF a QS complex.

and heart rate 60 b.p.m. Laboratory analysis revealed serum sodium 136 mEq/l, potassium 8.2 mEq/l, creatinine 12 mg/dl, CK 54 U/l (normal), glucose 145 mg/dl, haematocrit 28%, normal WBC, and moderate metabolic acidosis. In addition to a left anterior fascicular block, QRS widening, and peaking of the T waves, the ECG on admission showed the ST segments in V1 to V2 to be considerably elevated and a QS complex in leads 3 and aVF (Fig. 1). The patient’s lessened pain after sublingual nitroglycerin, and morphine e.v., but ECG abnormalities did not change. Because of the severe hyperkalaemia and the patient’s stability, haemodialysis was commenced 4 h after admission. At the end of the dialysis session (5 h) serum potassium was 4.8 mEq/l and the ECG had returned entirely to normal (Fig. 2).

Afterwards, treatment with diltiazem, nifedipine and atenolol was continued. Acute infarct scintigraphy with $^{99m}$Tc-PYP was performed 24 h later, and was negative. A cardiac catheterisation showed normal coronary arteries with preserved myocardial function, and an ergonovine test proved negative. An upper gastrointestinal X-ray showed a hiatus hernia with the presence of gastro-oesophageal reflux.

Eighteen months later the patient was healthy, in treatment with antacids, metoclopramide and ranitidine, and free of chest pain. The ECG was completely normal.

Discussion

The cardiototoxic effects of hyperkalaemia were first described by Hering in 1907, in a patient with cardiac arrest [21]. Since then numerous reports have indicated that gradually increasing plasma potassium induces a biphasic, more-or-less reproducible electrical sequence of initial increase, then profound decrease in impulse formation and conduction in all cardiac tissues [15–17]. Human
and animal studies have generally showed a relatively strong correlation between the serum potassium concentration and the ECG manifestations associated with hyperkalaemia [11,15,17]. On the other hand, a recent report has presented two patients with severe hyperkalaemia and without ECG abnormalities [22].

Peaking of the T wave is the earliest electrocardiographic manifestation of hyperkalaemia, usually before the ECG shows any measurable alteration of the QRS complex. If extracellular potassium continues to increase, intraventricular conduction slows and the duration of the QRS complex increases, widening the QRS complex to resemble the typical pattern of left bundle branch block. When plasma potassium exceeds 7 mEq/l, the P-wave amplitude decreases and its duration increases because of the slower conduction in the atria; it then tends to disappear. Subsequently a typical ECG of severe hyperkalaemia is seen: peaking of T waves, widening of the QRS complex, and disappearance of the P wave [11,15–17,24].

Electrocardiographic patterns simulating acute myocardial infarction (ST segment elevation) have occasionally been encountered in potassium intoxication and in severe hyperkalaemia accompanying renal failure [18–20], as in the case of our patient. Such ST segment elevation is secondary to the increase in the QRS duration, because the 'injury pattern' is rapidly reversible when the QRS duration decreases during treatment with dialysis. The ST segment elevation is due to an electrical potential gradient between normal/negative cells and those injured or depolarised by potassium. An epicardial electrode over the injured area records ST elevation, or current injury pattern.

Whatever the exact mechanism, when electrocardiographic elevations of ST segments are present in haemodialysed patients, hyperkalaemia must be ruled out. ST segment elevations must also be included in the electrocardiographic alterations induced by hyperkalaemia.

References
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Accepted in revised form 17.11.88