Case Report

Life threatening hyperkalemia requiring prolonged temporary pacing support: A case report and an analysis of electrocardiographic changes

Chakraborty B, Rahman M, Ahmed QS, Rahman MM

The ORION Medical Journal 2006 Sep;25:411-413

Abstract
Hyperkalemia is a life threatening metabolic condition that can induce deadly cardiac arrhythmias. Here we present a case of severe hyperkalemia in a 46-year-old woman with pre-existing mild renal impairment who presented with severe fatigue, drowsiness and hypotension. 12-lead electrocardiogram (ECG) was suggestive of junctional rhythm with marked bradycardia. Her initial serum potassium level was 8.5 mmol/L. She was commenced on standard treatment for hyperkalemia but her bradycardia and hypotension continued unabated. Temporary endovascular pacing was considered essential for restoration of normal heart rate and to combat hypotension. Her condition improved dramatically after emergency cardiac pacing. Heart rate and blood pressure normalized immediately after pacing. She remained pacemaker dependent for two days. On the third day of admission her own sinus rhythm returned back and she was discharged from the hospital on the 5th day in a haemodynamically stable state.

Introduction
Hyperkalemia is an acute life-threatening clinical condition seen in the emergency department. It results from electrolyte imbalance and commonly occurs in patients with known chronic renal insufficiency or end stage renal disease. The other causes include drug induced such as angiotensin-converting enzyme inhibitors or use of potassium-sparing diuretics, insulin deficiency or resistance and haemolysis. Hyperkalemia is usually asymptomatic until severe when the patient may complain of paraesthesias or weakness, progressing in extreme cases to a flaccid paralysis. Death by asystole or ventricular fibrillation may be the first clinical presentation. The ECG is the single most important clue that the patient is at risk of cardiac arrest.

The final ECG changes in hyperkalemia include bradycardia leading to asystole or a sine wave pattern in which the widened QRS complex merges with the T wave or ventricular fibrillation. Ventricular tachycardia can occur in hyperkalemia but is more often associated with hypokalemia. Pulseless electrical activity has also been reported.

Here we present a case of life threatening hyperkalemia who presented with severe bradyarrhythmia and shock. The management guidelines and ECG changes associated with hyperkalemia are also discussed.

Case report
A 46-year-old lady was admitted in a general hospital with a history of increasing lethargy and drowsiness for two days. She was known to have diabetes mellitus with mild renal impairment for two years. She was on oral hyoglycaemic agent and angiotensin converting enzyme inhibitor, enalapril 10 mg once a day. On admission she had profound bradycardia, pulse rate 32/ min and blood pressure was 70/42 mm of Hg. On admission her serum potassium level was 8.5 mmol/L and her serum creatinine was 1.7 mg/dl.
Case Report

Serum sodium and chloride level were within normal limits. She was commenced on intravenous fluid, injection calcium gluconate and glucose/insulin infusion. Within two hours of treatment her potassium level decreased by 1.5 mmol/L but her haemodynamic status remained unchanged. So she was referred to a tertiary centre for further management. On admission in our hospital she had severe generalized muscle weakness along with cold extremities. Clinically she was profoundly bradycardic at 35 beats per minute with systolic blood pressure 60 mm of Hg. Electrocardiogram showed junctional rhythm with occasional atrial ectopics (Fig-1).

![Figure 1: ECG on admission showing Junctional rhythm with profound bradycardia before temporary pacing](image)

As she was haemodynamically unstable, a temporary pacemaker was immediately installed (Fig-II).

![Figure 2: ECG after temporary pacing](image)

Her haemodynamic status improved immediately after temporary pacing. Her alertness improved and blood pressure normalized. We continued her treatment with glucose/insulin and oral cation exchange resins along with pacing. Her potassium level recorded 5.1 mmol/L after six hours of commencement of treatment. But her arrhythmia remained unabated and we failed to take off pacing support for two days. On third day of admission she reverted back to her normal sinus rhythm (Fig-III) and the pacing wire was taken off. Afterwards she had an uneventful recovery and was discharged from the hospital on fifth day in a stable state.

![Figure 3: ECG after restoration of patient's own normal sinus rhythm](image)

Discussion

Hyperkalemia is a life threatening metabolic condition and is defined as serum potassium level of greater than 5.5 mmol/L\(^8\). The common risk factors for hyperkalemia include renal insufficiency, use of angiotensin converting enzyme inhibitors (ACEI), angiotensin receptor blockers (ARB), potassium sparing diuretics, potassium supplementation and excessive consumption of potassium containing diets\(^8\)-\(^10\). The patient we described here had this life threatening condition as she was on ACEI on the top of mild renal impairment. Hyperkalemia in this particular patient occurred partly because of ACEI and partly as a result of pre-existing mild renal impairment. ACEI or ARBs are considered as the standard regimen for patients with renal disease\(^4\)-\(^6\). However there have been many reports that administration of ACEI or ARBs may cause a higher risk of development of hyperkalemia in patients with renal impairment\(^11\),\(^12\).

The patient was commenced on intravenous calcium gluconate and intravenous glucose/insulin immediately after admission.
Case Report

Intravenous calcium, insulin with dextrose infusion, intravenous sodium bicarbonate, nebulised salbutamol and cation exchange resins are the standard treatment of hyperkalemia\(^1\)^2. Resistant hyperkalemia may require emergency dialysis\(^3\)^5. Temporary pacing is a life saving procedure with or without dialysis\(^7\)^9. In this particular patient hyperkalemia was quickly normalized by medication with calcium gluconate and glucose/insulin infusion but her bradyarrhythmia persisted. So we kept the pacing wire in situ for two days for patient’s safety.

Our patient presented with ECG features of junctional rhythm. Junctional rhythm is an indicator of life threatening hyperkalemia. As the hyperkalemia worsens and the potassium level approaches to reach 10 mmol/L, sinoatrial conduction no longer occurs and passive junctional pacemakers take over the electrical stimulation of the myocardium. If hyperkalemia continues unabated, the QRS complex continues to widen and eventually blends with the T wave, producing the classic sine-wave electrocardiogram. Once this occurs, ventricular fibrillation and asystole are imminent\(^6\).

Although laboratory tests are the gold standard in the diagnosing changes in the serum electrolyte concentration, delays may be experienced in obtaining results. Hence, in many cases, early diagnosis and empiric treatment of hyperkalemia is dependent on the physician’s ability to recognize the electrocardiographical manifestations of hyperkalemia. The most common electrocardiographical changes associated with hyperkalemia include the earliest manifestation of tall tented or peaked T waves with narrow base, reduction in the amplitude and eventual loss of P waves. This is followed by bizarre widening of the QRS interval, then idioventricular rhythm with the widened QRS merging with the T wave to form a "sine wave" which may culminate in ventricular fibrillation\(^1\)^2. Despite this, the classic ECG changes do not always manifest and the relationship between serum potassium concentration and ECG changes varies among people. Hence the ECG alone is not reliable for mild to moderate hyperkalemia. Minimal changes on the ECG may be seen in severe hyperkalemia. Early stages of hyperkalemia may manifest with only shortening of the PR and QT interval\(^2\). Complete heart block with widened QRS complex has already been reported\(^6\)^13\)^14. Many less known ECG changes associated with hyperkalemia have been reported. These include hemiblock due to depressed supraventricular conduction, left or right bundle branch block, bifascicular as well as trifascicular blocks\(^1\)^15. Of importance is the fact that hyperkalemia can produce ECG changes, such as ST segment elevation that mimic acute myocardial infarction\(^16\). ST segment depression and T wave inversion which similarly occur in cardiac ischaemia have also been reported. Anteroseptal and Inferior wall pseudoinfarction pattern due to hyperkalemia have been reported in literature\(^17\).

<table>
<thead>
<tr>
<th>ECG manifestations of hyperkalemia relative to serum potassium level</th>
<th>Serum potassium level</th>
<th>expected ECG abnormality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild hyperkalemia (5.5 - 6.5 mmol/L)</td>
<td>Tall peaked T waves with narrow base</td>
<td></td>
</tr>
<tr>
<td>Moderate hyperkalemia (6.5 - 8.0 mmol/L)</td>
<td>Peaked T waves</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Prolonged PR interval</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Decreased amplitude of P waves</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Widening of QRS complex.</td>
<td></td>
</tr>
<tr>
<td>Severe hyperkalemia (&gt;8.0 mmol/L)</td>
<td>Absence of P wave</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Progressive widening of QRS-complex</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Eventual &quot;Sine-wave&quot; pattern</td>
<td></td>
</tr>
<tr>
<td></td>
<td>VF</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Asystole</td>
<td></td>
</tr>
</tbody>
</table>


ECG changes usually resolve after correction of hyperkalemia but arrhythmia may remain unabated for some time even after normalization of serum potassium level in some cases. In our patient the bradyarrhythmia remained unabated even after correction of potassium level. A mild to moderate increase in serum potassium causes an increase in myocardial excitability but further increase leads to impaired myocardial responsiveness, including that to pacing stimulation\(^9\). In general, hyperkalemia
produces a gradual depression of the excitability, conduction velocity of the specialized pacemaker cells and conducting tissues throughout the heart. High serum potassium levels are also thought to impair the conduction in the purkinje fibers and ventricles. Excitability of the specialized pacemaker cells may remain depressed quite some time even after correction of hyperkalemia. It actually happened in our case and bradyarrhythmia persisted for 48 hours even after normalization of serum potassium level.

Conclusion
The diagnosis of hyperkalemia must be considered in any patient with clinical risk factors that would predispose them to its development. Most commonly, patients with hyperkalemia have underlying renal impairment or are taking ACEI or ARB to increase serum potassium level. Life threatening hyperkalemia may occur in patients who are taking ACEI or ARB in presence of renal dysfunction. The treatment of hyperkalemia must be swift and appropriate to prevent the development of fatal cardiac arrhythmias. Temporary pacing may be life saving in presence of profound bradyarrhythmia with haemodynamic instability.

References