



Electrocardiographical case. A tale of tall T's

H C Chew, S H Lim

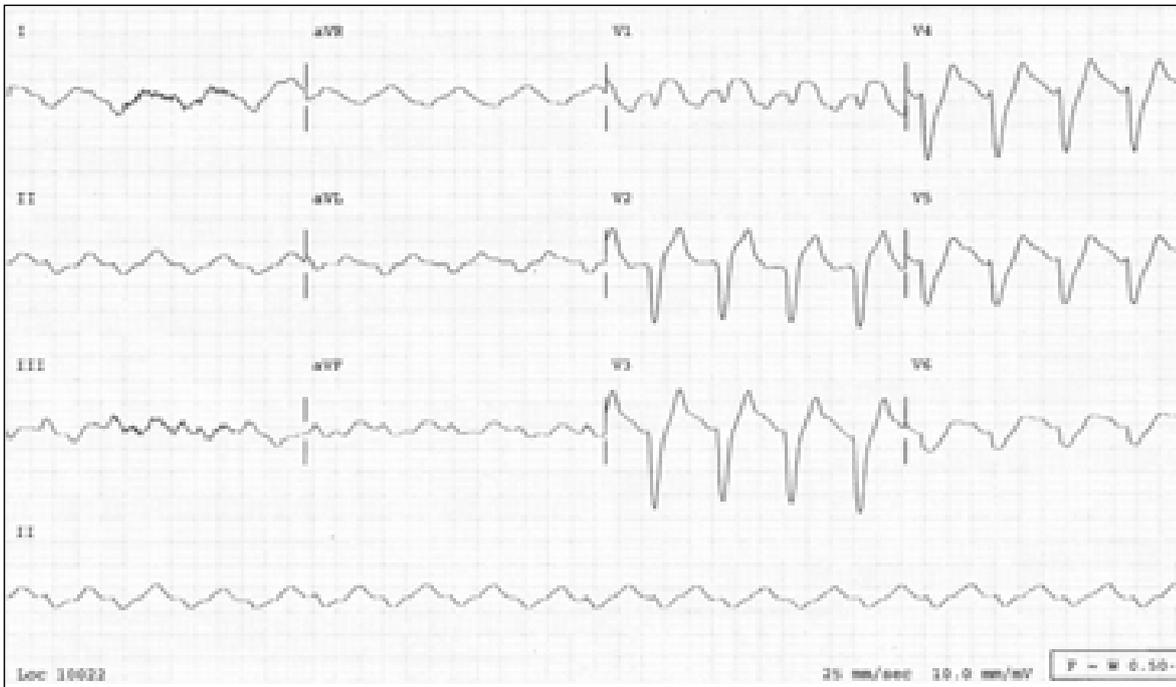


Fig. 1 Initial 12-lead ECG.

CLINICAL PRESENTATION

A 63-year-old woman presented at the emergency department (ED) with a history of increasing lethargy and drowsiness. She had underlying type II diabetes mellitus. A 12-lead electrocardiogram (ECG) was performed (Fig. 1). What is the diagnosis?

Department of
Emergency
Medicine
Singapore General
Hospital
Outram Road
Singapore 169608

H C Chew, MMed,
MRCSE, MRCP
Registrar

S H Lim, MBBS,
FRCSE, FAMS
Senior Consultant
and Head

Correspondence to:
Dr Chew Huck Chin
Tel: (65) 9856 0027
Fax: (65) 6462 4178
Email: chinjing@
pacific.net.sg

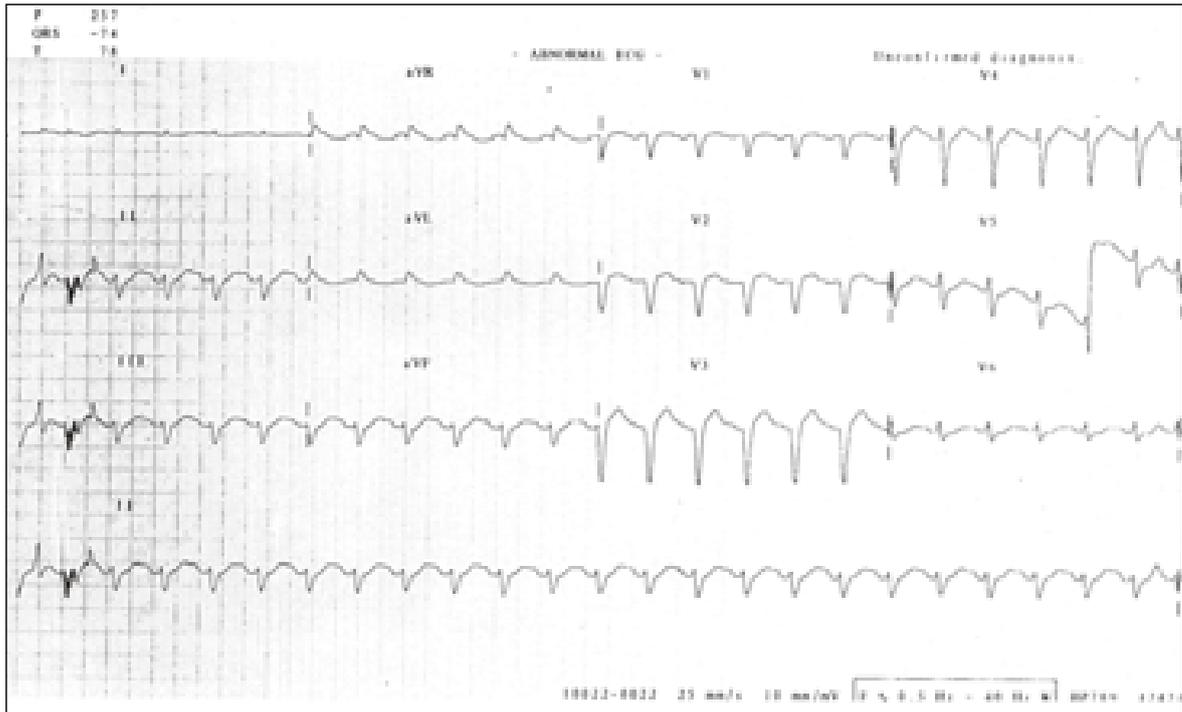


Fig. 2 Repeat 12-lead ECG done after intravenous calcium chloride and intravenous insulin with 50% dextrose solution were given.

ECG INTERPRETATION

ECG (Fig. 1) showed tall, peaked T waves (especially in the anterior leads V2 – V4), prolonged PR interval, small amplitude of P waves and broadening of the QRS complex. This was suggestive of hyperkalaemia.

DIAGNOSIS

Hyperkalaemia.

CLINICAL COURSE

She was given intravenous (IV) calcium chloride and units of IV insulin with 40ml of 50% dextrose. After this initial treatment, the repeat ECG (Fig. 2) showed flattening of the T waves with progressive decrease in the width of the QRS complexes. There was left axis deviation, suggestive of left anterior hemiblock. In addition, poor R wave progression with Q waves were seen in the anterior leads which may suggest a previous myocardial infarction.

The patient's potassium level was 7.1mmol/L. She was found to have a blood glucose level of 35mmol/L and an arterial blood gas done showed evidence of metabolic acidosis. She was diagnosed with diabetic ketoacidosis and hyperkalaemia, started on IV sodium bicarbonate and insulin infusion with aggressive intravenous hydration, and sent to the intensive care unit for further management.

DISCUSSION

Hyperkalaemia is a common acute life-threatening emergency seen in the ED. It results from electrolyte

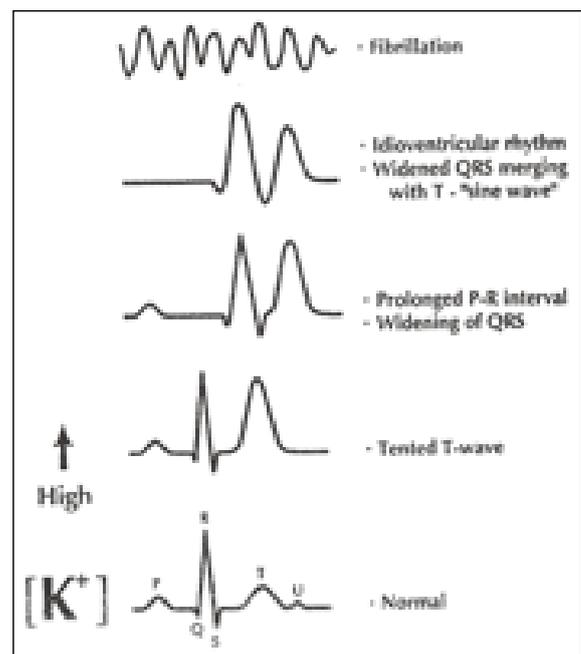


Fig. 3 Electrocardiographical manifestations of hyperkalaemia⁽¹⁾.

imbalance, and commonly occurs in patients with known chronic renal insufficiency or end-stage renal disease⁽¹⁾. The usual causes include renal insufficiency or failure, drug-induced such as angiotensin-converting enzyme inhibitors, or use of potassium-sparing diuretics, insulin deficiency or resistance, and haemolysis.

Although laboratory tests are the gold standard in diagnosing changes in the serum electrolyte concentration, delays may be experienced in obtaining

Table I. Electrocardiographical manifestations of serum hyperkalaemia relative to serum potassium level⁽²⁾.

Serum potassium level	Expected ECG abnormality
Mild hyperkalaemia 5.5 - 6.5 mmol/L	Tall peaked T waves with narrow base
Moderate hyperkalaemia 6.5 - 8.0 mmol/L	Peaked T waves Prolonged PR interval Decreased amplitude of P waves Widening of QRS complex
Severe hyperkalaemia > 8.0 mmol/L	Absence of P wave Progressive widening of QRS complex Eventual "sine-wave" pattern VF Asystole

results. Hence, in many cases, early diagnosis and empiric treatment of hyperkalaemia is dependant on the physician's ability to recognise the electrocardiographical manifestations of hyperkalaemia.

The most common electrocardiographical changes associated with hyperkalaemia include the earliest manifestation of tall tented or peaked T waves, 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 (Table I and Fig. 3).

These electrocardiographical changes are the result of the physiological effect of the electrolytes on the myocardial cells. Mild levels of elevated potassium result in the acceleration of terminal repolarisation, causing the T-wave changes that are the earliest manifestation on the ECG. Mild to moderate hyperkalaemia depresses conduction between cardiac myocytes and results in prolongation of PR and QRS intervals. P wave flattening reflects the sensitivity of atrial myocytes to hyperkalaemia. With increasing severity of hyperkalaemia, the sino-atrial and atrio-ventricular conduction is further suppressed, and results in the appearance of escape beats and escape rhythms^(3,4). The QRS complex will continue to widen and may blend with the T wave, creating a "sine-wave" appearance on the ECG. Further rises in the potassium level will result in ventricular fibrillation⁽³⁾.

Despite this, the classic ECG changes do not always manifest and the relationship between serum potassium concentration and ECG changes varies among people^(5,7). Hence, the ECG alone is not reliable for mild to moderate hyperkalaemia⁽⁶⁾. Minimal changes on ECG may be seen in severe hyperkalaemia⁽⁷⁾.

Many less known ECG changes associated with hyperkalaemia have been reported. These include complete heart block as a result of depressed atrioventricular conduction, hemiblock due to depressed

supraventricular conduction, left or right bundle branch block, bifascicular as well as trifascicular blocks^(3,8-11). In addition, the QRS complex axis may shift as a result of intraventricular conduction delay⁽¹²⁾.

Of importance is the fact that hyperkalaemia can produce ECG changes, such as ST segment elevation, that mimic acute myocardial infarction (Fig. 4)^(10,13-16). Generally, no Q wave development occurs however. ST segment depression and T wave inversion which similarly occur in cardiac ischaemia have also been observed^(13,17). These changes resolve after treatment of hyperkalaemia.

ABSTRACT

A 63-year-old woman presented at the emergency department (ED) with a history of increasing lethargy and drowsiness. The electrocardiogram (ECG) showed tall peaked T waves with broadening of the QRS interval, suggestive of hyperkalaemia. This patient had an elevated serum potassium level due to diabetic ketoacidosis. She was treated with intravenous calcium chloride and insulin with 50% dextrose. The ECG changes associated with hyperkalaemia are discussed, with illustrations from a second 48-year-old male patient with renal failure who presented with malaise, lethargy and generalised weakness.

Keywords: diabetic ketoacidosis, hyperkalaemia, tall T waves, widened QRS complex

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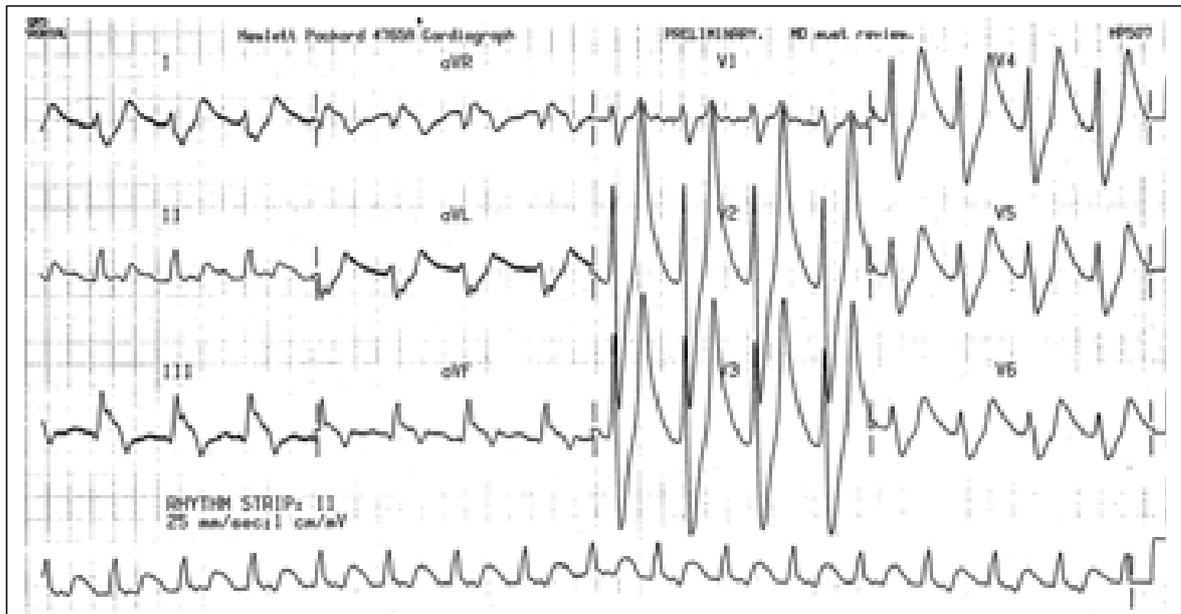


Fig. 4a 48-year-old man who presented with malaise, lethargy and generalised weakness. He had renal failure and had defaulted dialysis for the past 5 days. 12-lead ECG shows diffuse tall T waves with broadened QRS complexes, best seen in the precordial leads. In addition, the P wave amplitude is small with pseudo-ST elevation seen in leads II, III and aVF. His serum potassium was 8.2 mmol.

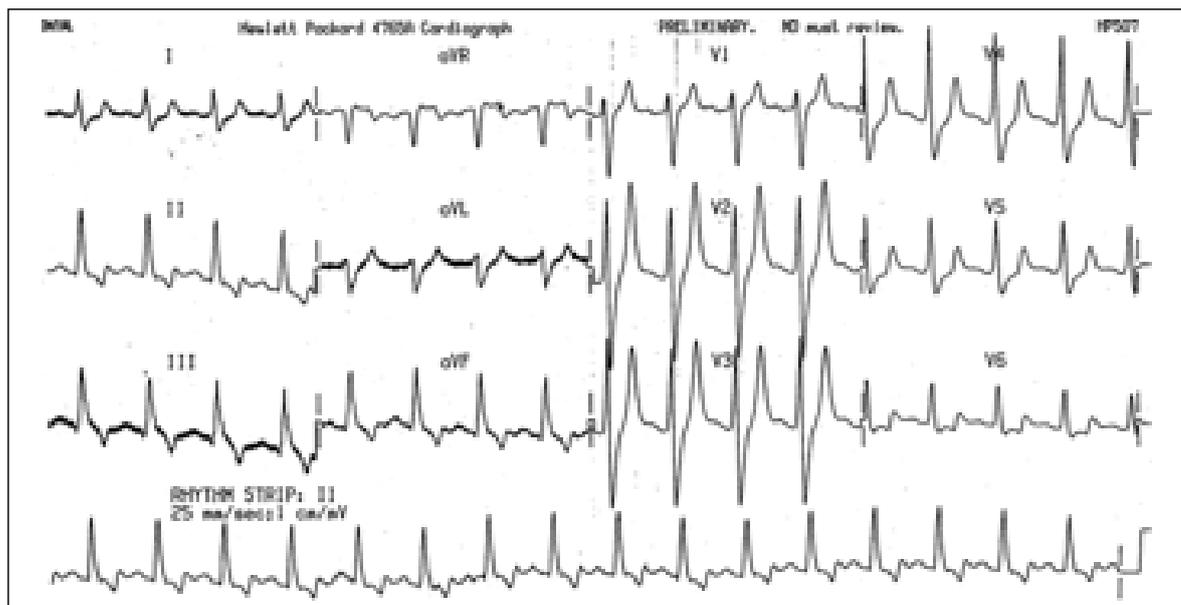


Fig. 4b Following diagnosis of hyperkalaemia, he was treated with IV calcium chloride and insulin with 50% dextrose. Repeat ECG shows that the QRS complexes have narrowed slightly, tall T waves are still present and the P waves have regained their prominence. This patient was subsequently admitted to the intensive care unit where he underwent dialysis.

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME

Multiple Choice Questions (Code SMJ 200508B)

	True	False
Question 1. ECG features of hyperkalaemia include:		
(a) Tall peaked T waves.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Prolonged PR interval.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Widening of QRS complex.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Absence of P waves.	<input type="checkbox"/>	<input type="checkbox"/>
Question 2. The common causes of hyperkalaemia include:		
(a) Renal insufficiency or failure.	<input type="checkbox"/>	<input type="checkbox"/>
(b) ACE (angiotensin-converting enzyme) inhibitors.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Potassium sparing diuretics.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Insulin deficiency or resistance.	<input type="checkbox"/>	<input type="checkbox"/>
Question 3. Other ECG changes associated with hyperkalaemia include:		
(a) Complete heart block.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Left or right bundle branch block.	<input type="checkbox"/>	<input type="checkbox"/>
(c) ST segment elevation.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Ventricular fibrillation.	<input type="checkbox"/>	<input type="checkbox"/>
Question 4. The following statements are true:		
(a) The earliest manifestation of hyperkalaemia on the ECG is tall peaked T waves.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Hyperkalaemia can cause ventricular fibrillation.	<input type="checkbox"/>	<input type="checkbox"/>
(c) T wave changes persist after treatment of hyperkalaemia.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Hyperkalaemia can produce ECG changes that mimic an acute myocardial infarct such as Q waves.	<input type="checkbox"/>	<input type="checkbox"/>
Question 5. Treatment of hyperkalaemia includes:		
(a) IV calcium chloride.	<input type="checkbox"/>	<input type="checkbox"/>
(b) IV sodium bicarbonate.	<input type="checkbox"/>	<input type="checkbox"/>
(c) IV insulin and 50% dextrose.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Oral resonium.	<input type="checkbox"/>	<input type="checkbox"/>

Doctor's particulars:

Name in full: _____

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Submission instructions:**A. Using this answer form**

1. Photocopy this answer form.
2. Indicate your responses by marking the "True" or "False" box
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1. Log on at the SMJ website: URL <<http://www.sma.org.sg/cme/smj>> and select the appropriate set of questions.
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1. Answers will be published in the SMJ October 2005 issue.
2. The MCR numbers of successful candidates will be posted online at <<http://www.sma.org.sg/cme/smj>> by 20 October 2005.
3. All online submissions will receive an automatic email acknowledgment.
4. Passing mark is 60%. No mark will be deducted for incorrect answers.
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