



# Electrocardiographical case. Young man with generalised myalgia

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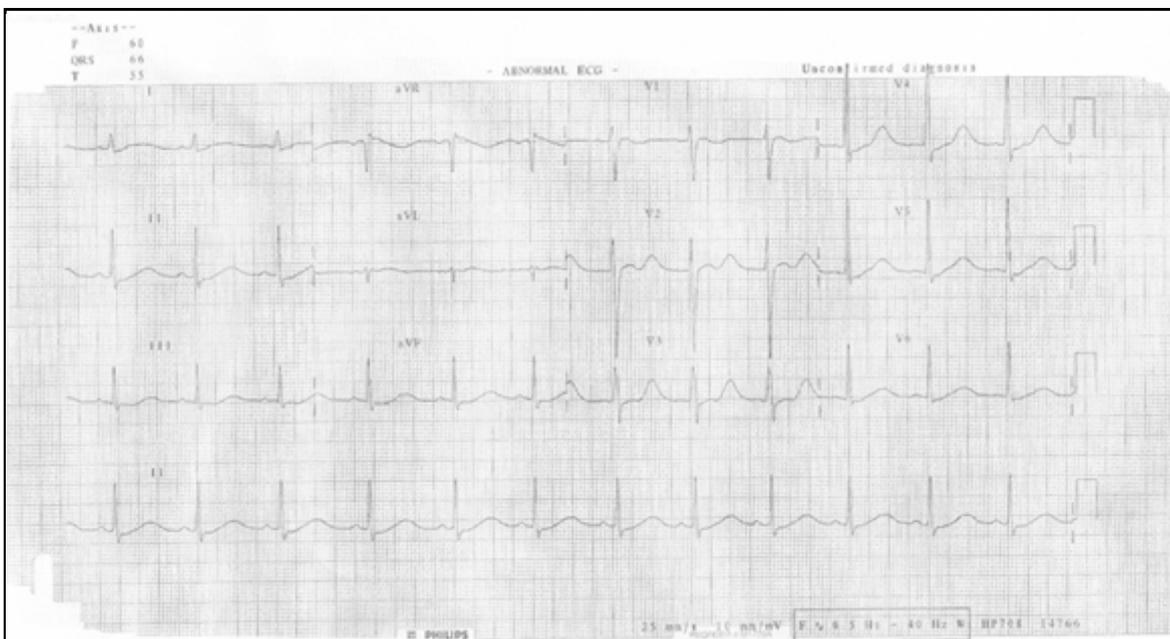


Fig. 1 12-lead ECG.

## CLINICAL PRESENTATION

A 24-year-old Myanmar man presented with generalised malaise and myalgia for three days. On the day of presentation, the patient got up and tried to walk, but fell on his face due to his weakness. He presented to the Emergency Department after

a fall at his workplace due to this weakness. He was previously well prior to this presentation. On clinical examination, he was found to have proximal muscle weakness of both the upper and lower limbs. There was no sensory loss. A 12-lead electrocardiogram (ECG) was done (Fig. 1). What was the diagnosis?

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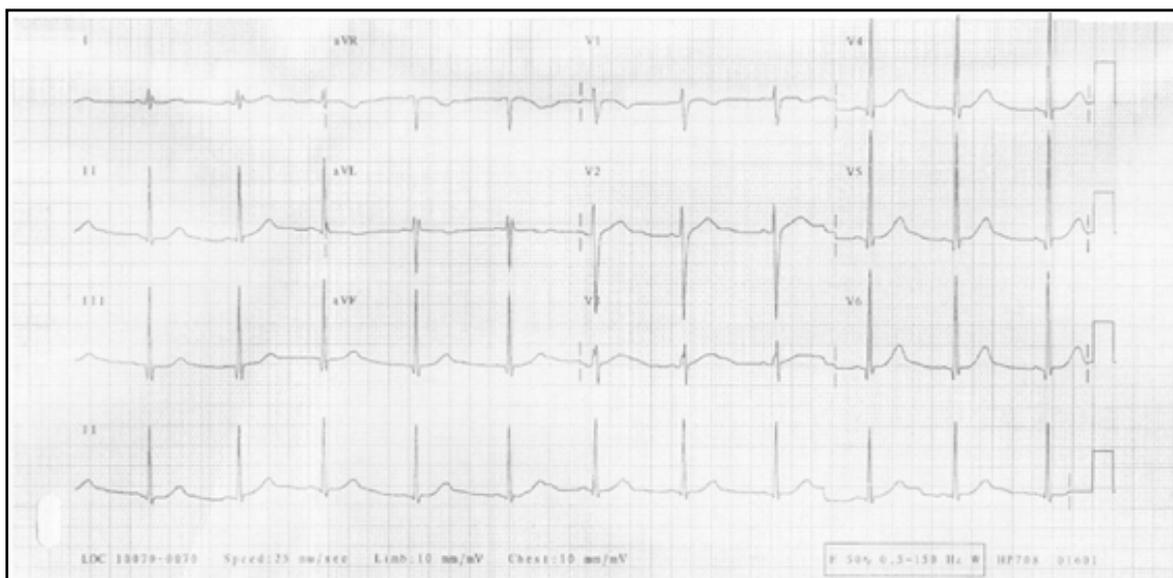
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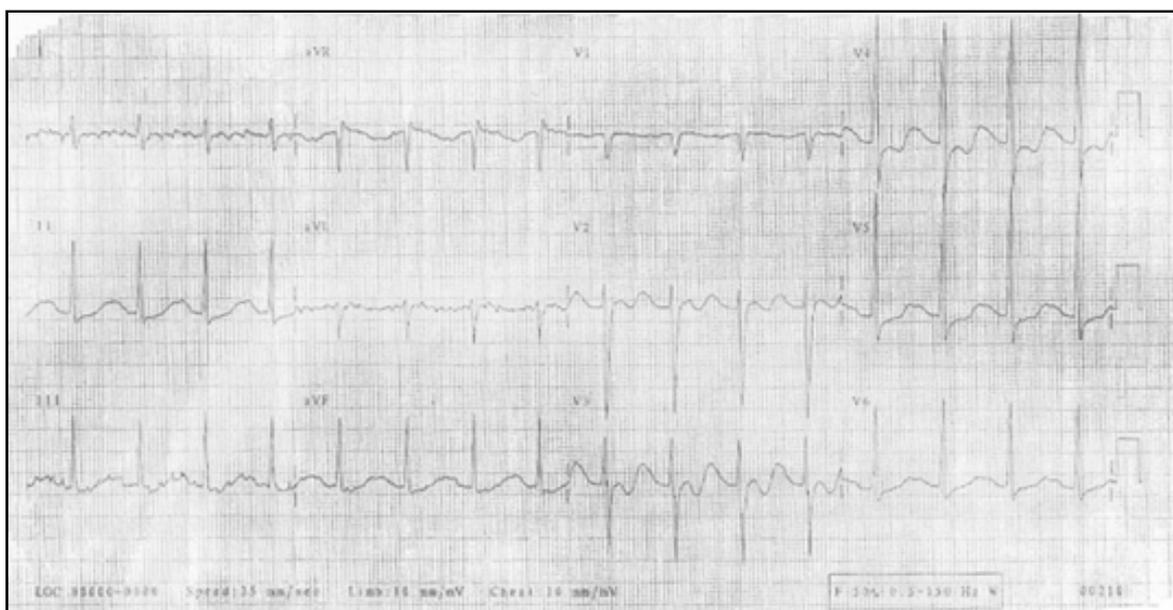
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**Fig. 2** Post-treatment ECG. Patient's potassium level was 3.0 mmol/L.



**Fig. 3** 25-year-old man with thyrotoxic periodic paralysis who presented with weakness on waking up in the morning. He had proximal muscle weakness of both the upper and lower limbs. He had hypokalaemia and thyroid function tests confirmed a diagnosis of thyrotoxicosis (TSH 0.001 mU/L, FT4 44.6 pmol/L). ECG shows normal sinus rhythm with ST depression in the leads V4 to V6, with a U wave (tallest in V3).

### ECG INTERPRETATION

The ECG (Fig. 1) shows a normal sinus rhythm. There appears to be ST depression in the leads V4 to V6, with the "T" assuming a more rounded appearance than usual. This is a U wave. The tallest U wave appears in V3 with amplitude of 5.0mm.

### DIAGNOSIS

Hypokalaemia

### CLINICAL COURSE

On further questioning, the patient admitted to having had ten episodes of watery diarrhoea for the past six days without any fever. There was no history of recent travel. There were no clinical features of

hyperthyroidism. He was found to have hypokalaemia of 1.6 mmol/L with no other sodium or chloride abnormalities. He had raised cardiac enzyme levels (creatinine kinase 954U/L, creatinine kinase-MB 6.8UG/L), but troponin-T level was normal. Aldolase level was raised at 36.9U/L. Thyroid function tests were normal.

The clinical impression was that of diarrhoea resulting in hypokalaemia and rhabdomyolysis. The patient was started on intravenous potassium replacement and his proximal weakness gradually improved. A repeat ECG (Fig. 2) was performed the next day with the patient having a potassium level of 3.0 mmol/L. The patient was discharged four days later with a potassium level of 4.6mmol/L.

## DISCUSSION

A progressive diminution in the serum potassium level below normal results in the following electrocardiographical manifestations<sup>(1,2)</sup>.

1. Progressive diminution with eventual disappearance of the T wave.
2. Progressive increase in the amplitude of the U wave.
3. First- and second-degree AV block of the Wenckebach type.
4. Depression of the ST segment.
5. Prolonged PR interval.
6. Rarely, tall and peaked U wave.

Changes in the ECG usually are not seen unless the potassium concentration falls below 3 mmol/L. However the changes are unpredictable and are most likely to occur in patients with both hypokalaemia and hypomagnesaemia<sup>(3)</sup>.

According to Schamroth<sup>(1)</sup>, the amplitude of the T wave decreases progressively and may eventually disappear completely, leaving only a "remnant" of the T wave visible as a minimal upward "bump" on the ST segment. The U wave also increases in magnitude, but still maintains its rounded appearance. This is seen in leads V2 and V3 (Fig. 1) and in leads V2, V3, V4 and V5 (Fig. 3). The increasing prominence of the U wave, especially when associated with a low or absent T wave may be mistaken for a T wave, leading to an incorrect diagnosis of a prolonged QT interval when in fact, it is the QU interval that is being monitored. Hence when QT is seen as very long, more than 0.44s, there is a good chance that there is a U wave merged with a T. This is seen in the limb leads, V4, V5 and V6 (Fig. 1), as well as in the limb leads, V2, V3, V4, V5 and V6 (Fig. 3).

Lepeschkin<sup>(4)</sup>, has stated that the normal U wave is tallest in lead V2 to V3 and the largest precordial U wave amplitude averages 11% of the largest T wave amplitude (ranging from 3% to 24% in 98% of cases). Occasionally, there may be electrical alternans of the enlarged U wave (alternation in the amplitude of the U wave)<sup>(4)</sup>. First degree AV block is common with low levels of serum potassium and this prolongation of the PR interval also means that the P wave encroaches progressively on the preceding U wave until it is inscribed immediately after the U wave. This may cause even more diagnostic confusion, since the enlarged U wave may be mistaken for a T wave and the ensuing P wave may be mistaken for the U wave.

Differential diagnosis may include hypomagnesaemia. The ECG changes include attenuation of the T wave, increased magnitude of the U wave, ST depression and prolongation of the PR interval<sup>(1)</sup>. There have been no definitive criteria to separate the two conditions. Other differentials include ischaemic heart disease<sup>(6)</sup>, the use of quinidine, digitalis or phenothiazines, bradycardia, and ventricular hypertrophy<sup>(7)</sup>.

Patients with mild hypokalaemia (serum potassium 3.0-3.5 mmol/L) usually have no symptoms. Both mild

and severe hypokalaemia can increase the incidence of cardiac arrhythmias<sup>(8)</sup>. Hypokalaemia leads to symptoms primarily involving the gastrointestinal tract, nervous system, skeletal muscle and heart by causing an impaired response to the parasympathetic stimulation and weakness of the gastrointestinal smooth muscles, resulting in decreased intestinal mobility. Neuromuscular symptoms occur typically when potassium concentrations fall below 2.5 mmol/L, resulting in muscle cramps, paraesthesia and generalised muscle weakness<sup>(3)</sup>. In addition, patients with severe hypokalaemia can develop muscle necrosis (rhabdomyolysis) and paralysis. Early morning weakness and paralysis with hypokalaemia should suggest thyrotoxic periodic paralysis (Fig. 3) to the emergency physician managing male Asian patients as this may be the first manifestation of thyrotoxicosis<sup>(9)</sup>.

In summary, the ECG is a very useful tool for the diagnosis of hypokalaemia. An accurate sign of hypokalaemia is a "camel-hump" – shaped U wave equal to or greater than the T wave in height<sup>(6)</sup>. Other features include disappearance of the T wave, depression of the ST segment, and prolonged PR interval.

## ABSTRACT

**A 24-year-old man presented with generalised malaise and myalgia for three days. He presented to the Emergency Department after a fall at his workplace due to weakness. 12-lead electrocardiogram (ECG) showed normal sinus rhythm with ST depression in the leads V4 to V6, with a U wave. The tallest U wave appeared in V3. These ECG features are characteristic of hypokalaemia. ECG changes in hypokalaemia and differential diagnosis are discussed. A second case of thyrotoxic periodic paralysis with similar ECG changes of hypokalaemia is also presented.**

**Keywords:** hypokalaemia, hypokalemic periodic paralysis

*Singapore Med J 2005; 46(1):38-41*

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## SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME

### Multiple Choice Questions (Code SMJ 200501B)

	True	False
<b>Question 1.</b> The following are electrocardiographical manifestations of decreased serum potassium level:		
(a) Disappearance of the T wave.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Shortened PR interval.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Second-degree AV block of the Wenckebach type.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Elevation of the ST segment.	<input type="checkbox"/>	<input type="checkbox"/>
<b>Question 2.</b> The following are differential diagnosis of patients with ECG changes of flattening of the T wave, increased magnitude of the U wave, ST depression and prolongation of the PR interval:		
(a) Hypomagnesaemia.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Wolff-Parkinson White Syndrome.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Hypokalaemia.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Hyperkalaemia.	<input type="checkbox"/>	<input type="checkbox"/>
<b>Question 3.</b> Causes of hypokalaemia include the following:		
(a) Chronic glucocorticoid administration.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Diarrhoea.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Insulin.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Decreased glomerular filtration rate (e.g. acute or end-stage chronic renal failure).	<input type="checkbox"/>	<input type="checkbox"/>
<b>Question 4.</b> Patients with hypokalaemic periodic paralyses may present with the following:		
(a) Slight transient weakness of an isolated muscle group.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Severe generalised weakness.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Severe attacks begin in the morning.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Precipitation by strenuous exercise or a high carbohydrate meal on the preceding day.	<input type="checkbox"/>	<input type="checkbox"/>
<b>Question 5.</b> Patients with severe hypokalaemia may present with the following signs:		
(a) Ileus.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Decreased muscle strength.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Increased tendon reflexes.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Sensory loss.	<input type="checkbox"/>	<input type="checkbox"/>

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3. Passing mark is 60%. No mark will be deducted for incorrect answers.
4. The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.