

## Hypokalaemia re-visited: a case series

### 重訪低鉀血症：個案系列

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Hypokalaemia is a relatively common biochemical abnormality found at the emergency department. Sequential changes in the electrocardiogram occur as the serum potassium is lowered. Correction of hypokalaemia can be life-saving. It is essential for physicians to be able to recognise these changes and initiate prompt replacement when necessary. We present a case series of patients presenting to the emergency department with symptomatic hypokalaemia and discuss the electrocardiographic manifestations and recognition of hypokalaemic changes. (*Hong Kong j.emerg.med.* 2005;12:232-236)

低鉀血症是急症室比較常見的生化異常情況。心電圖會隨著血鉀降低而出現連續的變化。糾正血鉀過低情況可以救活病者，能夠認出這些變化及在需要時迅速實施補充對醫生而言是必要的。現描述一系列因血鉀過低出現臨床徵狀到急症室求診的病者個案，並討論認識血鉀過低時心電圖的變化現象。

**Keywords:** Electrocardiography, hypokalaemia

**關鍵詞：**心電圖、低鉀血症

### Case 1

A 25-year-old Chinese male presented on 4th July 2004 to the emergency department with generalised weakness and inability to walk. He had no apparent past history of note. His electrocardiogram (ECG) showed evidence of hypokalaemia with ST segment depression seen in leads II, III and aVF as well as flattening of the T waves with appearance of U waves in the anterior leads. The QT interval appeared prolonged as a result of the fusion of T and U waves, best seen in leads V5 and V6 (Figure 1). In addition, the QRS complexes in the anterior leads were widened, measuring 200 msec. This patient's serum

potassium level was 2.5 mmol/L and he was started on intravenous potassium chloride replacement. He was found eventually to have hypokalaemic periodic paralysis and his symptoms resolved once his serum potassium normalised.

### Case 2

A 65-year-old Chinese female patient developed generalised weakness for the last two days with inability to walk. She had been having diarrhoea with occasional bouts of vomiting for the last three days. She presented to the emergency department on 20th May 2004. The ECG showed diffuse ST depressions in the precordial leads with U waves causing pseudo-prolongation of the QT interval (Figure 2). Her serum potassium level was 2.9 mmol/L and symptoms resolved after potassium replacement and treatment of her gastroenteritis.

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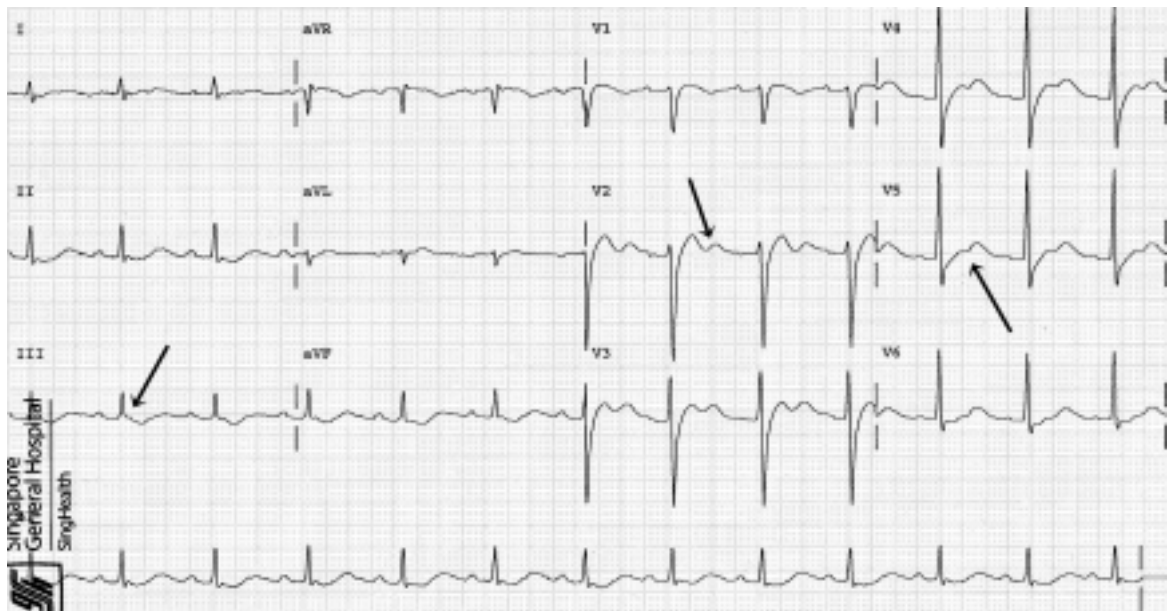


Figure 1. Hypokalaemia with ST-T and U wave changes (arrows).

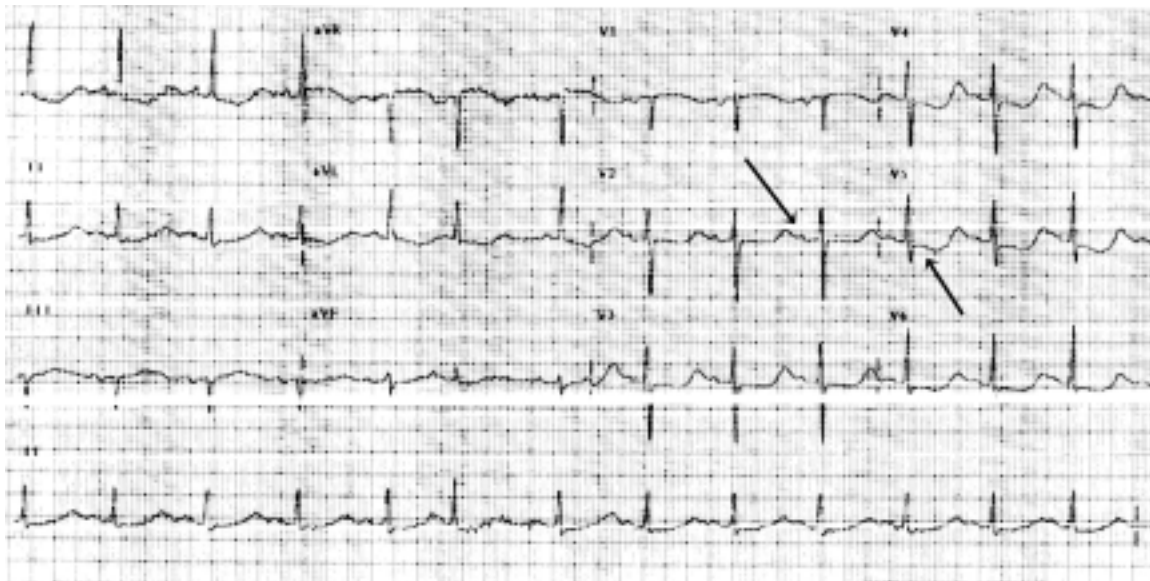


Figure 2. Hypokalaemia with ST and U wave changes (arrows).

### Case 3

A 40-year-old Chinese female patient developed bilateral lower limb weakness one week after being started on antihypertensive medications by her general practitioner. She presented to the emergency department on 18th February 2005. The ECG done at the emergency department showed flattening of the T waves in the inferior leads II, III and aVF. The QT interval was prolonged which was best noted in the precordial leads, measuring 500 msec. In addition, T wave inversions were seen in leads V2 and V3 with U waves seen in leads V4, V5 and V6 (Figure 3). The patient's serum potassium was 2.7 mmol/L and this was eventually attributed to the usage of diuretic therapy.

### Discussion

Hypokalaemia is a relatively common biochemical abnormality found on routine investigation at the emergency department. This is usually a result of depletion due to either inadequate intake or abnormal losses of potassium. Less common causes include those caused by transcellular shift of potassium, whereby extracellular potassium is shifted to the intracellular

space in cells. The causes of hypokalaemia are further illustrated in Table 1.

The effect of hypokalaemia on the cell membrane is to increase the resting membrane potential, and increase the duration of the action potential and refractory period. This explains the arrhythmogenic effects of hypokalaemia.<sup>2,3</sup>

Sequential changes in the ECG take place as the serum potassium is lowered gradually (Table 2).<sup>4</sup> The classically described changes have been: -

1. Reduction in the T wave amplitude
2. Depression of the ST segment
3. U waves, small positive deflections after the T waves, which are best seen in leads V2 and V3

It is essential for physicians to be able to recognise these changes and initiate prompt replacement when necessary.

In severe hypokalaemia, large U waves have been described which may be mistaken for peaked T waves.<sup>8</sup> Differentiating between peaked T waves and giant U waves can be done (Table 3).

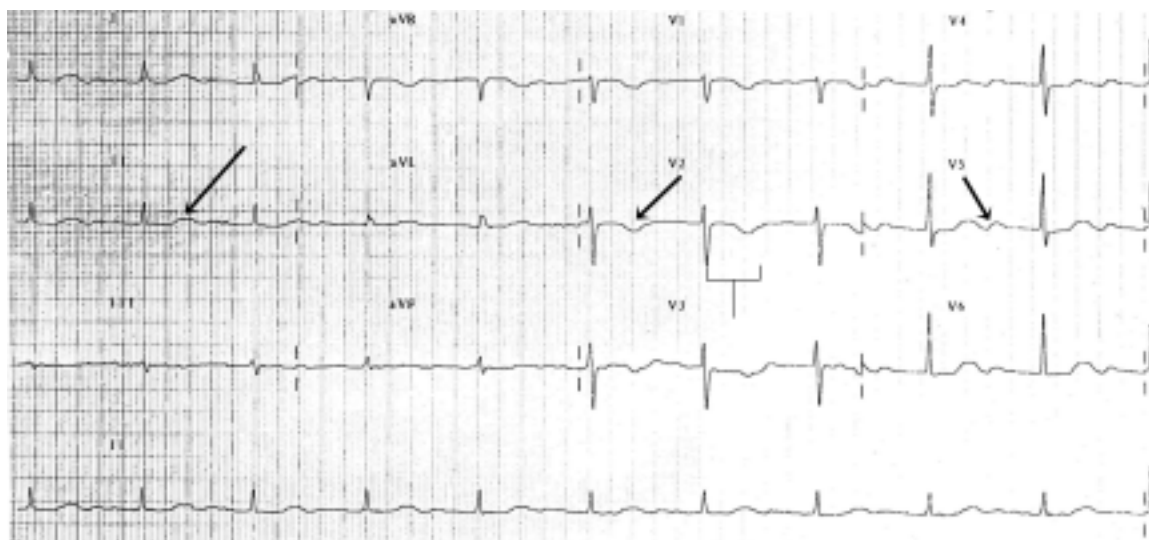


Figure 3. Hypokalaemia with T and U wave changes (arrows).

**Table 1.** Aetiology of hypokalaemia<sup>1</sup>



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1. Inadequate intake
2. Increased excretion
  - Diarrhoea, laxative abuse
  - Renal losses
    - a. Diuretics (loop and thiazide)
    - b. Metabolic alkalosis (vomiting, nasogastric drainage)
    - c. Osmotic diuresis (uncontrolled diabetes)
    - d. Non-reabsorbable anions (penicillins)
    - e. Mineralocorticoid excess (primary hyperaldosteronism, glucocorticoid responsive aldosteronism, congenital adrenal hyperplasia)
  - Apparent mineralocorticoid excess
    - a. Liddle's syndrome
    - b. 11 $\beta$ -hydroxysteroid dehydrogenase deficiency
    - c. Licorice
  - Glucocorticoids (high dose)
    - a. Bartter's and Gitelman's syndrome
    - b. Magnesium depletion
    - c. Renal tubular acidosis (types 1 and 2)
3. Transcellular shift
  - Drugs
    - a.  $\beta$ -adrenergic agonists
      - Bronchodilators, decongestants, tocolytic agents
    - b. Theophylline
    - c. Caffeine
    - d. Insulin
  - Delirium tremens
  - Hyperthyroidism
  - Familial hypokalaemic periodic paralysis
  - Barium poisoning

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**Table 2.** ECG changes with increasing severity of hypokalaemia<sup>4</sup>


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Potassium level [K <sup>+</sup> ]	ECG changes
	ST segment depression Decrease in T wave amplitude Increase in U wave amplitude Fusion of T and U waves (resulting in pseudoprolongation of QT interval) Increase in QRS amplitude and duration <sup>5,6</sup> T wave inversion <sup>7</sup>

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**Table 3.** Differentiating peaked T waves and giant U waves on ECG<sup>3</sup>


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Peaked T waves (Hyperkalaemia)	Giant U waves (Hypokalaemia)
Narrow base	Broad base
Tall T with prominent peak	NA
QT interval normal or decreased	QT interval appears prolonged (actually QU interval)

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Other known ECG changes include

- Increases in P-wave amplitude and duration
- 1st and 2nd degree AV block, predisposing to the development of torsades de pointes which is a polymorphic ventricular tachycardia as a result of early after-depolarisation<sup>2,4</sup>
- Ventricular ectopic beats

Digoxin toxicity is potentiated by hypokalaemia.<sup>4,9</sup> As both digoxin and potassium compete for the same binding site in the membrane sodium-potassium ATPase, hypokalaemia allows more digoxin to bind to the site.

Correction of hypokalaemia can be life-saving and should be given intravenously in the setting of digoxin toxicity, severe myopathy and paralysis (which is usually associated with potassium concentration of <2.0 mmol/L). The rate should be no more than 20 mmol/hr with frequent re-assessment of serum potassium concentration.<sup>4,10</sup> A slightly reduced serum potassium level without ECG changes can simply be treated with oral supplementation.

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