

# The woman who could not hold up her head

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## ABSTRACT

**Low serum potassium concentration is a frequently encountered abnormality, seldom accompanied by life-threatening symptoms. We present a 55-year-old woman with a severe, symptomatic hypokalaemia. The pathogenesis and clinical manifestations are discussed.**

## INTRODUCTION

Hypokalaemia is perhaps the most common electrolyte abnormality encountered in clinical practice. It is found in over 20% of hospitalised patients.<sup>1</sup> Low concentrations may occur in up to 40% of outpatients treated with thiazide diuretics.<sup>2</sup> It is usually well tolerated. In extreme situations patients present with cardiac conduction abnormalities or paralysis. We present a patient with a life-threatening hypokalaemia.

## CASE REPORT

A 55-year-old woman came to our emergency department complaining of difficulty in holding up her head. She had a history of Crohn's disease, for which she had undergone resections of the ileocecum and neoterminal ileum in the past, and recently a further neoterminal ileum resection. She also suffered from chronic obstructive pulmonary disease (FEV<sub>1</sub>: 1.29 l (57%)). She presented with severe weakness in her arms and legs, and stated that since the day before she had been unable to lift up her head. She also

complained of shortness of breath and a nonproductive cough. She had no fever. Since her last operation she had had continuous diarrhoea; five to ten times a day with no obvious signs of blood. She had been on prednisone (25 mg a day) for three days because of shortness of breath. On examination we saw an ill-looking, dehydrated woman, with breathing difficulties. Her blood pressure was 175/95 mmHg, pulse 105 beats/min and temperature 36.7°C. Laboratory tests showed a Hb of 8.8 mmol/l (N. 7.5-9.9), WBC 18.6 x 10<sup>6</sup>/l (N. 4-11 x 10<sup>6</sup>), CRP 65 mg/l (N. <5), Na 148 mmol/l (N. 132-144), K 1.1 mmol/l (N. 3.6-4.8), creatinine 64 µmol/l (N. 62-106), Mg 0.29 mmol/l (N. 0.74-1.48), LDH 1020 U/l (N. 114-235) and CK 4170 U/l (N. 0-50). Urine analysis showed low excretion of magnesium and a normal potassium excretion. Arterial blood gas analysis revealed a pH 7.27, pO<sub>2</sub> 10 kPa, pCO<sub>2</sub> 9.7kPa, bicarbonate 32 mmol/l and an O<sub>2</sub> saturation of 90%. The electrocardiogram showed an abnormal atrial rhythm, normal QRS complex and peaked T waves with prominent U waves (*figure 1*). It was concluded that she was suffering from potassium and magnesium deficiency with muscle weakness, rhabdomyolysis, respiratory insufficiency and cardiac arrhythmia.

She was immediately transferred to the ICU for potassium and magnesium supplementation. Her potassium and magnesium levels normalised within 12 hours. With this treatment she recovered dramatically. The diarrhoea was treated and her electrolytes remained stable with oral supplementation. She was discharged from the hospital in a good clinical condition.



**Figure 1**  
*Registration of lead U<sub>2</sub>: atrial rhythm, normal QRS complex with peaked T waves and prominent U waves*

## DISCUSSION

Patients with hypokalaemia often show no symptoms. When serum potassium levels drop below 3.0 mmol/l nonspecific symptoms may occur, such as muscle weakness. Also an increase in diastolic and systolic blood pressure can be seen. When the concentrations decrease below 2.5, rhabdomyolysis occurs and at concentrations below 2.0 an ascending paralysis can develop. Abnormalities in cardiac conduction can occur, especially in patients with an underlying cardiac disease. Typical electrocardiographic changes include flat T waves, ST-segment depression and prominent U waves. Causes of hypokalaemia can be divided into: 1) transcellular shifts due to insulin, catecholamines or B<sub>2</sub>-adrenergic receptor stimulation, 2) low intake (<1 gram/day) and 3) abnormal stool or urinary loss, because of diuretic therapy, drugs with mineralocorticoid or glucocorticoid effects, use of laxatives or disorders accompanied by acid-base imbalances. Magnesium depletion, either due to low intake or abnormal loss, reduces the intracellular potassium concentration further and causes renal wasting.<sup>2,3</sup> In most cases low serum potassium concentration is secondary to drug treatment, particularly diuretics.

Our patient presented with a severe, symptomatic hypokalaemia and magnesium deficiency due to chronic diarrhoea after her last bowel resection. The hypernatraemia was due to the dehydration. She presented with muscle

weakness, rhabdomyolysis, elevated blood pressure and cardiac conduction abnormalities. When we retrospectively analysed her laboratory results she already had a low serum potassium concentration (2.7 mmol/l) at discharge after her last operation. The acute lowering of the potassium concentration was probably due to the mineralocorticoid effect of prednisone.<sup>2,4</sup> Normally this leads to high urinary potassium loss. The reason why there was no high urinary potassium excretion probably lies in her longstanding and deep hypokalaemia. The acidosis was due to her extreme muscle weakness and therefore compromised respiration. Normally one would expect a higher bicarbonate level in a pure metabolically compensated respiratory acidosis but, probably due to faecal bicarbonate loss, this was not the case. So the acid-base disturbance in our patient was a combined one, due to respiratory and metabolic changes. The ECG abnormalities at presentation are in accordance with ECG changes seen in hypomagnesaemia (peaked T and U waves) and hypokalaemia (U waves).<sup>5</sup> After correction these abnormalities disappeared. In patients with extreme muscle weakness and ECG abnormalities, the possibility of severe electrolyte abnormalities should be suspected and immediate correction is warranted.

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## REFERENCES

1. Paice BJ, Paterson KR, Onyanga-Omara F, Donnelly T, Gray JMB, Lawson DH. Record linkage study of hypokalemia in hospitalized patients. *Postgrad Med J* 1986;62:187-91.
2. Gennari FJ. Hypokalemia. *N Engl J Med* 1998;339(7):451-8.
3. Cohn JN, Kowey PR, Whelton PK, Prisant LM. New guidelines for potassium replacement in clinical practice. *Arch Intern Med* 2000;160:2429-36.
4. Akerkar GA, Peppercorn MA, Hamel MB, Parker RA. Corticosteroid-associated complications in elderly Crohn's disease patients. *Am J Gastroenterol* 1997;92(3):461-4.
5. Schamroth L. The 12 lead electrocardiogram. Book 1. Blackwell Scientific Publications, 1989:337-9.