Hypokalaemia in a woman with eating disorder

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Abstract

Chronic hypokalaemia often remains a diagnostic challenge, especially in young women without hypertension. A concealed diuretic abuse should be suspected, especially in young women with eating disorders. This case describes a woman with chronic hypokalaemia in whom a thorough medical history and proper laboratory tests were essential to early and accurate diagnosis.

Keywords

Hypokalaemia; eating disorders; diuretics.

Introduction

Chronic hypokalaemia often remains a diagnostic challenge, especially in young women without hypertension. After the exclusion of the most obvious causes, a concealed diuretic abuse associated with or without surreptitious vomiting and laxative abuse should be suspected, especially in young women concerned with their body image. A conclusive diagnosis may be difficult as such patients often vigorously deny diuretic intake\cite{1}. Also, only a minority of patients with eating disorders (approximately 6\%) abuse diuretics\cite{2-4}. This case describes a woman with chronic hypokalaemia in whom a thorough medical history and proper laboratory tests were essential to an early and accurate diagnosis.

Case history

A 27-year-old woman was referred to the nephrology outpatient service because of severe, chronic unexplained hypokalaemia associated with chronic fatigue and amenorrhea. Hypokalaemia was first discovered in this patient in 2005 and confirmed (always <3.0 mmol/L) at least thrice yearly afterwards despite oral potassium supplements. Her past medical history was notable for hypokalaemia only, and a family history was unrevealing. She denied being extremely thin and underweight and had fear of becoming fat. Medications included oral potassium supplements and

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multivitamins. The patient denied any history of vomiting, diarrhoea or intake of diuretics and laxatives.

On clinical examination, her body weight was 48 kg for a height of 165 cm; her heart rate was 78 bpm and blood pressure 90/50 mmHg, without postural change. The rest of the examination was unremarkable. Laboratory studies revealed profound hypokalaemia of 2.1 mmol/L (normal range 3.6–5.0 mmol/L), serum chloride <80 mmol/L (normal range 100–110 mmol/L), and metabolic alkalosis with total CO₂ 41 mmol/L (normal range 22–29 mmol/L). Renal and liver function, serum sodium, and urinalysis were normal. Serum magnesium, plasma renin, aldosterone, urinary electrolytes and assay for diuretics were arranged; her oral potassium dose was increased, and the follow-up visit was scheduled at 2 weeks.

One week later the patient was admitted to the hospital because of chest pain and palpitations with serum potassium of 1.9 mmol/L, which improved with intravenous potassium. On the follow-up visit to our office her serum potassium was 3.1 mmol/L, serum magnesium, plasma renin, and aldosterone were at normal levels, spot urine potassium was 35 mmol/L. Urine assay for diuretics showed a large amount of furosemide. When specifically asked again about diuretics intake and shown the urine assay results the patient confessed. In particular, she preferred oral furosemide, which she obtained from her native country, Russia. This had been going on for quite some time, perhaps 4 years. After further discussion with the patient, she agreed to stop taking diuretics and undergo psychiatric evaluation.

**Diagnosis**

Severe hypokalaemia due to diuretic abuse in a woman with eating disorder.

**Discussion**

Eating disorders (anorexia nervosa and bulimia nervosa) are syndromes characterized by severe disturbances in eating behaviour and by distress or excessive concern about body shape or weight. The essential features of anorexia nervosa are refusal to maintain a minimally normal body weight, intense fear of gaining weight, and significant disturbance in the perception of the shape or size of one’s body. Bulimia nervosa is characterized by binge eating and inappropriate compensatory behaviour such as fasting, vomiting, using laxatives, or exercising to prevent weight gain. Eating disorders have been reported in up to 4% of adolescents and young adults with anorexia nervosa affecting approximately 0.5–1% of women of college age. Both anorexia nervosa and bulimia nervosa are more commonly seen in girls and women. Estimates of the ratio of females to males range from 6:1 to 10:1.

Patients with anorexia nervosa or bulimia nervosa may develop major electrolyte abnormalities, primarily as a result of purging, with potentially serious medical consequences. The mortality rate for anorexia nervosa has been estimated at approximately 6%[2]. Most of the studies[2–4,7] reported that almost half of the subjects had electrolyte and haematologic abnormalities, including metabolic alkalosis, hypochloraemia, hypokalaemia, anaemia, thrombocytopenia and leukopenia. They also observed that low plasma levels of potassium and chloride were found most commonly in women who induced vomiting or abused laxatives. Hypokalaemia has been found in up to 20% of people with eating disorders and is the most clinically important of the electrolyte abnormalities, since it can cause life-threatening conditions, including myopathy, cardiac arrhythmias, rhabdomyolysis, and nephropathy. Hypokalaemia in eating disorders usually develops as a result of diuretic use and gastrointestinal losses, such as vomiting and diarrhoea. Potassium concentration in gastric fluid is less than 10 mEq/L and loss of this fluid only partially explains hypokalaemia in patients with vomiting[9]. Gastric acid secretion generates bicarbonate causing metabolic alkalosis. Increased tubular bicarbonate delivery and elevated aldosterone secondary to volume contraction accelerate the rate of potassium secretion, causing potassium depletion and worsening hypokalaemia. The concentration of potassium in lower gastrointestinal secretion can exceed 80 mEq/L. Diarrhoea because of laxative and enema abuse can be associated with excessive loss of potassium in stools. Both the thiazide and loop diuretics block chloride-associated sodium reabsorption (with each inhibiting a different membrane-transport protein) and, as a result, increase delivery of sodium to the collecting tubules[9].

Diuretic abuse was found only in approximately 6% of patients with eating disorders with or without hypokalaemia[2–4]. An accurate diagnosis of diuretic abuse can be very difficult because these patients often deny it and also take the diuretics only intermittently and, as a consequence,
their urinary chloride concentration may change between high (>70 mmol/L) and low (<20 mmol/L) values\textsuperscript{10}. Urinary samples with high concentration of chloride will test positive for diuretics, often for thiazide or for furosemide. Diuretic abuse can also be very similar to Gitelman syndrome. Therefore, the urine assay for diuretics is of great importance in these patients. Also, it would be prudent for clinicians to conduct routine screening for hypokalaemia in patients who are most at risk; i.e. those who are in the lower weight ranges and for whom intense purging is acknowledged or suspected.

### Teaching points

- Hypokalaemia is the most clinically important of the electrolyte abnormalities in patients with eating disorders.
- Only a minority of patients with eating disorders abuse diuretics and conclusive diagnosis can be very difficult as such patients often deny diuretic intake.
- Concealed diuretic abuse associated or not with surreptitious vomiting and laxative abuse should be suspected in young women concerned with their body image.
- Urine assay for diuretics and routine screening for hypokalaemia should be performed in patients who are suspected of having eating disorders.

### References