A Rare Case of Hypokalemia and Hypomagnesemia

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CASE DESCRIPTION

An 18-year-old adolescent male presented to our emergency department complaining of chest pain that started about 2 days earlier and remained unchanged. Chest x-rays revealed a right apical pneumothorax. The patient did not use any medication. Two months earlier he had presented to the emergency department with a similar episode that resolved spontaneously. Blood testing performed at the time of presentation showed severe hypokalemia with a potassium concentration of 2.5 mmol/L. He was admitted to our respiratory unit.

He was 180 cm (70.9 in) tall and weighed 64 kg (141 lb, body max index 19.8 kg/m^2). He was in good general health, and the physical examination was unremarkable. A renal ultrasound was normal. He noted an unintentional weight loss of 7 kg (15.4 lb) over the last 36 months. Furthermore, he reported fatigue and muscle weakness. His personal history and family history were unremarkable. He was delivered naturally at term and developed normally during childhood and puberty.

Further blood tests revealed moderate hypomagnesemia of 1.2 mg/dL [0.5 mmol/L; reference interval 1.7–2.2 mg/dL (0.7– 0.9 mmol/L)]. Arterial blood gas analysis showed metabolic alkalosis (pH 7.46, bicarbonate 28.9 mmol/L). Sodium, chloride, and calcium were within reference intervals. Renal function, fasting blood glucose, and a complete blood count were normal. Table 1 provides a selection of relevant laboratory results at presentation and during follow-up.

As recommended by the British Thoracic Society, the patient was scheduled for video-assisted thoracoscopic surgery *(1).* Before surgery, an endocrine specialist was consulted to further investigate the cause of the patient's hypokalemia and hypomagnesemia. The spontaneous pneumothorax was unrelated to the abnormal electrolyte results and is not further discussed here. The patient's pronounced hypokalemia and hypomagnesemia were initially treated with potassium canrenoate, potassium chloride, and magnesium (Fig. 1). With this therapy, serum potassium improved and stabilized at 2.8 –2.9 mmol/L. Subsequently, amiloride was added to the patient's therapy, which led to a further improvement of serum potassium. However, he reported increasing fatigue, which led to the discontinuation of potassium canrenoate, without which correction of potassium was insufficient. Therefore, it was decided to replace amiloride by potassium canrenoate and spironolactone. Three months after the initial presentation, his potassium had risen to 3.4 mmol/L.

Within 10 weeks after the initiation of spironolactone therapy, he developed gynecomastia, a well-known side effect of this drug. Consequently, spironolactone was again replaced by amiloride, although at a higher dose. As serum potassium remained low, at approximately 2.8 mmol/L, he was switched from amiloride to eplerenone, a mineralocorticoid receptor antagonist that does not cause gynecomastia. Potassium and magnesium supplementation were continued as before. With this therapy, serum potassium and magnesium concentrations stabilized near the lower limit of the reference interval.

DOI: 10.1373/clinchem.2015.242057

Reference

1. Management of spontaneous pneumothorax: British Thoracic pleural disease guideline 2010 BTS. Thorax 2010;65:18 –31.

Table 1. Laboratory results at presentation and on further investigation.[a]

Parameter	Result	Reference interval
Presentation		
Blood Chemistry		
Potassium, mmol/L	2.5	3.6-5.0
Calcium, mmol/L	2.46	2.15-2.60
Sodium, mmol/L	140	135-145
Chloride, mmol/L	96	95-108
Magnesium, mg/dL	1.21	1.7-2.2
Creatinine, mg/dL	0.78	0.50-1.20
Urea, g/dL	34	18-55
Total protein, mg/dL	7.1	6.6-8.3
Glucose, mg/dL	85	60-99
Uric acid, mg/dL	5.7	3.6-7.0
Aspartate transaminase, U/L	20	<40
Alanine transaminase, U/L	16	<40
Lactate dehydrogenase, U/L	134	120-230
Bilirubin, mg/dL	1.2	<1.4
Complete blood count		
Leukocytes, $\times 10^3$ cells/μL	4,000	3600-10 000
Erythrocytes, $\times 10^6$ cells/μL	5.36	4.50-5.90
Hemoglobin, g/dL	15.9	13.0-17.5
Platelets, $\times 10^3$ cells/μL	182	150-410
Further investigation		
Urine chemistry		
Potassium, mmol/24 h	126	25-125
Sodium, mmol/24 h	277	40-220
Calcium, mmol/24 h	1,5	2.5-7.5
Phosphate, mmol/24 h	21	13-42
Chloride, mmol/24 h	342	110-250
Hormones		
Testosterone, ng/mL	5.02	2.39-8.36
Cortisol, ng/mL	128	59-217
Adrenocorticotropic hormone, pg/mL	46.8	4.7-48.8
Thyroid-stimulating hormone, μIU/mL	2.49	0.5-4.3
Arterial blood gas analysis		
pH	7.45	7.35-7.45
pCO_2, mmHg	41.7	35-48
HCO_3^-, mmol/L	28.9	21-28
Base excess, mmol/L	4.9	−2 to 3

[a] To convert magnesium from mg/dL to mmol/L, multiply by 0.411; creatinine from mg/dL to μmol/L, multiply by 88.4; urea from g/dL to mmol/L, multiply by 166.5; glucose from mg/dL to mmol/L, multiply by 0.0555; uric acid from mg/dL to mmol/L, multiply by 0.059; bilirubin from mg/dL to μmol/L, multiply by 17.1; calcium from mmol/24 h to mg/dL, multiply by 40; and phosphate from mmol/24 h to g/24 h, multiply by 0.033.

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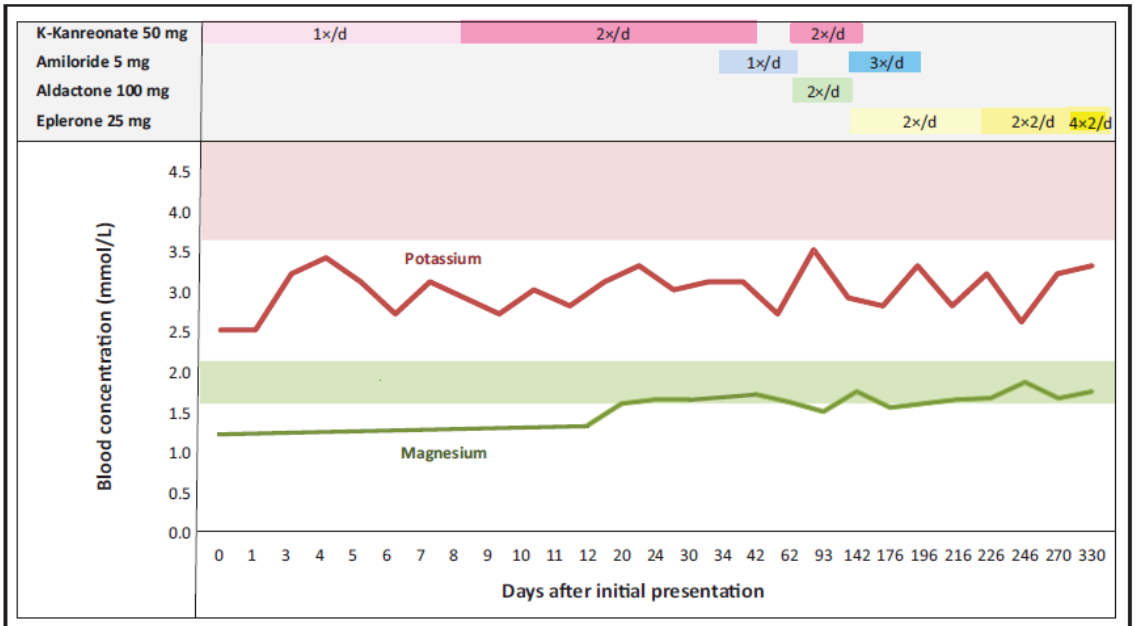

Fig. 1. Blood concentrations of potassium and magnesium over time.

QUESTIONS TO CONSIDER
• What are the most common causes of hypokalemia?
• Which laboratory tests can differentiate between renal and nonrenal causes of potassium loss?
• Describe some rare genetic causes of hypokalemia?

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the March 2016 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol62/issue3 and follow the link to the Clinical Case Study and Commentaries.

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