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# Hypokalemia in anorexia nervosa

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| Abstract: | The etiologies of hypokalemia in individuals with anorexia nervosa are frequently multifactorial (Table 1). Primary care physicians should carefully take medical and drug history to find underlying causes. Urinary electrolytes further provide us a clue to confirm the diagnosis. |
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# TITLE: Hypokalemia in anorexia nervosa

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**Keywords**: anorexia nervosa; hypokalemia; hypokalemic nephropathy

The 25-year-old woman was admitted to our department for weakness and lassitude of two months duration. She was diagnosed as having major depression disorder at age 20 and anorexia nervosa —binge-eating/purging subtype (ANp)— at age 21. She had noticed amenorrhea for one year. Her medical history revealed repeated admissions for hypokalemia caused by self-induced vomiting and laxative abuse. Three months before admission, she started to take health products with unknown content for thinness.

On examination, she had a height of 165 cm and a weight of 39 kg (body mass index 14.3 kg/$m^2$). Her blood pressure was 92/63 mmHg. Serum creatinine value was 0.58 (normal 0.2—1.2) mg/dl, sodium 135 (normal 135—145) mmol/l, potassium 1.6 (normal 3.5—4.9) mmol/l, calcium 9.4 (normal 8.6—10.3) mg/dl, phosphate 3.5 (normal 2.5—4.5) mg/dl, and chloride 96 (normal 97—110) mmol/l. Spot urinary sodium concentration was 106 mmol/l, potassium 12.7 mmol/l, calcium < 2 mg/dl, creatinine 10.2 mg/dl, and chloride 101.4 mmol/l. Arterial blood gas showed

pH 7.54 (normal 7.35—7.45), PO2 102 (normal 80—105) mmHg, PCO2 44 (normal 38—42) mmHg, and HCO3 39 (normal 22—26) mmol/l. The renal echo (Figure 1) revealed marked medullary hypertrophy in both kidneys. Intravenous pyelography did not disclose abnormalities. Chemical analysis via high performance liquid chromatography identified hydrochlorothiazide within health products.

The etiologies of hypokalemia in individuals with ANp are frequently multifactorial (Table 1) [1]. Primary care physicians should carefully take medical and drug history to find underlying causes. Urinary electrolytes further provide us a clue to confirm the diagnosis [2]. In this case, elevated urinary chloride and decreased urinary calcium values strongly imply the use of thiazide even if the patient denied it. On the other hand, chronic hypokalemia caused by repeated purging, laxatives, or diuretics abuses, will cause renal tubular cell hyperplasia involving the medullary collecting ducts. Eventually, it leads to tubulointerstitial fibrosis

and progressive loss of renal function – namely, “hypokalemic nephropathy” [3].

## References

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3. Arimura Y, Tanaka H, Yoshida T, et al. Anorexia nervosa: an important cause of chronic tubulointerstitial nephropathy. Nephrol Dial Transplant 1999;14:957–9.

**Figure caption**

Figure 1: Renal ultrasonography from a 25-year-old woman with anorexia nervosa, showing diffusely hyperechoic medullary lesions without shadowing or papillary necrosis (asterisks).

Table 1: Differential diagnosis of hypokalemia in subjects with anorexia nervosa

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| Gastrointestinal loss |
| Poor oral intake |
| Self-induced Vomiting |
| Laxative abuse |
| Renal loss |
| Diuretic abuse |
| Chronic tubulointerstitial nephritis |
| Metabolic alkalosis (i.e. loss of $HCO_3^-$) |
| Hypovolemia-induced hyperaldosteronism |


Renal ultrasonography from a 25-year-old woman with anorexia nervosa, showing diffusely hyperechoic medullary lesions without shadowing or papillary necrosis (asterisks).
147x107mm (96 x 96 DPI)