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Case Report

Does hypokalemia contribute to acute kidney injury in chronic laxative abuse?

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Abstract

Prolonged hypokalemia from chronic laxative abuse is recognized as the cause of chronic tubulointerstitial disease, known as "hypokalemic nephropathy," but it is not clear whether it contributes to acute kidney injury (AKI). A 42-year-old woman with a history of chronic kidney disease as a result of chronic laxative abuse from a purging type of anorexia nervosa (AN-P), developed an anuric AKI requiring hemodialysis and a mild AKI 2 months later. Both episodes of AKI involved severe to moderate hypokalemia (1.2 and 2.7 mmol/L, respectively), volume depletion, and mild rhabdomyolysis. The histologic findings of the first AKI revealed the remnants of acute tubular necrosis with advanced chronic tubulointerstitial nephritis and ischemic glomerular injury. Along with these observations, the intertwined relationship among precipitants of recurrent AKI in AN-P is discussed, and then we postulate a contributory role of hypokalemia involved in the pathophysiology of the renal ischemia-induced AKI.

Introduction

The term "kaliopenic nephropathy" was introduced by Conn and Johnson [1] in 1956 to describe a distinct clinical entity characterized by the pathologic lesions of vacuolar tubulopathy and functional disorders such as impaired urine concentration in patients resulting from long-term potassium depletion. Since then, chronic laxative or diuretic abuse in patients of anorexia nervosa of purging type (AN-P) has been reported as a cause of chronic tubulointerstitial nephropathy due to hypokalemic nephropathy, sometimes leading to end-stage renal disease (ESRD) requiring renal replacement therapy including dialysis or renal transplantation [2–4]. There have been some reports of acute kidney injury (AKI) from unidentified causes in preexisting chronic kidney disease (CKD) due to hypokalemic nephropathy in the presence of chronic hypokalemia in AN-P [2,4,5].

Through the present case, we wish to advance a postulate that hypokalemia plays an important role in precipitating renal ischemia-induced AKI in the presence of other contributors of AKI.

Case report

A 42-year-old woman presented at the emergency department (ED) with chief complaints of anergy and progressive weakness of lower extremities for the past 10 days. She was emaciated with a body mass index of 14 kg/m^2 (weight 34 kg, height 155 cm). On further questioning, she confessed to having taken large amounts of three types of nonprescription laxatives (up to 50 tablets daily) for at least 10 years after the

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birth of her second child. The laxative dosage was increased to almost 70 tablets a day over the previous month, and during this period she developed progressive loss of appetite. The laxatives contained bisacodyl, docusate sodium, sennoside calcium, *Lactobacillus acidophilus*, and carbenoxolone (glycyrrhetinic acid).

Vital signs in the supine position showed blood pressure 80/64 mmHg, heart rate 84/min, respiratory rate 24/min, and body temperature 37°C. She appeared severely volume depleted with poor skin turgor, dry oral mucosa, and shiny skin on both legs.

The initial laboratory data in the ED were as follows—complete blood examination: white blood cell count 12.1 × 10^3/mm^3, hemoglobin 9.0 g/dL, platelets 307 × 10^3/mm^3; serum chemistry: sodium 127 mmol/L, potassium 1.2 mmol/L, chloride 94 mmol/L, urea nitrogen 117 mg/dL, creatinine 11.7 mg/dL, calcium 6.3 mg/dL phosphate 8.5 mg/dL magnesium 2.2 mg/dL intact parathyroid hormone 80 pg/mL, creatine phosphokinase 3,371 units/L (30–180 units/L). Arterial blood gas on 4 L oxygen per minute showed pH 7.19, pO_2 163 mmHg, pCO_2 10.8 mmHg, HCO_3 4.2 mmol/L. Urinalysis yielded the following results: albumin (2+), occult blood (2+) with no red blood cells/high power field (HPF), pyuria 5–10/HPF with no bacteria.

A renal ultrasonogram on Day 3 showed 9.5 cm bilaterally with prominent medulla and nephrolithiasis in the left renal pelvis without hydronephrosis. In the supine position, the plasma renin activity and aldosterone level was 15.29 ng/mL/h (0.68–1.36 ng/mL/h) and 98.6 ng/dL (1.0–16 ng/dL), respectively. The serologic markers for glomerulonephritis were negative.

In the ED and the intensive care unit, 3 L of a half-normal saline mixed with KCl (60 mmol/L) and bicarbonate (100 mmol/L) were infused over 12 hours. However, the patient remained anuric (< 5 mL/h) with serum potassium of 1.7 mmol/L, and the electrocardiogram showed U waves, arterial pH 7.23, and blood bicarbonate 8.4 mmol/L. Subsequently, hemodialysis with 4 mmol/L of potassium in the dialysate was initiated and continued at a frequency of three times a week for 10 days until the patient passed urine (> 700–800 mL/d).

On Day 15, a kidney biopsy showed advanced chronic tubulointerstitial nephritis with some evidence of acute tubular necrosis (ATN). The predominant light microscopic lesions were moderate to severe residual atrophy or loss of tubules and scattered interstitial mononuclear cell infiltration (Fig. 1A). Also, focal tubular degenerative change and mild regenerative proximal tubules suggested ATN in the recovery phase, and a few nonatrophic tubular epithelial cells in the proximal tubule were focally enlarged and vacuolated. The glomeruli displayed ischemic changes such as wrinkling and thickening of the glomerular capillary walls but without advanced glomerular lesions (Fig. 1B). However, there was no evidence of juxtaglomerular hyperplasia or arteriolosclerosis. Immunofluorescence examinations showed no immunoglobulins or complements. Ultrastructurally, diffuse thickening of the glomerular basement membrane was noted with no electron dense deposits. Foot processes of podocytes showed moderate effacement (Fig. 1C).

On the day of discharge, the patient weighed 38 kg with serum potassium of 4.3 mmol/L and creatinine of 1.8 mg/dL. After intensive counseling from renal, psychiatric, social, and dietary consultants, she understood the seriousness of her condition, and agreed to refrain from laxative abuse.

However, on the second monthly follow-up at the outpatient renal clinic, her serum creatinine was increased to

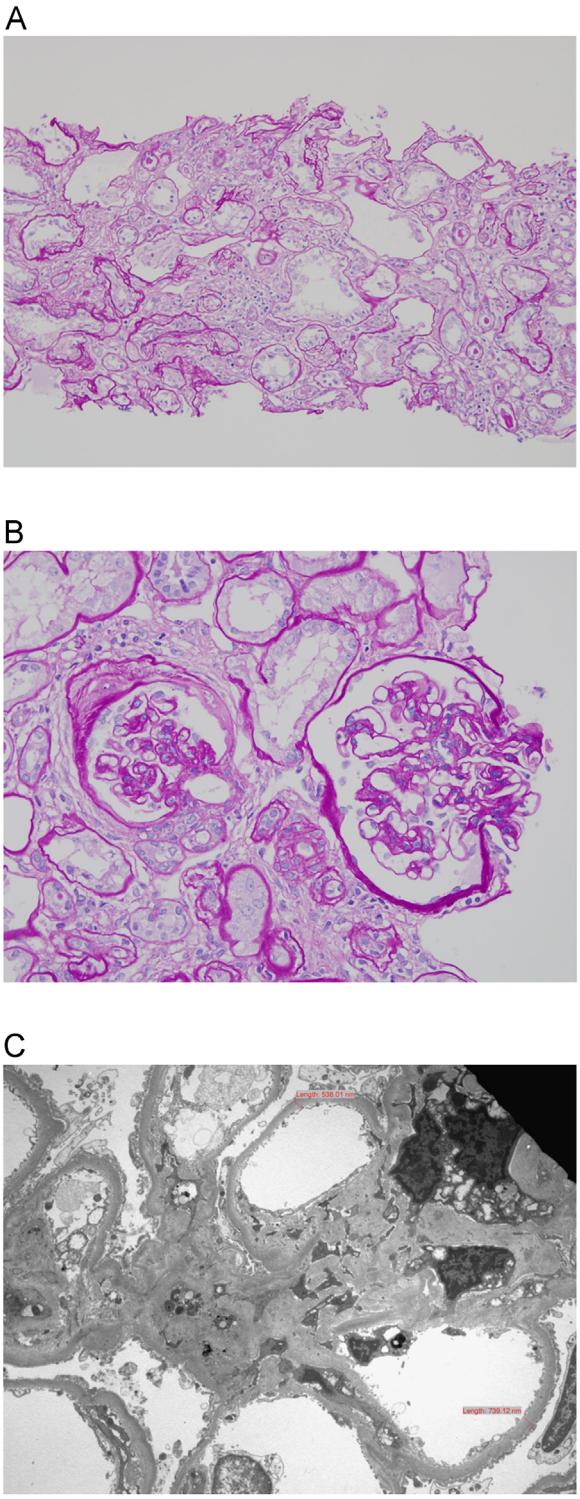

Figure 1. Renal histology. (A) The interstitium shows marked fibrosis with mild mononuclear cell infiltration. The tubules show various morphologic changes, including atrophy, detachment, and vacuolization of the epithelial cells, with nuclear atypia, which suggest tubular injury due to chronic damage (PAS, × 200). (B) The glomeruli show focal ischemic collapse (left) and segmental thickening of the glomerular basement membrane (right). The arterioles are unremarkable (PAS, × 400). (C) Electron microscopy shows diffuse thickening of the glomerular basement membrane, with average thickness of 574 nm. The foot processes are moderately effaced (× 3,500). PAS, periodic acid-Schiff.