Membranoproliferative Glomerulonephritis Presenting with Hypokalemia

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Dear Sir,

The association of tubulointerstitial structural or functional disorders with glomerular diseases has been a well-known phenomenon [1–3]. The immunopathogenetic mechanisms that cause glomerulonephritis may also induce tubulointerstitial nephritis (TIN) or TIN may be a nonspecific reaction of kidney to a variety of etiopathogenetic factors, including severe glomerular disease [4].

Here, we present a patient with membranoproliferative glomerulonephritis (MPGN) whose renal disease was dominated by rhabdomyolysis caused by hypokalemia.

Case Report

A 19-year-old male farmer was admitted for evaluation of severe hypokalemia. He was well until 4 months earlier when weakness, polyuria and nocturia first began. He had no history of use of diuretic, laxative, licorice, carbenoxolone or any other drug. Neither excessive vomiting, diarrhea nor similar disease in his family was present. His blood pressure was 170/105 mm Hg. Physical examination revealed mild lethargy, pretibial edema and generalized muscle tenderness.

Laboratory values at the beginning were as follows. The hemoglobin was 10.4 g/dl (104 g/l), the blood urea nitrogen 62 mg/dl (22.3 mmol/l), serum creatinine 5.1 mg/dl (452 µmol/l), potassium 1.1 mEq/l (1.1 mmol/l), calcium 8.7 mg/dl (2.1 mmol/l), phosphorus 7.4 mg/dl (2.5 mmol/l), albumin 2.8 g/dl (28 g/l), globulin 3.0 g/dl (30 g/l). Serum alanine aminotransferase was 205 U/l (3.4 mmol/s/l), aspartate aminotransferase 316 U/l (5.3 mmol/s/l), lactic dehydrogenase 867 U/l (14.7 µmol/s/l) and creatine kinase 28,764 U/l (489 mmol/s/l). Arterial blood pH was 7.38, PaCO₂ 32.3 mm Hg, PaO₂ 85.8 mm Hg, and bicarbonate 18.6 mmol/l. Plasma osmolality was 320 mosm/kg. In urinalysis, the pH was 5.5, density 1,009, osmolality 200 mosm/kg, sodium 0–17 mEq/day (0–17 mmol/day), potassium 24–63 mEq/day (24–63 mmol/day), calcium 255 mg/day (6.1 mmol/day), and phosphorus 287 mg/day (9.7 mmol/day). Urine volume varied between 1,200 and 2,100 ml per day. Daily urinary protein excretion was calculated as 1.8 g. Creatinine clearance was 10.7 ml/min. Electrocardiogram showed prominent U waves in precordial derivations. Plasma aldosterone levels were 35.7 pg/ml (1.00 nmol/l) in supine (normal value: 10–160 pg/ml or 0.28–4.52 nmol/l) and 31.6 pg/ml (0.89 nmol/l) in upright position (normal value: 40–310 pg/ml or 1.12–8.75 nmol/l). Plasma renin activities were 0.48

ng/ml/h in supine (normal value: 0.15–2.33 ng/ml/h) and 0.25 ng/ml/h in upright position (normal value: 1.30–3.95 ng/ml/h). Serum C3 and C4 levels were 46 mg/dl (normal value: 55–120 mg/dl) and 18 mg/dl (normal value: 20–50 mg/dl), respectively. Serum antinuclear antibody and anti-DNA antibody were negative. Computerized tomography indicated bilateral thickening of renal parenchyma and normal adrenal glands. Myopathy and demyelination were detected in electromyography.

Renal biopsy showed focal glomerulosclerosis, cellular proliferation and thickening of glomerular basement membrane (GBM) in addition to vacuolization, atrophy and dilatation of proximal tubuli and interstitial fibrosis. Congo red staining was negative. Immuno-fluorescent study showed mild granular deposition of IgG, IgM and third component of complements along the GBM and tubular basement membranes (TBM). These findings were consistent with MPGN with focal sclerosis and hypokalemic nephropathy. Microscopic examination of gastrocnemius muscle demonstrated diffuse inflammatory infiltration with necrosis of muscle.

The patient was placed on a diet containing 0.5 g/kg protein, 0.5 g sodium chloride, 6 g potassium, 300 mg phosphorus per day and was also given parenteral potassium-chloride supplementation. His blood pressure became under control with prazosin plus propranolol. Azathioprine was initiated for glomerulonephritis. Serum values of muscle enzymes declined following restoration of serum potassium level. After 20 days of hospitalization, the patient was discharged with azathioprine, prazosin, propranolol and phosphate-binding agent.

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Comment

In this patient, severe potassium depletion caused by excessive renal losses, in the face of suppressible aldosterone levels and no adrenal hyperplasia or mass, was thought to be originated from tubular changes accompanying primary glomerulonephritis.

There have been many explanatory studies demonstrating how TIN accompany glomerular diseases. First, circulating immune complexes may localize along tubular structures and interstitium as well as GBM. Chronic serum sickness glomerulonephritis in rabbit and systemic lupus erythematosus in human are such examples. Second, anti-TBM disease has been reported in conjunction with anti-GBM nephritis. Third, in patients with MPGN, type II, deposits of terminal components of complement system, properdin and minor amounts of IgM are found not only along the GBM, but also along the TBM. The other pathogenetic mechanisms which induce TIN are autoantibodies against renal cellular antigens, cell-mediated hypersensitivity and immediate IgE-type hypersensitivity [4].

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