Exercise-Induced Renal Failure in a Patient with Hyperuricosuric Hypouricemia

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Dear Sir,

Renal isolated hypouricemia is a rare condition. The patient is asymptomatic, and the main complication is known as urolithiasis. We experienced a case of exercise-induced acute renal failure who had renal isolated hypouricemia.

A 14-year-old girl was referred to our hospital on September 13, 1991, because of acute renal failure. On September 11, she complained of nausea and abdominal pain while she took lessons in an athletic meeting. She consulted her familial doctor, and protein-uria, but not hematuria, and renal dysfunction (blood urea nitrogen 22.6 mg/dl and serum creatinine 2.3 mg/dl, were first pointed out. On admission, she was normally developed and well nourished, and physical examination revealed no abnormalities. Urinalysis showed a 1+ test for protein and no hemoglo-

binuria, and the sediment was normal. Blood chemistry findings showed renal failure (blood urea nitrogen 24.7 mg/dl, serum creatinine 3.4 mg/dl, sodium 140 mEq/l, potassium 5.1 mEq/l, chloride 106 mEq/l, calcium 9.7 mg/dl and phosphate 4.4 mg/dl), but her serum uric acid was within normal limits (2.3 mg/dl). Serum creatinine kinase was 57 IU/l. Serum and urinary myoglobin were 42 ng/ml and less than 5 ng/ml, respectively. The concentrations of C3 and C4 were normal, and the titer of antinucleatic antibody was normal. Renal echography showed no evidence of urolithiasis and hydronephrosis. Thus, collagen diseases and rhabdomyolysis were denied. She received sufficient transfusion, and her serum creatinine decreased to 1.0 mg/dl on September 20, 1991. Simultaneously, her serum uric acid decreased to 0.8 mg/dl. Urinary excretion of uric acid was 320 mg/day. Fractional excretion of uric acid (FE_{ua}; C_{ua}/C_{cr}) was high (55%). Neither glycosuria nor aminoaciduria was shown. To evaluate the disturbance in the renal handling of uric acid, a benzbromarone (100 mg)-pyrazinamide (3.0 g) test was done on March 20, 1992, when she had normal renal function (serum creatinine 0.6 mg/dl and serum uric acid 0.8 mg/dl). FE_{ua} was not affected by either drug and was maintained at high levels (62-68%). The pattern of renal defect was classified as presecretory reabsorption defect [1]. Thereafter, she stopped moderate to severe exercises, and she did not meet with an episode of acute renal failure.

To our knowledge, 6 renal hypouricemic cases with acute renal failure were reported [2-5] (table 1). Six cases of 7, our case in-

Table 1. ARF in renal hypouricemic patients

ARF = Acute renal failure; Creat. = serum creatinine; UA = serum uric acid; CK = serum creatine kinase; ND = not done; IS = increased secretion of uric acid; pre. RD = presecretory reabsorption defect of uric acid.

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cluded, developed acute renal failure after moderate exercises. Rhabdomyolysis was denied in all cases by laboratory data although case 4 had a high creatine kinase level. The concentrations of serum uric acid were normal or low in spite of their renal dysfunction. When all cases returned to normal renal function, their uric acid levels became very low. Erley et al. [2] showed intratubular urate crystals in the renal biopsy specimen of case 1. Uric acid nephropathy was suspected as a cause of his renal failure. Although our case could not confirm the tubular obstruction by uric acid crystals histologically, other causes of acute renal failure could not be revealed. Exercise increases production of uric acid and sweating concentrates urine [6]. Thus, in renal hypouricemic patients, the concentration of uric acid at the collecting duct is expected to increase more easily by exercises, resulting in uric acid nephropathy. Exercise-induced renal failure might be one of the complications in a renal uricosuric patient. The prognosis is good because all reported cases returned to normal renal function within 1 month. However, 2 patients (cases 1 and 5) needed hemodialysis treatment. Renal hypouricemic patients may take care on exercises. The disturbed portion of renal uric acid handling in our case was presecretory reabsorption defect. In reported cases (table 1), 4 patients had presecretory reabsorption defect and 1 had increased secretion of uric acid. The relation between the disturbed portion and the onset of acute renal failure remains unclear.

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