Acute renal failure with severe loin pain and patchy renal ischaemia after anaerobic exercise (ALPE) (exercise-induced acute renal failure) in a father and child with URAT1 mutations beyond the W258X mutation

Sir,

ALPE is defined as (i) acute renal dysfunction or failure due to (ii) anaerobic exercise, accompanied by (iii) loin pain and (iv) normal or only slight elevation of creatine phosphokinase or serum myoglobin [1,2]. It has been reported that 51% of ALPE cases involve patients with renal hypouricaemia [1]. After cloning of the SLC22A12 gene (URAT1: uric acid transporter 1) [3], seven renal hypouricaemic patients with exercise-induced acute renal failure were analysed for mutations of URAT1 [3–6]. Five patients were homozygotes for W258X, one patient was a heterozygote for W258X and one patient was a compound heterozygote for W258X,Q297X [3–6].

We describe a father and son who both developed ALPE with renal hypouricaemia beyond the W258X mutation of URAT1.

Case 1. One day after performing a 150 m dash at a neighbourhood athletic meeting, a 40-year-old man (father) developed severe loin pain and consulted our clinic. His serum creatinine and uric acid levels were 2.9 and 2.1 mg/dl. Fractional Excretion of Uric Acid (FEUA) was 49.7% at that time. Delayed computed tomography (CT) scans after administration of contrast media demonstrated patchy wedge-shaped enhancement. He recovered from ALPE in 4 weeks, and serum creatinine and uric acid decreased to 1.0 and 0.6 mg/dl, respectively.

Case 2. Five years later, the 14-year-old son of case 1 developed acute renal failure after performing the 400 m dash twice. Three hours after the race, severe loin pain developed and he consulted a local doctor (serum creatinine 1.45 mg/dl). A non-steroidal anti-inflammatory drug (NSAID) was administered under a diagnosis of muscle pain. Three days later, he was admitted to our hospital, complaining of loin pain and oliguria. His serum creatinine and uric acid levels were 11.3 and 7.4 mg/dl, respectively. FEUA was 65.7%. ALPE was diagnosed and the patient was haemodialysed six times. He recovered from ALPE 4 weeks later (serum creatinine 1.0 mg/dl, serum uric acid 0.7 mg/dl). During the recovery stage, delayed CT demonstrated faint multiple wedge-shaped areas of contrast enhancement at a serum creatinine of 3.8 mg/dl.

Family analysis for URAT1 was performed after obtaining written informed consent for the testing of each patient. As shown in Figure 1, the father was an R90H homozygote and the son was a compound heterozygote for R90H/W258X. The mother was a heterozygote for W258X with a normal serum uric acid level, and the daughter was a compound heterozygote for R90H/W258X.

Therefore, this is the first report that ALPE developed in a patient with R90H homozygous mutation and in a patient who was a compound heterozygote for R90H/W258X mutation.

Conflict of interest statement. None declared.

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Statins and progression of renal failure: is a reconsideration of clinical practice guidelines justified?

Sir,

In the 2002 and 2003 clinical practice guidelines (CPG) [1], it is stated that there is ‘insufficient evidence to recommend lipid-lowering therapy for the purpose of slowing the progression of chronic kidney disease (CKD)’. Recently, these guidelines were questioned on the basis of post hoc