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P433 -Recurrent AKI with loin pain in a Caucasian competitive cyclist. Urate is part of the story.

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Acute kidney injury with loin pain is unusual. One cause is 'night out syndrome', AKI occurring in young adults 12-48 hours after substantial alcohol intake, sometimes with other agents (1). It has been associated with thrombi in renal arcuate veins (2). The NSAID Suprofen was withdrawn in the 1980s for unpredictably inducing pain and AKI (3). A rare cause in Europe is ALPE, 'AKI with loin pain following anaerobic exercise', a condition frequently described in Japan and Korea, but rarely elsewhere. Many isolated cases there, and most recurrent examples, are associated with genetic hypouricaemia (4).

A 20 year old Caucasian competitive sprint cyclist presented with recurrent episodes of severe bilateral loin pain following exercise. She described 12 episodes in the preceding 18 months; each one commencing within an hour or two of a training session. Competitions, involving a single effort, or two widely spaced races, never precipitated symptoms. This was her first presentation to secondary care.

Blood tests revealed AKI in the absence of significant dehydration or rhabdomyolysis. Creatinine on admission was 138µmol/L, peaking at 229µmol/L at 24 hours from her historic baseline of 79µmol/L. Creatine kinase (CK) was only marginally elevated at 254U/L (reference range 30-135). Urine dipstick showed 1+ blood. Urine culture was negative. CT-KUB demonstrated structurally normal kidneys and urinary tract. Both the loin pain and AKI improved spontaneously.

To clarify the diagnosis and management she agreed to deliberately attempt to induce her symptoms. This was successful, and accompanied by AKI with peak creatinine 118µmol/L. CT with contrast showed patchily reduced enhancement of renal parenchyma (figure 1). 12 hours later there were characteristic focal dense segmental areas of retained contrast in both kidneys. Again, symptoms and AKI settled without further intervention.

Resting fractional excretion of urate was normal, and serum concentration low-normal, but urate rose five-fold at 5 hours after anaerobic exercise (peak 0.6mmol/L, reference range 0.12-0.36mmol/L). Administration of allopurinol for 1 week prior to subsequent training sessions prevented both the rise in urate and recurrence of symptoms or AKI.

Even in ALPE without genetic hypouricaemia, increased urate production seemed to be important. However pathogenesis is not adequately explained. Does this have lessons for other painful AKI?