UNRECOGNISED ACUTE RENAL FAILURE FOLLOWING THE COMRADES MARATHON

Roal van Zyl-Smit, Phillip Mills, Louis Vogelpoel

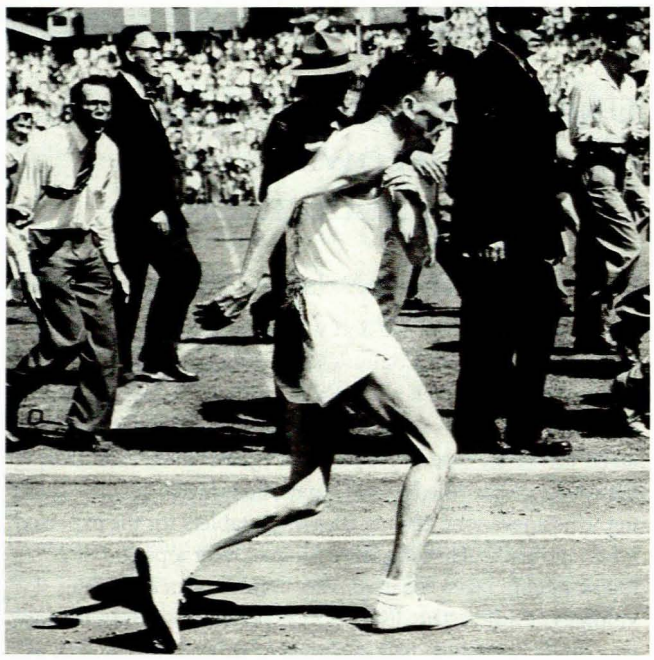
A 40-year-old experienced and well-trained marathon runner attempted his second Comrades Marathon (a 90 km ultramarathon). He felt perfectly fit and well before the race and, as was his custom, took one 2 mg loperamide (Imodium) tablet before the race as prophylaxis against diarrhoea. During the course of the race he took 6 paracetamol 500 mg/chlormezanone 100 mg (Bessenol) tablets for the expected aches and pains. He completed the race over a period of 9 hours, having taken what appears to have been adequate volumes of fluids. The discomfort experienced during the race was similar to that he had experienced during previous races.

On the day following the race he experienced bilateral loin pain for which he took another 2 Bessenol tablets, and during the following 5 days he used a total of 11 Stopayne tablets (paracetamol/caffeine/codeine phosphate/meprobamate). During the entire period following the race he had mild muscle aches and pains and did not notice any diminution in urine output.

Nine days after the marathon the patient attended a social function and was noted to have an elevated jugular venous pressure by an astute general physician also present at the function. A medical consultation was arranged for the following day.

The patient was noted to be generally well but with a blood pressure of 200/140 mmHg, pulse 60/min, heart clinically normal, bilateral basal pulmonary crackles and mild tenderness over both renal angles. His urine contained 2+ protein, 1+ blood and moderate numbers of granular casts. Plasma creatinine was 713 μmol/l, urea 32.7 mmol/l, potassium 5.5 mmol/l, lactic dehydrogenase (LDH) 568 U/l (normal range 170 - 350 U/l), alanine aminotransferase (ALT) 159 U/l (normal range 1 - 25 U/l), gamma-glutamyltransferase (GGT) 175 U/l (normal range 0 - 65 U/l), serum calcium 2.3 mmol/l, inorganic phosphate 2.08 mmol/l and serum urate 0.68 mmol/l (normal

Roal van Zyl-Smit is a consultant nephrologist at Groote Schuur Hospital, and has completed two Comrades Marathons. Louis Vogelpoel is the astute physician who noted the raised venous pressure in the case described. Philip Mills, the medical registrar involved in handling the case, is now a consultant cardiologist.



Near collapse — for runner Jim Peters these were the last moments of the 1954 Vancouver Marathon. He is obviously in serious distress, but a life-threatening situation in a distance runner can develop much less dramatically, days after a race, as in the case reported here.

range 0.12 - 5.0 mmol/l). Ultrasound investigation revealed bilaterally unobstructed and moderately enlarged kidneys.

A diagnosis of acute tubular necrosis (ATN) was made. Furosemide 250 mg was given intravenously and followed by similar oral doses. As treatment for his hypertension he received nifedipine 10 mg, 8-hourly. A diuresis was established and over the following 5 days the plasma creatinine fell to 260 μmol/l and further to 93 μmol/l over the next 10 days. His creatinine clearance had improved to 102 ml/min.

Three months later the patient completed a standard marathon uneventfully and without loss of renal function.

Discussion

The clinical picture of acute renal failure (ARF) is often dominated by its cause. When the presence of ARF goes undetected, potentially life-threatening situations may develop. Most athletes expect to feel somewhat 'unwell' after completing gruelling endurance events. These symptoms may totally obscure underlying ARF, as was demonstrated in this case.

In most situations where life-threatening hyperkalaemia or fluid overload does not supervene, ATN will fortunately resolve spontaneously over the ensuing days, with the athlete being totally unaware of his/her potentially extremely serious medical condition.

ARF related to physical exercise is generally associated with rhabdomyolysis and some degree of volume contraction.[1,2] The role of painkillers and other drugs used by athletes has not been resolved fully. Unsuspected ARF has been described in many situations, including adverse reactions to drugs, toxins, acute pancreatitis, and unsuspected rhabdomyolysis with myoglobinuria.[3] Particularly hazardous situations have been noted in assault victims sustaining what appeared to be relatively minor trauma, but returning a few days after discharge from hospital in established ARF.[4]

This patient presumably developed rhabdomyolysis and ATN following the gruelling ultramarathon. There were no obvious precipitating factors other than possibly the consumption of analgesics during and after the race. The patient did not have a renal biopsy but the rapid recovery to total normal renal function in this case is entirely consistent with a diagnosis of ATN.

While ATN after endurance exercise is frequently encountered and well recognised,[3] the prevalence of subclinical ATN has not been determined, and if missed could have lethal consequences. Athletes and doctors should be aware that oliguria, oedema, dyspnoea, or prolonged ill health following endurance exercise should be considered as potentially very serious until ARF has been excluded. They should also be warned against the use of analgesics both during and after such exercise.

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