Exertion-induced rhabdomyolysis in a patient on statin therapy

Sir,

We describe a patient on long-term statin treatment who developed acute renal failure (ARF) from rhabdomyolysis following severe unaccustomed exertion. Reviewing the available literature, we did not find reports of exertion-induced rhabdomyolysis and ARF requiring dialysis while on statins.

A 57-year-old male, with hypertension and hyperlipidaemia (on ramipril 2.5 mg, atorvastatin 10 mg daily for several years), went trekking and described it as ‘the most exhausting exercise in recent times’. Coming downhill, he noticed thigh pain and took two doses of a non-steroidal anti-inflammatory drug (NSAID). On day 3, his urine output decreased; examination showed blood pressure of 160/100 mmHg and clinical evidence of fluid overload. Investigations showed: blood urea 141 mg/dl, creatinine 9.4 mg/dl, potassium 6.4 mEq/l, mildly deranged liver functions, no proteinuria, and no urinary casts or urine eosinophils. Serum creatine kinase (CK) was elevated: 3389 IU/l (normal 38–174 IU/l), as was lactate dehydrogenase 469 U/l (140–300 U/l) and serum myoglobin 617 μg/l (5–85 μg/l); urine myoglobin was negative. Haemodialysis was performed for hyperkalaemia and volume overload. Previous medications were stopped. Urine output improved by the fourth day of hospitalization and renal functions started improving in 1 week. At 6 weeks, urea was 32 mg/dl and creatinine 1.0 mg/dl.

In this patient, treatment with statins and the development of rhabdomyolysis following exertion raises the possibility of an association between the two, either direct or indirect. Our patient had several factors predisposing to ARF, i.e. volume depletion, use of an angiotensin-converting enzyme inhibitor (ACEI) and an NSAID, and severe exertion that could by itself cause rhabdomyolysis. It is probable that treatment with statins amplified the muscle damage caused by intense exertion. We can only speculate as to whether he would have developed rhabdomyolysis if he was not taking statins.

In studies looking at statins and exertion-induced muscle damage, patients receiving lovastatin had 62–77% higher CK levels after exercise compared with those on placebo, showing that statins increase exercise-induced muscle injury [1]. In another study, several patients on statins showed a post-exercise rise in serum CK, although there was no difference in average CK levels compared with placebo [2]. A personal report documented an exercise-induced CK rise with atorvastatin; the symptoms and CK levels normalized on stopping statins and reappeared after switching to pravastatin [3]. Theories of statin-induced rhabdomyolysis include effects on muscle cholesterol synthesis and isoprenoid levels [4]. Statins inhibit GTP activation. Exercise may unmask the effects of statins on skeletal muscle because GTP-dependent protein kinase pathways are important in muscle recovery following exercise [4].

Based on the association noted, it needs to be considered whether statins should be withheld prior to engaging in ‘more than an accustomed range’ of physical exertion. Most statins have a short half-life and withholding the drug for 2 days would be reasonably intelligent to avoid its contribution to muscle injury [5].

Conflict of interest statement. None declared.

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Acute nephropathy due to Salmonella typhimurium septicaemia

Sir,

Salmonella typhimurium (STM) is a non-typhoid salmonella which essentially entails intestinal infections. Extra-intestinal involvements such as septicaemia or renal lesions are uncommon in developed countries and mainly occur in frail people [1]. We describe a case of STM septicaemia with acute nephropathy in an elderly subject.

A 92-year-old man was hospitalized for epigastralgia; vomiting and fever of 40°C suddenly appeared. His medical history consisted of non-insulin-dependent diabetes, deep-vein thrombosis, chronic respiratory insufficiency, mild chronic renal insufficiency, a pacemaker and chronic alcoholism. He had been treated by omeprazole, fluindione, furosemide, gliclazide, molsidomine, trinitrine, ramipril, and allopurinol. He had alcoholism. He had been treated by omeprazole, fluindione, furosemide, gliclazide, molsidomine, trinitrine, ramipril, trimetazidine and sertraline. Except for a mild abdominal pain without contracture, the physical examination was normal. He was hypertensive and had 2+ pitting oedema of the lower extremities. The patient had an haemoglobin of 10.5 g/dl, white cell count 13,000/μl, C reactive protein (CRP) 1.0 mg/dl. Nephrol Dial Transplant (2005) 20: 244–247