In April, 2007, a 47-year-old man presented with a short history of severe muscle ache and dark-brown discolouration of his urine. He had enjoyed a brisk walk on Haystacks, one of the fells in the Lake District, 2 days earlier. There was no medical history of note, he was on no medication, and denied illicit drug use. He was a foreman in a paper mill and exercised frequently. Physical examination showed tender quadriceps muscles. Laboratory test results showed a creatine kinase (CK) of 277 480 U/L (normal <170). A diagnosis of rhabdomyolysis was made. Forced alkaline diuresis was initiated but oliguric renal failure ensued. Continuous veno-venous haemofiltration was started in the intensive care unit. Renal function eventually recovered and after 10 days of treatment haemofiltration was stopped. The patient was discharged in good health (creatinine 182 μmol/L) after 3 weeks in hospital.

When seen for follow-up in July, 2007, he was well and denied muscle pain. CK was high at 395 U/L and serum creatinine concentration was normal. Further history-taking elicited a casual comment from the patient that since the age of 30 years, he had developed muscle pain and brown urine every time he walked on the fells. We suspected a metabolic myopathy, and biopsy of the quadriceps was done; histology was normal on light microscopy. Staining for glycogen and lipid distribution, ATPase histochemistry, and myophosphorylase gave normal results. Mitochondrial histochemistry was normal. A metabolic disorder was still suspected, and the patient was referred to the supra-regional unit for mitochondrial diseases. Analysis of blood acylcarnitines showed the typical pattern of intermediates for deficiency of very-long-chain acyl-CoA dehydrogenase (VLCAD), a rare disorder of mitochondrial fatty-acid oxidation. We advised our patient not to exercise for long periods while fasting or unwell. Dietary advice was to limit fat intake to 40–60 g per day and to have extra carbohydrates before exercise. When last seen in January, 2008, the patient was well and had had no episodes of muscle pain although he had avoided the fells.

The most common cause of rhabdomyolysis is trauma, and medications, illicit drugs, endocrine disorders, and inherited myopathies are less frequently encountered. Recurrent rhabdomyolysis after exercise must prompt further investigation for an underlying metabolic myopathy, including analysis of acylcarnitines. Fatty acids are needed for long-term exercise, whereas glucose and complex carbohydrates are needed for short bursts of exercise. Patients with inherited abnormalities of fatty-acid metabolism develop symptoms after prolonged exercise or fasting. Inborn errors of mitochondrial β-oxidation, which commonly present with rhabdomyolysis, include carnitine palmitoyltransferase deficiency and VLCAD deficiency. The latter disorder is classified into three forms: a severe early-onset form with cardiomyopathy and high mortality; an intermediate more benign form with childhood onset; and an adult-onset form, as seen in our patient. The latter features isolated skeletal muscle involvement after exercise or fasting. In a series of 54 patients, 25 had the severe childhood form, 21 had the milder childhood form, and eight had the myopathic adult form with onset after age 13 years. Adult onset is associated with a good prognosis. In our patient, life-threatening rhabdomyolysis developed after a brisk walk on Haystacks (figure), the famous mountain of Lakeland walker and guidebook author Alfred Wainwright (1907–91). Wainwright commented that, as well as being beautiful, Haystacks was “a place of surprises around the corner”. A casual remark by our patient indeed led to a surprise around the corner, a search for the proverbial needle in a haystack, and a diagnosis of mitochondrial myopathy.

Contributors
OJ wrote the report. OJ, DMT, and AW were involved in caring for the patient. DMT made the diagnosis. All authors contributed to the report.

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