Acute myopathy in a patient with oesophageal stricture

LUCIA POSSAMAI, WILLIAM STEPHEN WARING
The Royal Infirmary of Edinburgh, Edinburgh, UK

Address correspondence to: W. S. Waring. Tel: (+44) 131 242 1385; Fax: (+44) 131 242 1387. Email: s.waring@ed.ac.uk

Abstract
Background muscle injury may be caused by any one of a number of factors, including trauma, drugs, hyperthermia and metabolic derangement. Symptoms and signs are often non-specific, and myopathy may be easily overlooked.
Case an elderly woman was referred to hospital for investigation of rapidly declining mobility in the setting of anorexia and vomiting due to benign oesophageal stricture. The patient had generalised muscle weakness and tenderness. Investigations showed severe metabolic alkalosis and hypokalaemia, and creatinine kinase (CK) activity was grossly elevated at 40,000 U/l.
Outcome CK activity remained elevated for several days, and a diagnosis of polymyositis was considered. However, muscle tenderness resolved and CK activity declined after correction of the underlying metabolic disturbances. Acute myopathy was attributed to hypokalaemia.
Conclusions hypokalaemia is an important cause of acute myopathy, and older patients may present with non-specific symptoms. A high degree of clinical suspicion is needed to establish the diagnosis. The clinical features and the pathogenesis of hypokalaemia-induced myopathy are reviewed.

Keywords: creatine kinase, hypokalaemia, oesophageal stricture, rhabdomyolysis, elderly

Case Report
A 73-year-old woman was referred to hospital for investigation of progressive malaise and generalised muscle weakness over 2 days. She had suffered dysphagia for solid foods during the preceding 4 weeks, associated with intermittent vomiting. Past history was reflux oesophagitis 3 years ago, and intermittent leg cramps. Her only medication was quinine sulphate. Examination findings were dry mucous membranes, cool peripheries, blood pressure 136/80 mmHg, resting heart rate 94 min⁻¹ and generalised tenderness overlying proximal muscle groups of upper and lower limbs. Initial investigations were serum sodium 142 mmol/l, potassium 1.4 mmol/l, calcium 2.12 mmol/l, albumin 36 g/l, creatinine 62 µmol/l, bicarbonate 56 mmol/l, and creatinine kinase (CK) 39912 U/l. Serum osmolality was 290 mOsm/kg, and urine sodium 223 mmol/l and osmolality 536 mOsm/kg. Thyroid stimulating hormone and free thyroxine concentrations were within normal limits; troponin I concentration was <0.2 µg/l, and a 12-lead electrocardiogram was normal.
Intravenous 0.9% saline was administered with cautious potassium supplementation. Endoscopy found a narrow oesophageal stricture at 25 cm that was treated by dilatation, and histological examination of oesophageal biopsy material showed mild inflammation only. Serum bicarbonate and potassium concentrations gradually corrected over 72 h, and the patient’s clinical condition improved. Serum CK activity remained substantially elevated, and a diagnosis of polymyositis was considered. However, CK activity began to decline after the underlying metabolic derangements had been corrected (Figure 1). The patient made a full recovery, and was discharged home.

Discussion
Anorexia and vomiting may cause profound electrolyte disturbance in elderly patients, and symptoms related to myopathy may be vague [1]. This case highlights the
Hypokalaemia and myopathy

A limitation of this report is that CK isoenzyme analyses were not available. Despite this, we conclude that skeletal muscle is the most likely source of the raised CK in view of the patient’s presenting symptoms, and normal cardiac investigations. A further weakness is that a causal relationship between hypokalaemia and myopathy cannot be proven. Nonetheless, we could find no plausible explanation for muscle injury in this patient, and our hypothesis was supported by the temporal relationship between correction of hypokalaemia and initial decline in CK activity.

We are reminded that acute myopathy may present with non-specific symptoms in elderly patients. The diagnosis requires a high degree of clinical suspicion, and confirmation of raised CK activity. Hypokalaemia is a recognised cause of myopathy, and correction of serum potassium concentrations allows resolution of symptoms and biochemical markers of muscle injury.

Key points
• Hypokalaemia is an independent cause of reversible acute myopathy.
• Electrolyte disturbance should be considered as a potential cause of acute myopathy and raised creatine kinase activity in older patients.
• Correction of serum potassium concentration allows resolution of symptoms and biochemical indices of muscle injury.

Conflicts of interest
No conflicts of interest

References

Received 8 January 2007; accepted in revised form 24 May 2007