

Increased Serum Creatine Kinase

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CASE DESCRIPTION

A 38-year-old white male taxi driver was referred to a lipid clinic by his general practitioner for management of combined hyperlipidemia. He was a smoker with a 20 pack-year history. His father had died of myocardial infarction at age 55 years. Examination of this patient revealed a blood pressure of 136/99 mmHg and a body mass index of 29 kg/m², with no clinical signs of hyperlipidemia. Cholesterol testing revealed a total serum cholesterol value of 251 mg/dL (6.5 mmol/L; reference interval, 3.7–7.0 mmol/L), an HDL concentration of 30.9 mg/dL (0.8 mmol/L; reference interval, 0.7–1.8 mmol/L), and triglycerides of 539 mg/dL (6.1 mmol/L; reference interval, <1.7 mmol/L), results consistent with metabolic syndrome. The patient's calculated 10-year cardiovascular risk was >20%, indicative for primary prevention of hyperlipidemia with a statin, after changes in lifestyle (1). Baseline biochemical investigations before starting the statin showed a serum creatine kinase (CK)⁴ activity of 889 U/L (reference interval, 24–195 U/L) and an alanine aminotransferase activity of 61 U/L (reference interval, <50 U/L). The results of other tests were within their respective reference intervals: total bilirubin, 0.8 mg/dL (13 μmol/L; reference interval, <14 μmol/L); alkaline phosphatase, 95 U/L (reference interval, <150 U/L); and γ-glutamyltransferase, 49 U/L (reference interval, <50 U/L). The results of other laboratory investigations (including renal function, blood count, serum vitamin B₁₂, folate, serum protein electrophoresis, antinuclear antibodies, C-reactive protein, and thyroid function) were normal. At 30 years of age, the patient's mother had been diagnosed with lower limb muscle weakness, which made her unable to dorsiflex her feet. His 48-year-old brother and 38-year-old maternal half-brother were both fine.

On questioning, the patient admitted to a weekend of heavy physical activity before the test but had no other major complaints.

Reference

1. Joint British Societies. JBS 2: Joint British Societies' guidelines on prevention of cardiovascular disease in clinical practice. *Heart* 2005;91(Suppl 5):v1–52.

Questions to Consider
<ul style="list-style-type: none">• What conditions can cause increased serum CK?
<ul style="list-style-type: none">• What other testing would be useful to help determine the cause of the increased CK?
<ul style="list-style-type: none">• Given this patient's family history, what condition should be considered to explain the mothers' symptoms and the patient's increased CK?

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the February 2014 issue of *Clinical Chemistry*. To view the case and comments online, go to <http://www.clinchem.org/content/vol60/issue2> and follow the link to the Clinical Case Study and Commentaries.

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