

A 61-Year-Old Woman with Muscle Fatigue and Increased Cardiac Troponin

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CASE DESCRIPTION

A 61-year-old woman presented with a 12-month history of progressive muscle weakness, especially involving the proximal limb muscles. Her symptoms were associated with muscular fatigue such that she was compelled to sit and rest after walking only 30–50 m. She reported difficulty climbing stairs and standing from a sitting position. There was no rash or Raynaud phenomenon but she did report some episodes of feeling hot and perspiring. She denied any paresthesia, headache, or chest pain. Her past history included sigmoid diverticular disease, osteoarthritis of the cervical spine, and depression for over 20 years with psychotic features in the last 2–3 years. Her medications were aripiprazole 5 mg daily, irbesartan 150 mg daily, amisulpride 150 mg daily, imipramine 50 mg nocte, lercanidipine 10 mg daily, and sertraline 300 mg daily. She had also recently started replacement therapies for B12 and vitamin deficiencies. Salient examination findings included a general reduction in muscle power, proximal greater than distal, with intact reflexes and sensation.

Initial investigation included an erythrocyte sedimentation rate of 32 mm/h [reference interval (RI) <20], creatine kinase (CK) activity of 1900 U/L (RI <140), negative screen for antinuclear and extractable nuclear antigen antibodies, and a negative myositis autoantibody panel. The patient was biochemically euthyroid [thyroid stimulating hormone 1.95 mU/L (RI 0.1–5.0)] and had no evidence of renal dysfunction, with creatinine of 0.75 mg/dL [66 μmol/L; RI 0.51–1.02 mg/dL (45–90 μmol/L)]. MRI of the right thigh did not show features of myositis. The patient was admitted to the hospital for a trial withdrawal of her antipsychotic medication but after 5 days there was no change in her condition. CK remained increased at 510 U/L with a CK-MB fraction of 11.8% (RI <4%). Cardiac troponin T (cTnT) was 83 ng/L (99th percentile <14) measured using the highsensitivity cTnT (hs-cTnT) assay (Roche Diagnostics). The electrocardiogram showed normal sinus rhythm and was otherwise unremarkable. Transthoracic echocardiography showed normal left ventricular size and systolic function without segmental abnormalities. Right ventricular function was normal. There were minor, nonspecific degenerative valve abnormalities only. Estimated pulmonary artery pressures were mildly increased but there was no pericardial effusion.

Measurement of cardiac troponin I (cTnI) was 2 ng/L (99th percentile in females <16) with a highsensitivity cTnI (hs-cTnI) assay (Abbott Diagnostics). The following day paired samples revealed hs-cTnT as 82 ng/L, and hs-cTnI as 4 ng/L. Testing for cTnT was repeated with the hs-cTnT assay after treatment with heterophile antibody blocking agents and remained increased.

Cardiac MRI (CMR) using steady-state-free precession cine imaging, T2 imaging, and late gadolinium enhancement according to the Lake Louise Criteria for CMR in Myocarditis (1)

showed normal left ventricular mass, dimensions, and function. There was no CMR evidence of myocarditis or myocardial edema, nor any late gadolinium enhancement, with normal T2 ratio and normal postcontrast signals. There was mild, nonspecific enhancement of the pericardium with gadolinium.

A skeletal muscle biopsy was performed.

QUESTIONS TO CONSIDER
<ul style="list-style-type: none"> • What are the potential causes of increased cardiac troponin concentrations other than acute myocardial infarction?
<ul style="list-style-type: none"> • What are possible mechanisms of discrepant concentrations of troponin T and troponin I?
<ul style="list-style-type: none"> • Is it possible for cardiac troponin to be released from skeletal muscle?

Reference

1. Friedrich MG, Sechtem U, Schulz-Menger J, Holmvang G, Alakija P, Cooper LT, et al. Cardiovascular magnetic resonance in myocarditis: a JACC white paper. *J Am Coll Cardiol* 2009;53:1475–87.

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the January 2017 issue of *Clinical Chemistry*. To view the case and comments online, go to <http://www.clinchem.org/content/vol63/issue1> and follow the link to the Clinical Case Study and Commentaries.

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