Persistent Increase of Cardiac Troponin I in Plasma without Evidence of Cardiac Injury

Lindsay A. Legendre-Bazydlo,[1] Doris M. Haverstick,[1] Jamie L.W. Kennedy,[2] John M. Dent,[2] and David E. Bruns[1*]

[1] Department of Pathology and [2] Division of Cardiovascular Medicine, Department of Medicine, University of Virginia School of Medicine and Health Science Center, Charlottesville, VA.
* Address correspondence to this author at: Department of Pathology, University of Virginia Health Science Center, Charlottesville, VA 22908. Fax 434-924-2574; e-mail DEB6J@virginia.edu.

CASE

A 69-year-old man with diabetes mellitus type II, hypertension, dyslipidemia, and prior ischemic strokes presented to the emergency department with complaints of balance difficulties and inability to stand unassisted of 2 weeks' duration. The patient's home medication regimen included atenolol, lisinopril, amlodipine, metformin, and glipizide. He is a retired chef and a former smoker (20 pack-years). He has 2 brothers, both of whom had myocardial infarctions in their 50s. The patient's physical examination was remarkable for frequent premature contractions, left lower extremity weakness, and impaired coordination. His electrocardiogram revealed sinus rhythm with frequent premature ventricular contractions and diffuse nonspecific T-wave abnormalities.

Results of a comprehensive metabolic chemistry panel were within the reference intervals except for increases in glucose (158 mg/dL; reference interval, 74–99 mg/dL) and creatinine (1.5 mg/dL; reference interval, 0.7–1.3 mg/dL). The hemoglobin A_{1c} value was 7.4% (reference interval, <6.0%). Cardiac troponin I (cTnI) concentrations were increased at 0.27, 0.22, and 0.25 μg/L (Abbott Architect assay; 99th percentile, <0.03 μg/L) over a span of approximately 8 h. The patient was admitted to the cardiology service on the basis of these abnormal results.

A transthoracic echocardiogram revealed a preserved left ventricular systolic function with evidence of impaired diastolic filling. A cardiac catheterization evaluation revealed an ulcerated plaque in the left anterior descending artery with >70% stenosis, which was treated with a bare-metal stent. The presenting symptom of balance difficulty failed to resolve after the coronary intervention, and the patient remained unable to stand unassisted. A neurologic evaluation included magnetic resonance imaging, which found no evidence of an acute stroke. He was discharged with a diagnosis of orthostatic hypotension.

Three months later, the patient presented to the emergency department with several days of balance difficulty. The initial cTnI value was increased at 0.10 μg/L, prompting admission to the cardiology service. Over the subsequent 12 h, cTnI values for 2 additional samples remained stable at 0.10 μg/L. Other laboratory tests included a comprehensive metabolic panel and a complete blood count, with results within reference intervals except for a hemoglobin concentration of 12.5 g/dL (reference interval, 14.0 - 8.0 g/dL) and a hematocrit of 35.5% (reference interval, 40.0%–52.0%). The patient's symptoms were not consistent with a cardiac etiology, and no further cardiac evaluation was pursued. A neurologic evaluation again found no evidence of acute stroke, and his symptoms were believed to reflect a combination of orthostatic hypotension, pontine

DOI: 10.1373/clinchem.2009.138164

gliosis, and cerebellar atrophy. The unexplained abnormal troponin results prompted the attending cardiologist to contact the director of clinical chemistry.

Questions to Consider
• Describe common pathologic reasons for increased plasma concentrations of cardiac troponins in the absence of an acute coronary syndrome.
• What analytical interferences increase measured cTnI concentrations?
• How would you investigate the abnormal cTnI results in this patient?

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the May 2010 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol56/issue5 and follow the link to the Clinical Case Study and Commentaries.

Educational Centers

If you are associated with an educational center and would like to receive the cases and questions 2-3 weeks in advance of publication, please email clinchem@aacc.org.

All previous Clinical Cases Studies can be accessed and downloaded online at http://www.aacc.org/resourcecenters/casestudies/ .

DOI: 10.1373/clinchem.2009.138164