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Authors Lin, Trevor Vampola, Stephen P.

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High-Risk Unstable Angina in the Era of High-Sensitivity Troponin Assays

Trevor Lin and Stephen P. Vampola, MD

Case Report

A 54-year-old male with hypertension, hypercholesterolemia and gastroesophageal reflux disease presented to the emergency department complaining of substernal chest pain at rest. The patient reported 2 months of chest pain while jogging on a treadmill. However, 2 hours prior to the arrival, he noted similar pain at rest. The pain was constant, pressure-like and nonradiating with an intensity of 2 out of 10. There was moderate nausea and dry cough associated. He denied shortness of breath, vomiting, abdominal pain, fevers and chills. He contracted SARS-CoV-2 several weeks ago, after his chest discomfort started. However, his chest pain persisted and progressed after other COVID symptoms resolved. His hypertension was treated with two agents and he was not taking medications for hypercholesterolemia. Prior labs were remarkable for an LDL cholesterol of 178 mg/dL. Family history was significant for a father with treated hypercholesterolemia and mitral valve prolapse.

Initial vital signs included blood pressure of 142/100 mmHg and a heart rate of 78 bpm. He was given nitroglycerin and aspirin and his pain resolved. An electrocardiogram showed normal sinus rhythm and chest X-ray was unremarkable.

Laboratory results included serial high-sensitivity cardiac troponin (hs-cTn) T assays at the 0 and 3 hours of 9 ng/L and 7 ng/L, respectively (normal range < 15 ng/L). Following the hscTn results, the patient remained pain free and was discharged home with outpatient follow-up as with a cardiologist.

After outpatient evaluation, he underwent urgent stress echocardiography and initiated medical therapy with aspirin and a high-intensity statin. Stress echocardiography revealed stressinduced regional hypokinesis suggestive of ischemia in the left anterior descending artery (LAD) territory. His ejection fraction was normal and no structural heart disease was noted. He underwent coronary angiography, which demonstrated a subtotal occlusion in the proximal segment of the LAD, resulting in Thrombolysis in Myocardial Infarction (TIMI) Flow Grade of 1 (incomplete filling of the distal vessel). He also had severe stenosis in a large diagonal artery, the ramus intermedius, the left circumflex and the right posterolateral branch.

Discussion

When obstructive coronary artery disease is suspected, it is essential to differentiate acute coronary syndrome (ACS) from

stable ischemic heart disease (SIHD). ACS, which consists of myocardial infarction (MI) and unstable angina (UA) requires urgent or emergent inpatient management, while the initial focus of SIHD is on lifestyle modification, medical therapy and symptom management.¹ MI diagnosis is aided by objective electrocardiogram changes or elevations in biomarkers such as cardiac troponins, which represent necrosis of the myocardium.² UA, on the other hand, is a clinical diagnosis that is made when a patient presents with prolonged rest angina, significant new-onset angina or angina that is increasing in severity. Historically, UA has included a group of patients with the same biology as MI, but with insufficient myocardial necrosis be detected by available biomarker assays.³

Prior, biomarker assays used in the diagnosis of MI include myoglobin, creatinine kinase and cardiac troponins.⁴ The first two lack specificity to the myocardium, and historical assays for all three lacked sensitivities to detect small levels of injury. In recent years, high-sensitivity cardiac troponin (hs-cTn) assays have been used routinely and are capable of detecting serum troponin concentrations of less than 10 ng/L.⁵ These highly-sensitive tests have become the cornerstone of early evaluation of patients with suspected ACS in the emergency department.

The availability of hs-cTn has called into question the meaning of UA diagnoses.^{6,7} Many advocated that the subset of UA patients with critical coronary stenosis or acute coronary injury should present with detectable elevated cardiac troponin hs-cTn. "Early discharge" strategies were developed to send patients with suspected ACS home after clinical evaluation and a single hs-cTn measurement.⁸

Our case illustrates the importance of clinical evaluation in assessing patients who present with chest pain. History and physical exam remain relevant in the era of hs-cTn. Our patient was initially incorrectly evaluated as low-risk based on his biomarkers when he presented to the emergency department, despite well-known high-risk features of UA in his clinical presentation. When he did undergo angiography, as an outpatient, it was evident that he had critical stenosis in a high-risk location as evidenced by TIMI 1 flow in his LAD.

This case reinforces that, with the implementation of hs-cTn assays, clinical evaluation and judgment remain relevant in identifying high-risk patients with ACS. When features of the

patient's history are not obvious, clinical prediction tools, such as the HEART Score remain helpful in initial risk stratification.⁹ Despite advanced diagnostic technology, the role of the clinician in the evaluation of chest pain remains paramount.

Case Outcome

The patient was admitted to the hospital and underwent successful coronary artery bypass surgery (CABG). He tolerated the procedure well and has no postoperative complications. Following initial cardiac rehabilitation, he remains asymptomatic.



Image 1: Coronary angiogram showing multivessel coronary artery disease with subtotal stenosis of the left anterior descending artery (arrow) in left (A) and right (B) anterior oblique views.

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