ORIGINAL ARTICLE

TROPONIN-I POSITIVITY IN PATIENTS REFERRED TO RAPID ACCESS CHEST PAIN CLINIC

Umar Hayat, Motwani J*, Burrell CJ*

Department of Cardiology, Ayub Medical College, Abbottabad, Pakistan, *Derriford Hospital, Plymouth, UK

Background: Rapid Access Chest Pain Clinics (RACPCs) are set up to access patients with new onset chest pain (within the preceding three weeks), of possible cardiac origin. These patients are seen in the clinic within two weeks of referral and the attending physician takes a history, performs a routine clinical examination, and if clinically justified, a treadmill exercise test is performed according to Bruce Protocol. Within the group of patients referred to the RACPC with new onset but otherwise stable angina, there is a potential overlap with patients who in fact may have an evolving acute coronary syndrome, i.e., unstable angina. The aim of this study was to assess the prevalence of Troponin-I positivity as an indicator of acute coronary syndrome. **Methods:** This cross-sectional descriptive study included 60 consecutive patients referred to the RACPC with history of recent onset chest pain (within the last three weeks) of possible cardiac origin and positive ETT or confirmed abnormal ischemic ECG at baseline. Troponin-I was measured in these patients. **Results:** Out of the total 60 patients, 8.33% of the patients referred to RACPC with new onset angina had positive cTnI. **Conclusion:** Point of care test (POCT) for cTnI can help to identify the high risk patient referred to RACPC.

Keywords: Troponin-I, cTnI, Troponin-T, Rapid Access Chest Pain Clinic, Angina, Unstable angina, Myocardial infarction, Non-ST elevation MI, Acute coronary syndrome

INTRODUCTION

Various definitions of unstable angina have been proposed, but in 1989, Braunwald devised a classification system to ensure uniformity of categorisation, as well as diagnostic and prognostic information.[1] This system is used to classify angina according to the severity of the clinical manifestations, defined as acute angina while at rest (within the 48 hours before presentation), sub-acute angina while at rest (within the previous month but not within the 48 hours before presentation), or new onset of accelerated (progressively more severe) angina; the clinical circumstances in which unstable angina develops, defined as either angina in the presence or absence of other conditions, or angina within two weeks after an acute myocardial infarction; and whether or not electrographic abnormalities are present. [1]

The term 'acute coronary syndrome' has been used to describe the spectrum of condition that includes unstable angina, non-ST elevation myocardial infarction (NSTEMI), and ST elevation myocardial infarction (STEMI). Patients with NSTEMI and unstable angina present in similar manner and the pathophysiology of an acute coronary event includes systemic and intracoronary activation of both the platelets and the distinction between two is made only with the results of cardiac markers. [1]

Rapid Access Chest Pain Clinics (RACPC) are designed to evaluate patients referred by their general physicians (GP's) with new onset chest pain of possible cardiac origin. The aim of chest pain clinics is to establish rapid access to provide a diagnosis, treatment and follow-up plan for each patient, and to optimise the use of hospitalisation for appropriate patients.

Exercise test is a diagnostic method frequently employed in patients with chest pain.[2] The exercise stress test (EST) is the most important non-invasive investigation for patients with angina pectoris. It can provide objective evidence of presence of disease, and is an independent predictor of cardiovascular death. It is cheap and has a widespread clinical use for several decades. The incidence of death and/or myocardial infarction during and just after exercise test has been reported to be as high as 1 in 2,500 tests.[3] Multiple surveys confirm that as many as 10 MIs or death may be expected per 10,000 tests in those with coronary artery disease (CAD). A review summarising 8 studies of estimates of sudden death during exercise testing revealed rates from 0.0 (4 studies) to 5 per 100,000 test.[4]

Sensitivity and specificity of exercise-induced ST segment depression can be determined by comparing the results of exercise testing and coronary angiography. From these studies, it can be seen that the exercise test cut of 0.1 mV (1 mm) of horizontal or down sloping ST segment depression has 84% specificity for angiographically significant CAD, i.e., 84% of those without significant angiographic disease had a normal exercise test. These studies had a mean sensitivity of 66% for significant angiographic CAD, with range of 40% to 90% for 1-vessel disease to 3-vessel disease.[5]

Cardiac specific troponins are useful not only because they come close to fulfilling many of the criteria for an ideal biological marker, but they also convey prognostic information and can help frame

therapeutic decisions regarding patients with acute coronary syndrome.[5]

In majority of cases, unstable angina is due to compromise of the myocardial blood flow caused by disruption of the atherosclerotic plaque and associated thrombus formation. After the loss of integrity of cardiac myocytes membranes, intracellular macromolecules (cardiac troponins) are released into the cardiac interstitium, lymphatics, and microvasculature; eventually they are detected in the peripheral circulation.[6]

Troponin-T positivity has been assessed during exercise test in patients with stable angina pectoris.[6] Troponin was not positive in any patient with normal coronary arteries. In this study of 100 patients only four had positive troponins, two had positive ETT and two had negative ETT. In another study exercise –induced severs ischemia did not result in the elevation of plasma levels of TnT in patients with chronic CAD.[7]

Cardiac specific troponin-I levels predict the risk of mortality in patients with acute coronary syndrome.[8] Cardiac troponins are highly sensitive and specific tests for myocardial cell necrosis and injury. Consequently, the American College of Cardiology/ American Heart Association[9] guidelines as well as the European Society of Cardiology Task Force Report[10] have incorporated troponin measurement into their algorithms for patients with acute coronary syndrome.

MATERIAL AND METHODS

This cross-sectional descriptive study included 60 consecutive patients referred to the RACPC with history of recent onset chest pain (with in the last three weeks) of possible cardiac origin and positive ETT or confirmed abnormal ischemic ECG at baseline.

Informed consent was obtained from all the patients. Clinically stable patients who were able to perform ETT, and the Consultant in charge agreed with the interpretation of ETT result being positive were included.

Patients with history of prolonged chest pain, unstable angina, and significant valvular heart disease were excluded.

Patients undertook exercise stress test according to Bruce protocol. Stress test was deemed positive if there was 2 mm or more ST depression, ST elevation, systolic blood pressure (SBP) falling >20 mm Hg, exercise induced SVT, VT and multifocal VE's.

Five ml of blood sample was taken from the ante-cubital vein observing universal precaution of venipuncture and obtaining informed consent at the end of ETT. The blood was sent to the hospital laboratory in vacutainer for quantitative determination of cardiac troponin-I (cTnI) in serum using ADVIA Centaur System.

Patients' serum was analysed after allowing the samples to clot adequately before centrifugation. ADVIA Centaur cTnI Ready Pack primary reagent was used. The ADVIA Centaur cTnI assay is two-site sandwich immunoassay using direct chemiluminometric technology, which uses constant amounts of polyclonal and monoclonal antibodies. Patients were put on list for coronary angiography as per protocol of RACPC.

RESULTS

Sixty patients were included in this study. All, except one who had baseline ECG consistent with ischemia, had positive ETT, and blood was taken for analysis of cardiac troponin-I. The demographic and baseline characteristics of the selected patients are shown in Table-1.

Five (8.33%) out of 60 patients attending RACPC showed positivity for troponin-I, suggesting a significant overlap of what is said to be new onset angina and acute coronary syndrome based on their history of cardiac sounding chest pain, positive ETT and raised troponin-I.

Nine (15%) patients out of sixty had ETT positive in stage 1.

Table-1: Demographic and baseline characteristics (n=60)

Characteristics	Number	%
Mean Age (Years)	62.48	
Male	36	60
Female	24	40
Diabetics	6	10
Hypertension	30	50
Family history +ve	20	33
Hypercholesterolemia	20	33
Smoking history		
Ex-smokers	18	30
Current smokers	8	13
Non-smokers	34	57
Taking Beta-blockers	20	33
Statins	22	37
Nuseal Aspirin	12	20
Mean ETT time (Min)	5.63	
Admitted after ETT	7	11
Angiogram performed	20	33
Normal	3	
CABG	4	
PCI	10	
Troponin-I Level		
0.0–0.16 µg/L	55	91.66
>0.16 µg/L	5	8.33

DISCUSSION

Slightly above 8% of the patients referred to RACPC with new onset angina had positive cTnI in our patients. The troponin-I positivity cannot be predicted with certainty on the basis of exercise time. Five out of sixty patients who had positive cTnI, two had ETT positive in stage 1, one in recovery after reaching stage 4, and one had baseline ECG changes.

Patients with early positive ETT are more likely to be troponin-I positive. Fifteen percent of our patients had ETT positive in stage 1.

The mortality in this group of patient was not assessed due to the limitations of the study. Based on prognostic value of cTnI this group may represent high risk group.[8]

CONCLUSION

Point of Care Test for cTnI can help to identify the high risk patient referred to RACPC. Larger studies are needed to confirm these findings.

REFERENCES

1. Braunwald E. Unstable angina: a classification. Circulation 1989;80:410–14.
2. Chaitman BR. Exercise stress testing. In: Heart Disease. Braunwald E, ed. Philadelphia: WB Saunders Company; 1997.p.153–76.
3. Stuart RJ, Ellestad MH. National survey of exercise testing facilities. Chest 1980;77:94–7.
4. Gordon NF, Kohl HW. Exercise testing and sudden death. J Cardiopulm Rehabil 1993;13:381–6.
5. Gianrossi R, Detrano R, Mulvihill D, Lehmann K, Dubach P, Colombo A, *et al*. Exercise-induced ST depression in the diagnosis of coronary artery disease, a meta-analysis. Circulation 1989;80:87–98.
6. Eryol N.M, Basar E, Oguzhan A, Çiçek Y, Abaci A, Oguzhan A, *et al*. Should Troponin-T be assessed during exercise stress testing in patient with angina pectoris. Anadolu Kardiyol Derg 2002;2(2):132–7.
7. Akdemir I, Aksoy N, Aksoy M, Davutoglu V, Dinckal H. Does exercise-induced severe ischaemia result in elevation of plasma troponin-T level in patients with chronic coronary artery disease? Acta Cardiol 2002;57(1):13–8.
8. Antman EM, Tanasijevic MJ, Braunwald E, Schactman M, McCabe CH, Cannon CP, *et al*. Cardiac-specific troponin I levels to predict the risk of mortality in patients with acute coronary syndrome. N Engl J Med 1996;335:1342–9.
9. Anderson JL, Adams CD, Antman EM, Bridges CR, Califf RM, Casey DE Jr, *et al*. Guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction. J Am Coll Cardiol 2007;50(7):e1–e157
10. Bertrand ME, Simoons CML, Fox KAA, Wallentin LC, Hamm CW, McFadden E, *et al*. Recommendation of the Task Force of the European Society of Cardiology on management of Acute Coronary syndrome without ST-elevation. Eur Heart J 2000;21:1406–32.

Address for Correspondence:

Dr. Umar Hayat, Department of Cardiology, Ayub Teaching Hospital, Abbottabad, Pakistan. **Cell:** +92-333-5066000
Email: omerhayat@hotmail.com