

# Clinical presentation and ECG changes - how good is it in diagnosing troponin positive acute coronary syndrome

Anthony Leslie Innasimutthu, Sandeep Shivananada Siddhi\*, Gopala Krishna Balappa Rao\*\*

University Hospital Aintree, Liverpool

\*Department of Acute Medicine, Royal Albert Edward Infirmary, Wigan

\*\*Department of Cardiology, University Hospital Aintree, Liverpool, UK

## ABSTRACT

**Objectives:** To study the relation between troponin positive acute coronary syndrome (ACS) and electrocardiogram (ECG) changes on admission.

**Methods:** It was a prospective cohort study looking at patients admitted to the Heart Assessment Center over a period of a month, who were suspected to have an ACS. There were 126 patients in the cohort. The groups were classified depending on the number of cardiovascular risk factors: 0, 1, 2, 3, 4 and >4. The history and ECG changes during presentation were analyzed and the cardiac enzymes (12-hour troponin) were done. The final diagnosis was based on the expert opinion of the Consultant Cardiologist in combination with troponin positive results, and they were only further studied.

**Results:** Of the 126 patients that were analyzed 31(25%) were diagnosed as ACS of which 26(21%) were troponin positive ACS. Among them, 13(50%) had ECG changes during admission, and 13(50%) did not have any noticeable ECG changes.

**Conclusion:** Certain elements of the chest pain history and ECG changes are associated with increased or decreased likelihood of a diagnosis of ACS, none of them alone or in combination identify a group of patients that can be safely discharged without further diagnostic testing.

(*Anadolu Kardiyol Derg 2007; 7 Suppl 1; 168-70*)

**Key words:** acute coronary syndromes, troponin T, electrocardiography

## Introduction

Patients with suspected acute coronary syndromes (ACS) constitute a heterogeneous population with variable outcomes. Risk stratification in this population of patients is difficult due to the complexity in patient risk profile. Acute coronary syndrome is one of the most severe forms of heart disease, and is the most frequent cause of morbidity and mortality in the developed world. Patients with ACS are at serious risk of developing cardiovascular events within the first year of acute coronary syndrome (1).

Identifying patients with classic symptoms presentation and ECG changes has never been a problem, but the problem arises when patients present with atypical symptoms and with no ECG changes. The complexity is because of the heterogeneity of this syndrome, as it includes patients with varying clinical picture. Many patients present with atypical presentation with little or no ECG changes, and it presents a major challenge to the admitting clinician and it presents concerns for patient's safety.

Acute coronary syndrome is one of the most common clinical diagnoses suspected among patients admitted to the acute medical unit. Hence junior doctors who are initially assessing the patients should rely heavily on the history, clinical findings and ECG changes to diagnose and treat patients. It is very important to recognize those patients who have a high probability of having an ACS and treating them appropriately and no delay must be made in starting these patients on the appropriate treatment (2, 3).

How reliable is our clinical diagnosis and is it safe to discharge patients with atypical presentation and normal ECG?

## Methods

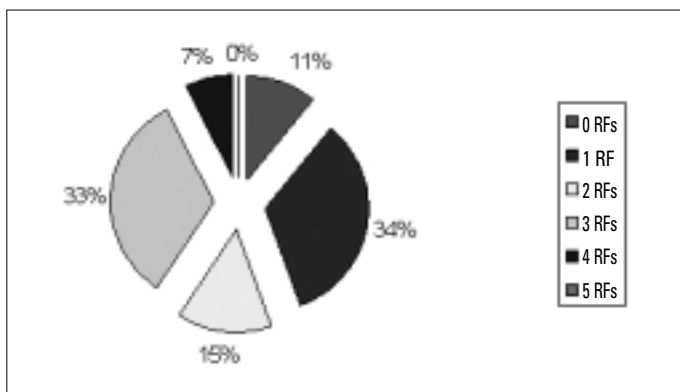
We looked at patients presenting to the Heart Assessment Centre in a month. It is a 20-bed assessment center for patients presenting with chest pain in a tertiary care university hospital. We had an opportunity to study the population presenting with chest pain to the center and their clinical picture with final diagnosis. The aim of the study was to determine the predictability of clinical evaluation in diagnosing ACS.

The study was a prospective cohort study. All patients who were admitted to the Heart Assessment Centre with a provisional diagnosis of ACS or patients in whom ACS was to be ruled out were selected. There were a total of 126 patients that were initially considered. The study period was a period of one month (January 2006). They were initially assessed by a Senior Medical Officer or a Clinical Fellow. The character of pain and duration of pain was documented. The character of pain was documented as cardiac chest pain and atypical or non-cardiac chest pain. Cardiac chest pain was defined as pain in the left side of chest presenting with exertion, radiating to the left side of arm and relieved with nitrate spray and rest. Any other pain was classified as non-cardiac chest pain or atypical. Depending on the duration of chest pain the patients were divided as having chest pain for <5, 5-10, 10-30, >30

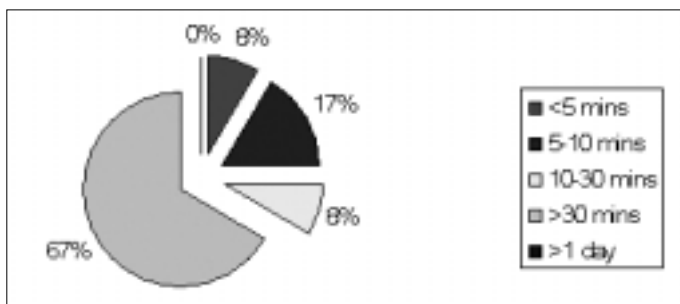
minutes and >1 day. The patients were looked for risk factors - diabetes, hypertension, previous ischaemic heart disease, stroke, chronic renal impairment, smoking, dyslipidemia, male gender, obesity and family history of premature cardiovascular events (<65 years). They were further subdivided based on the number of risk factors as 0, 1, 2, 3, 4 and >4. Their ECG was assessed and any ischaemic or progressive changes were documented. The ECG changes that were considered included ST-T changes suggestive of ischaemia and non specific changes like poor R wave progression, arrhythmias, and pathological Q waves were not considered. All patients had troponin T tested 12 hours post chest pain. The final diagnosis was based on the expert opinion of the Consultant Cardiologist in combination with troponin positive results, and these patients were only further studied. The case notes were analyzed and information regarding the chest pain, risk factors and ECG changes were obtained on a purpose designed data collection sheet. The predictability of clinical evaluation, which included type of chest pain, duration of chest pain, historical risk factors and ECG changes in accurate diagnosis of troponin positive ACS was then studied.

**Results**

Among the 126 patients that were assessed, 31 (25%) were diagnosed as acute coronary syndrome. Out of these 26 (21%) were troponin T positive acute coronary syndrome patients. Only these 26 patients were included in the further analysis. The study cohort was further stratified according to 0, 1, 2, 3, 4, >4 risk factors and they were 3, 9, 4, 9, 2 and 0 respectively (Fig. 1).



**Figure 1. Distribution of patients with troponin positive ACS according with number of risk factors**  
 ACS- acute coronary syndrome, RF- risk factor



**Figure 2. Distribution of patients with troponin positive ACS according with the duration of chest pain**  
 ACS- acute coronary syndrome

Of all the patients included 12 (46%) had typical cardiac sounding chest pain and the rest 14 (54%) had atypical symptoms. The duration of cardiac chest pain was further classified on duration of pain, <5, 5-10, 10-30, >30 minutes, >1 day and the results were 1 (8%), 2 (17%), 1(8%), 8 (67%) and 0 respectively (Fig. 2).

Thirteen patients (50%) out of the study group had significant ECG changes on admission and 13 (50%) did not have any noticeable changes. On taking both the cardiac chest pain and ECG changes into account it predicted only 6 (23%) patients. While using cardiac chest pain or ECG changes as a guide it predicted 20 (77%) of patients with ACS. Six (23%) patients were diagnosed with ACS with no cardiac chest pain or ECG changes (Fig. 3).

**Discussion**

Acute coronary syndrome encompasses a wide spectrum of clinical presentations with different baseline risk and extent of coronary disease. Early aggressive medical and interventional treatment has been shown to substantially reduce subsequent cardiac events (2). Patients are risk stratified based on clinical presentation, risk factors (4-6) and ECG changes during the initial assessment and they are further assessed and risk stratified by doing the cardiac enzyme markers. Because of the variety of clinical presentation and the heterogeneity of the disease it is difficult to assess and discharge patients without further assessment.

If both cardiac sounding chest pain and ECG changes were considered together, it predicted ACS in only 6 (23%) patients. If either cardiac sounding chest pain or ECG changes were considered separately at presentation, as either one being present, then it predicted 20 (77%) of troponin T positive ACS. Six (23%) were diagnosed to have ACS without cardiac chest pain or ECG changes.

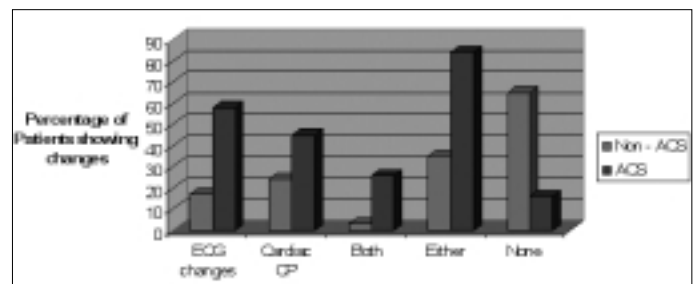
Combining cardiac sounding chest pain and ECG changes as either one out of the two being present on admission predicted a significant proportion of patients who went on to be diagnosed as troponin T positive ACS (70%).

**Clinical presentation**

Classic angina history is obtained in only about half of the patient presenting with ACS. The other half of the patients present with atypical presentation and picking up these patients, diagnosing and treating them appropriately is challenging (6).

**Electrocardiogram**

The 12-lead ECG is a non-invasive and widely available test for the evaluation of patients ting with chest pain. Ischemic changes in the ECG (ST changes, T wave changes and new arrhythmias or bundle branch block) are seen in only half of the patient population presenting with suspected ACS. Hence the ECG is non-diagnostic in at least half of the patients. The qualitative features of an ECG taken upon hospital admission identify a gradient of risk across the spectrum of ACS (7-11).



**Figure 3. Cardiac chest pain/ECG changes in prediction of ACS**  
 ACS- acute coronary syndrome, ECG- electrocardiogram

### Cardiac markers

Time-dependent release of cardiac biochemical markers identifies necrosis of the myocardium. It helps to risk stratify and early treatment of the patient. The biochemical markers used in the cardiology wards are myoglobin, creatine kinase and troponins (12-17). Serial serum markers have been found to be more diagnostic than a single serum marker.

### Modalities for detecting exercise-induced ischaemia

Patients with ischaemia may present with atypical presentation, normal ECG and with normal serum markers. At least one-fourth of the population might be with the challenging above presentation. Patients presenting in this category should have provocative stress testing like exercise stress test, stress echocardiography or stress nuclear imaging. Exercise stress test predict coronary artery disease by increasing the cardiac metabolic demand, causing flow limiting ischaemia in diseased vessels and subsequently resulting in angina and showing ischaemic changes in ECG (18). Similarly, in stress echocardiography we look for regional wall motion abnormalities (19) and in stress nuclear imaging we look for relative myocardial perfusion as suggestive of myocardial ischaemia (20-21).

### Conclusion

Although certain elements of the chest pain history and ECG changes are associated with increased or decreased likelihood of a diagnosis of ACS, none of them alone or in combination identify a group of patients that can be safely discharged without further diagnostic testing.

### References

1. Yeghiazarians Y, Braunstein JB, Askari A, Stone PH. Unstable angina pectoris. *N Engl J Med* 2000; 342: 101-14.
2. Braunwald E, Antman EM, Beasley JW, Califf RM, Cheitlin MD, Hochman JS, et al; American College of Cardiology; American Heart Association. Committee on the Management of Patients With Unstable Angina. ACC/AHA 2002 guideline update for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction—summary article: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (committee on the management of patients with unstable angina). *J Am Coll Cardiol* 2002; 40: 1366-74.
3. Bertrand ME, Simoons ML, Fox KA, Wallentin LC, Hamm CW, McFadden E, et al; Task Force on the Management of Acute Coronary Syndromes of the European Society of Cardiology. Management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. The task force on the management of acute coronary syndromes of the European Society of Cardiology. *Eur Heart J* 2002; 23: 1809-40.
4. Antman EM, Cohen M, Bernink PJ, McCabe CH, Horacek T, Papuchis G, et al. The TIMI risk score for unstable angina/non-ST elevation MI: a method for prognostication and therapeutic decision making. *JAMA* 2000; 284: 835-42.
5. Mueller C, Neumann FJ, Roskamm H, Buser P, Hodgson JMB, Perruchoud AP, et al. Women do have an improved long-term outcome after non-ST-elevation acute coronary syndromes treated very early and predominantly with percutaneous coronary intervention: A prospective study in 1,450 consecutive patients. *J Am Coll Cardiol* 2002; 40: 245-50.
6. Zaacks SM, Liebson PR, Calvin JE, Parrillo JE, Klein LW. Unstable angina and non-Q wave myocardial infarction: does the clinical diagnosis have therapeutic implications? *J Am Coll Cardiol* 1999; 33: 107-18.
7. Nyman I, Areskog M, Areskog NH, Swahn W, Wallentin L, for the RISC Study Group. Very early risk stratification by electrocardiogram at rest in men with suspected unstable coronary heart disease. *J Intern Med* 1993; 234: 293-301.
8. Cannon CP, McCabe CH, Stone PH, Rogers WJ, Schactman M, Thompson BW, et al, for the TIMI III Registry ECG Ancillary Study Investigators. The electrocardiogram predicts one-year outcome of patients with unstable angina and non-Q-wave myocardial infarction: results of the TIMI III Registry ECG Ancillary Study. *J Am Coll Cardiol* 1997; 30: 133-40.
9. Savonitto S, Ardissino D, Granger CB, Morando G, Mafri A, Lee KL, et al. Prognostic value of the admission electrocardiogram in acute coronary syndromes. *JAMA* 1999; 281: 707-13.
10. Hathaway WR, Peterson ED, Wagner GS, Granger CB, Zabel KM, Pieper KS, et al. Prognostic significance of the initial electrocardiogram in patients with acute myocardial infarction. *JAMA* 1998; 279: 387-91.
11. Mauri F, Franzosi MG, Maggioni AP, Santoro E, Santoro L. Clinical value of 12-lead electrocardiography to predict long-term prognosis of GISSI-1 patients. *J Am Coll Cardiol* 2002; 39: 1594-600.
12. Roberts R, Fromm RE. Management of acute coronary syndromes based on risk stratification by biochemical markers: an idea whose time has come. *Circulation* 1998; 98: 1831-3.
13. Savonitto S, Granger CB, Ardissino D, Gardner L, Cavallini C, Galvani M, et al, for the GUSTO-IIb Investigators. The prognostic value of creatine kinase elevations extends across the whole spectrum of the acute coronary syndromes. *J Am Coll Cardiol* 2002; 39: 22-9.
14. Hamm CW, Ravkilde J, Gerhardt W, Jorgensen P, Peheim E, Ljundahl L, et al. The prognostic value of serum troponin T in unstable angina. *N Engl J Med* 1992; 327: 146-50.
15. Antman EM. Decision making with cardiac troponin tests. *N Engl J Med* 2002; 346: 683-93.
16. Ohman EM, Armstrong PW, Christenson RH, Granger CB, Katus HA, Hamm CW, et al, for the GUSTO-IIa Investigators. Cardiac troponin T levels for risk stratification in acute myocardial ischemia. *N Engl J Med* 1996; 335: 1333-41.
17. Antman EM, Tanasijevic MJ, Thompson B, Schactman M, McCabe CH, Cannon CP, et al. Cardiac-specific troponin I levels to predict risk of mortality in patients with acute coronary syndromes. *N Engl J Med* 1996; 335: 1342-9.
18. McNeer JR, Margolis JR, Lee KL, Kisslo JA, Peter RH, Kong Y, et al. The role of the exercise test in the evaluation of patients for ischemic heart disease. *Circulation* 1978; 57: 64-70.
19. Mazeika PK, Nadazdin A, Oakley CM. Dobutamine stress echocardiography for detection and assessment of coronary artery disease. *J Am Coll Cardiol* 1992; 19: 1203-11.
20. Laarman GJ, Brusckhe AV, Verzijlbergen JF, Go TL, Bal ET, Van Der Wall EE, et al. Thallium-201 scintigraphy after dipyridamole infusion with low exercise: quantitative analysis versus visual analysis. *Eur Heart J* 1990; 11: 162-72.
21. Go RT, Marwick TH, MacIntyre WJ, Saha GB, Neumann DR, Underwood DA, et al. A prospective comparison of rubidium-82 PET and thallium-201 SPECT myocardial perfusion imaging utilizing a single dipyridamole stress in the diagnosis of coronary artery disease. *J Nucl Med* 1990; 31: 1899-905.