

Topographic patterns of Myocardial Infarction in Pakistani Population

MOHAMMAD YASIR IMRAN, ZAHID HUSSAIN SHAH, MUHAMMAD FAHR HAYAT, MUHAMMAD KHIZER HAYAT, IRSHAD HUSSAIN QURESHI

ABSTRACT

Background: Myocardial infarction is a leading cause of morbidity and mortality in the world as well in Pakistan. It usually presents with retrosternal squeezing or crushing chest pain radiating to arms, neck, back, abdomen, or lower jaw. It is usually accompanied with pallor, sweating, nausea, vomiting, breathlessness or syncope.

Aim: To determine the frequency of different myocardial areas in acute myocardial infarction and describe the myocardial infarction of different areas with various modes of presentations.

Study design: Case series study

Setting: Department Of Emergency & Medical Unit-Ii Of Services Hospital, Lahore And Mayo Hospital Lahore.

Duration of study: From 1ST August 2006 TO 31ST January 2007.

Methods: One hundred patients fulfilling the inclusion criteria were selected for this study. Informed consent was obtained from the patients. Patient included in the study were to undergo detailed history and physical examination. All the patients were diagnosed by history, ECG, raised cardiac enzymes, non-enzymatic marker that is troponin-T.

Results: Out of 100 cases, 84 were male and 16 were female. Major symptom noted were chest pain (100%), sweating (47%), breathlessness (29%), vertigo (13%) and syncope (8%). CK – MB was elevated in 33% and Troponin-T in 57%. Among 100 patients, 39 had inferior wall MI, 34 had extensive anterior wall MI, 13 had posterior wall MI, 6 had anteroseptal MI, 3 had anterolateral MI, 4 had non Q wave MI and 2 had lateral wall MI.

Conclusion: Anterior and inferior wall myocardial infarction is more in number and their common symptoms are typical chest pain, sweating and breathlessness. High morbidity and mortality is associated with these myocardial infarction. Male more than 45 years with factors like smoking, hypertension and diabetes mellitus are more prone to get myocardial infarction.

Keywords: Myocardial infarction, cardiac enzymes, ECG.

INTRODUCTION

Myocardial infarction is a leading cause of morbidity and mortality in the world as well in Pakistan.¹ It usually presents with retrosternal squeezing or crushing chest pain radiating to arms, neck, back, abdomen, or lower jaw.² It is usually accompanied with pallor, sweating, nausea, vomiting, breathlessness or syncope. It may also take the form of mild retrosternal discomfort, may mimic symptoms of indigestion or alternatively may be asymptomatic. The incidence of painless infarction is greater in women, diabetics and increases with age. Pain usually extends to more than half an hour and the onset may be sudden or gradual.

There are different types of myocardial infarctions. The location and extension of infarction depend upon the anatomic distribution of the occlusion, emboli and spasm of coronary vessels.³ Occlusion of the anterior descending branch of the left coronary artery results in infarction of the anterior wall.⁴ Occlusion of the left circumflex artery, branch of left coronary artery produces anterolateral or posterolateral infarction. Right coronary artery involvement leads to posteroinferior infarction. The classic evolution of changes is from hyper acute T waves, ST-segment elevation, T wave inversion, and Q wave development. Another type of infarction called non Q wave infarction results in the injury of partial thickness of the myocardium. It is characterized by absence of Q waves development of deep inverted T wave and more commonly ST segment depression than elevation.

The diagnosis of myocardial infarction rests mainly on history, clinical features and investigations. Investigations include serial electrocardiographic changes⁵ cardiac enzymes and echocardiography. Creatinine phosphokinase (CPK) or (CK) actively starts to rise after six hours of myocardial infarction, peaks within 24 hours and returns to normal within 3-4 days CK-MB is an isoenzyme and is specific for cardiac muscle and is less affected by skeletal muscle injury or intramuscular injections. Aspartate aminotransferase (AST) peaks at 24-48 hours and falls to normal by 72 hours. Lactate dehydrogenase (LDH) begins to rise within 3-4 days and remains elevated for up to 14 days. Troponin-T is not an enzyme but is a regulatory protein and is highly specific and sensitive indicator for cardiac muscle injury⁶. It is released within 2-4 hours of myocardial infarction and remains elevated up to 14 days. The combination of CK-MB and troponin testing can have even higher sensitivity and is increasingly employed for the purpose of ruling out myocardial infarction.

Treatment of myocardial infarction is directed towards prompt analgesia, bed rest, oxygen therapy, antiplatelet therapy and thrombolysis with either streptokinase or tissue plasminogen activator. A recent study found that beta blocker therapy given within twenty four hours of the onset of acute myocardial infarction resulted in decreased incidence of cardiac rupture and hospital mortality.⁷ Complications of myocardial infarction include arrhythmias, cardiac failure, cardiogenic shock, pericarditis, septal rupture, cardiac aneurysm, valvular heart disease and sudden death^{8,9}.

The objectives of this study were to determine the frequency of different myocardial areas in acute myocardial infarction and to describe the myocardial infarction of different areas with various modes of presentations.

Department of Medicine, King E M Uy/ Mayo Hospital, Lahore
Correspondence to Dr. Zahid Hussain Shah, Assistant Professor
Medicine Email: zahidhamdani65@gmail.com Cell: 03009466289

OPERATIONAL DEFINITIONS**Myocardial Infarctions**

1. Necrosis of a region of the myocardium caused by an interruption in the blood supply usually as a result of occlusion of a coronary artery or its branches is called cardiac infarction.

2. Typical chest pain with diagnostic ECG changes and raised cardiac enzymes.

Topographic patterns: The description of the regions of the body or of a body part like heart, especially the region of a definite and limited area is called topographic patterns.

MATERIALS AND METHODS

This non-interventional case series study was conducted in emergency department and Medical unit-II of Services Hospital Lahore and Mayo hospital Lahore. The duration of the study was six months from 1st August 2006 to 31st January 2007. The sample size comprised of one hundred patients of acute myocardial infarction. Convenient non-probability sampling technique was used.

Inclusion criteria

1. History of typical chest pain
2. Definite fresh ECG changes with evolutionary change.
3. Serial rise in cardiac enzymes.
4. Positive troponin-T test.
5. Male and female between 20-70 years.

Exclusion criteria

1. ECG changes equivocal.
2. Cardiac bypass surgery, LBBB (left bundle branch block) or pre-excitation syndrome.
3. ECG changes due to drugs, electrolyte imbalance and cardiovascular accident (CVA).

Data collection procedure: One hundred patients fulfilling the inclusion criteria were selected from emergency department and Medical unit-II of Services Hospital Lahore. Informed consent was obtained from the patients.

A detailed history of the patients was taken and they underwent a thorough physical examination. History included the demographic profile of the patient like age, sex, marital status, occupation and addresses. History of any other associated problem like diabetes mellitus, hypertension, smoking, psycho – social stress and presence of peripheral vascular disease was noted. All the relevant basic investigations were done. All the patients were diagnosed by history, ECG, raised cardiac enzymes, non enzymatic marker that is troponin-T.

Topographic diagnosis of myocardial infarction was made according to following criteria.

- a) I, avl, V6 Lateral wall myocardial infarction
- b) VI-V6 Extensive anterior myocardial infarction.
- c) VI-V6 Anteroseptal myocardial infarction
- d) I, avl, V4=V6 Anterolateral myocardial infarction
- e) II, III, avf Inferior wall myocardial infarction
- f) Without Q wave non Q wave myocardial infarction

• Daily ECG (to see the myocardial infarction and arrhythmias).

- Troponin-T on day first
- Cardiac enzymes daily for first week then on second and third week to observe their rising and declining pattern.

Data analysis procedure: Data was collected on a detailed pro forma. It was compiled in the computer and

analyzed by using SPSS 10 software. The frequency of various topographic patterns of acute myocardial infarction and variables (age, sex, marital status, and presenting complaints of the patients) were determined. The association between variables and pattern of acute myocardial infarction were determined. Chi Square test was applied to find out the significance of association.

RESULTS

We studied 100 cases of acute myocardial infarction. There age was between 41 – 70 years. 84 patients were male and 16 were female. Out of 100, 70 patients presented with severe chest pain. Major symptom noted were chest pain (100%), sweating (47%), breathlessness (29%), vertigo (13%) and syncope (8%). Major risk factors noted in these patients were smoking (71%), diabetes mellitus (46%), hypertension (44%), sedentary lifestyle and psychosocial stress (36%). Family history of diabetes was present in 45%, of hypertension in 42% and ischemic heart disease in 40%. Diagnosis was made on the basis of cardiac enzymes, definite ECG changes and Trop T level in blood. ST segment was elevated in 96% and 4% had non Q wave myocardial infarction. CPK was elevated in 33%, CK – MB in 39%, aspartate aminotransferase in 30%, LDH in 27%, Trop T in 57%.

Among 100 patients, 39 had inferior wall MI, 34 had extensive anterior wall MI, 13 had posterior wall MI, 5 had anteroseptal MI, 3 had anterolateral MI, 4 had non Q wave MI and 2 had lateral wall MI. Acute pulmonary edema and left ventricular failure was noted in 31% and cardiogenic shock in 11%. No tachy or bradyarrhythmia was noted.

Table 1: Distribution of patients by presenting complaints

Symptoms	Positive	Percentage
Breathlessness	29	29
Sweating	47	47
Vertigo	13	13
Syncope	8	8

Table 2: Distribution of patients by CK – MB and Trop T

Enzymes	Positive	Negative
Trop T	57	43
CK – MB	39	61

P value = 0.01

Table-3: Distribution of patients by specification of myocardial region on electrocardiography

MI region	Yes	No
Posterior wall MI	13	87
Lateral wall MI	2	98
Extensive anterior MI	34	66
Anteroseptal MI	5	95
Anterolateral MI	3	97
Inferior wall	39	61
Q wave/non Q wave MI	4	96

Myocardial infarction is a leading cause of morbidity and mortality in the world as well in Pakistan. It is number one killer in United States and worldwide. Every minute, an American dies of coronary heart disease. It is responsible for more than one in five deaths and nearly 700,000 deaths per year in United States.

In this study, frequency of different myocardial areas in acute myocardial infarction with various modes of presentations has been described. Out of 100 patients, 96% were Q wave (STEMI) and 4% were non-Q wave

(NSTEMI). 34 had extensive anterior wall MI, 5 had anteroapical MI, 3 had anterolateral MI, 13 had posterior wall MI, 4 had non Q wave MI and 2 had lateral wall MI. In one study by Gomez, Q wave MI was 62% and non-Q wave MI was 10%. Anterior wall MI was 57%, inferior wall MI was 20% and combined was 23%¹⁰. Another study by Jose showed 57% anterior wall MI, 39.1% inferior wall MI, 62.7% posterior wall MI and 3.9% anteroapical MI¹¹.

In my study, main presentations were chest pain, breathlessness (especially with LVE and pulmonary edema) and syncope along with sweating and vertigo. Some patients presented with shock. Pulmonary edema and LVE was noted in 31%, syncope in 8%, and cardiogenic shock in 11%. In study by Tipoo, 8.6% patients had cardiogenic shock.¹² Another study by Corrada showed that 16% patients were presented with signs of LVE and 7.6% with cardiogenic shock¹³. Study of Conde – Vella showed 16% patients with cardiogenic shock. Syncope was found in 4%¹⁴.

In my study, 84% were male and 16% were female. Their age was between 20–70 years. Majority were between 41–70 years. 71% were smokers 46% were diabetic, 44% were hypertensive, 36% with Psychosocial stress, sedentary lifestyle, 10% were obese and dyslipidemia was present in 13%. Family history of diabetes mellitus, hypertension and IHD was present in 45%, 42% and 40% respectively. A study by Jose showed 83.6% male, 16.2% female. 43.1% were smokers, 38.2% had hypertension, 40.2% were diabetic and 11% had past MI history¹⁵. Study by Rasool showed psychosocial stress in 68%¹⁶. Study by Tipoo indicated 62.3% male, 52.3% were hypertensive and 43.4% were diabetic¹⁷. Study by Banasiak showed history of dyslipidemia in 57.6% and obesity in 79.3%. This study also showed family history of coronary artery disease in 17.3% and diabetes in 23.3%. Peripheral artery disease was found in 9.9% of coronary artery disease patients¹⁷. The percentage of patients with dyslipidemia is less in my study as compared to above mentioned study because most patients were diabetic, hypertensive and had ischemic heart disease and for that they were taking anti hyperlipidemic drugs.

In my study, diagnosis was made by cardiac enzymes and Troponin T. Troponin T was positive in 57%, CK – MB in 39%, aspartate aminotransferase in 30% and LDH in 27%. This indicates that Troponin T is more sensitive and specific. Study by Sawlha showed that CK – MB and Troponin T was positive in 69% of patients. In 36%, only Troponin T was positive¹⁸. This study also showed that Troponin T is more sensitive and specific. Tachy and bradyarrhythmias were not found in my study but in one study, among patients with heart disease, approximately 21% patients had ventricular tachycardia and 34% had bradycardia¹⁸.

CONCLUSION

This study concludes that anterior and inferior wall myocardial infarction is more in number and their common symptoms are typical chest pain, sweating and breathlessness. High morbidity and mortality is associated with these myocardial infarction. Male more than 45 years with factors like smoking, hypertension and diabetes

mellitus are more prone to get myocardial infarction. Early diagnosis and treatment can prevent their complications. Further workup is required on myocardial infarction as it is very common in our community.

REFERENCES

1. Memon MA, Samad A. Acute myocardial infarction in women: prospective study from a developing country. *Pak J Cardiol* 1999; 10: 95-107.
2. Rich MW. Epidemiology, clinical features and prognosis of acute myocardial infarction in the elderly. *Am J Geriatr Cardiol* 2006; 15: 7-11.
3. Bashir Y. Murray longmore ian Wilkinson Eslee Torioic. Cardiovascular medicine. Bashir Y. (ed). Oxford Handbook of Clinical Medicine. 5th ed. New York: Oxford Universality Press, 2001; 104.
4. Ishaq M, Beg MS, Ansari SA, Hakeem A, Ali S. CAD risk profiles at a specialized tertiary care center in Pakistan. *Pak J Cardiol* 2003; 14: 61-8.
5. Ryan TJ, Anderson JL, Anman EM. ACC/AHA guidelines for the management of patients with acute myocardial infarction. A report of the American College of Cardiology / American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *J Am Coll Cardiol* 2001; 28: 1328-1428 (Medline).
6. Myocardial infarction redefined – a consensus document of the Joint European Society of Cardiology / American College Cardiology committee for the redefinition of myocardial infarction. *J Am Coll Cardiol* 2000; 36: 959-61.
7. Anzai T, Yoshikawa T, Takahashi T. Early use of beta blocker is associated with attenuation of C-reaction protein elevation and favorable short term prognosis after acute myocardial infarction. *Cardiology* 2003; 99: 47-53.
8. Barry MM, Thomas AM. Tierney LM, Mephee SJ. Current medical diagnosis and treatment. 41st ed. University of California, San Francisco: McGraw Hill, 2002: 364.
9. Samad A. Coronary artery disease in Pakistan: preventive aspects. *Pak J Cardiol* 2003; 14: 5-60.
10. Gomez JF, Zereba, Moss AJ, Mcniff S, Hall J. Prognostic value of location and type of myocardial infarction in the setting of advanced left ventricular dysfunction. *Am J Cardiol* 2007; 99: 642 – 6.
11. Jose VJ, Gupta SN. Mortality and morbidity of acute myocardial infarction in the current era. *Indian Heart J* 2004; 56: 210 – 14.
12. Tipoo FA, Qurashi AR, Najaf SM, Kazmi KA, Jafary F. Outcome of cardiogenic shock complicating acute myocardial infarction. *J Coll Physicians Surg Pak* 2004; 14 :6 - 9.
13. Corrada E, Mauri F, Mafri A, Alberti A, Corato A. Clinical and instrumental elements of left ventricular insufficiency in acute myocardial infarction. *G Ital Cardiol* 2001; 24: 825– 38.
14. Conde-Vela C, Moreno R, Hernandez R, Alfonso F. Cardiogenic shock at admission in patients with multivessel disease and acute myocardial infarction treated with percutaneous coronary intervention. *Int J Cardiol* 2007; 20: 146 – 9.
15. Rasul F, Stansfeld SA, Davey Smith G, Shlomo YB, Gallacher J. Psychological distress, physical illness and risk of myocardial infarction. *Psychol Med* 2007; 56: 1 – 9.
16. Banasiak W, Pocipancy R, Wilkims A, Ponikowski P. Characteristics of patients with coronary artery disease managed on an outpatient basis in the population of Poland. *Kardiologia* 2007; 65: 132 – 40.
17. Sawlha WA, Badaine Y. Diagnosis of heart disease. *Saudi Med J* 2004; 25: 1971 - 4.
18. Kapoor WN. Syncope. *N Engl J Med* 2000; 343: 1856