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Clinical Profile of non-Q Wave Myocardial *Infarction in Pakistani Population*

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SUMMARY

Fifty consecutive patients of non-Q wave myocardial infarction were studied as regards their presenting features, admission electrocardiogram (ECG) changes and complications during the hospital stay. Results showed that 68% of these were male while 60% were more than 50 years of age. Previous history of hypertension, diabetes mellitus and ischemic heart disease was present in 56%, 60% and 60% respectively. 20% of patients had ST segment depression on the admission ECG, 4% had ST segment elevation, 34% had T wave inversion while 44% had a mixed pattern. 56%, 42%, 2% and 0% of patients were in Killip class I, II, III, and IV respectively on admission. During hospital stay, 62% of patients had post-infarct angina, 4% had atrial fibrillation and one patient had ventricular arrhythmias leading to death. Segmental wall motion abnormalities were present in 76% but ejection fraction was >50% in 80% of patients.

In summary, non-Q wave myocardial infarction was more common in hypertensives, diabetics and in those with previous history of ischemic heart disease. These patients presented mainly with ST depression or T wave inversion or a mixture of these two abnormalities, were only rarely in Killip III or IV, had a more stable hospital course with only 2% mortality and had generally good LV function but had higher incidence of post-infarct angina. Thus, they need to be investigated more aggressively for purposes of possible revascularization.

INTRODUCTION

Myocardial infarction is a focus of ischemic coagulative necrosis. It is one of the commonest medical emergencies. It affects about 1.5 million people each year in the United States of America and is responsible for 25% of the total annual mortality there[1] Myocardial infarcts may be transmural or non-Q wave (also called non-transmural and previously known as subendocardial infarcts). Transmural myocardial infarction involves all three layers of the ventricular wall while non-transmural infarction may involve the endocardium or intramural myocardium but does not extend all the way through the ventricular wall to the epicardium.

Non-Q wave myocardial infarction is more common in old age, in those who suffered previous myocardial infarction and in cocaine abusers.[2,3,4]

Patients with non-Q wave myocardial infarction show significantly lower ischemic threshold, and experience more ischemic events like unstable angina and reinfarction due to the presence of larger residual mass of viable but jeopardized myocardium within the perfusion zone of the infarct-related vessel. Despite less myocardial necrosis and better left ventricular function, the non-Q wave myocardial infarction has the same or even shorter long term survival as compared to Q wave myocardial infarction.[5,6,7] Moreover, this jeopardized or depressed myocardium after non-Q wave myocardial infarction has the potential to achieve normal contraction after successful coronary angioplasty[8].

Therefore, if we can diagnose and manage the non-Q wave myocardial infarction at an early stage, we can save the lives of many patients. This study intends to elaborate the clinical profile of non-Q wave myocardial infarction in our population.

PATIENTS AND METHODS

Fifty consecutive patients with acute, non-Q wave myocardial infarction admitted to the coronary care unit (CCU) of Sheikh Zayed Hospital, Lahore, were included in the study and followed till discharge from the hospital. Non-Q wave myocardial infarction was diagnosed on the basis of the following criteria;

- Typical anginal pain.
- ST segment elevation of > 2mm or ST segment depression of > 1mm or T wave inversion or a mixed pattern consisting of a combination of the above ST,T changes with lack of development of Q waves in subsequent serial ECGs.
- Elevation of cardiac enzymes, creatine phosphokinase (CPK) and/or aspartate aminotransferase (AST) and/or creatine phosphokinase MB fraction (CK-MB) in the typical pattern.

The latter two criteria were a must for the diagnosis to be made while the presence of typical anginal pain was not always necessary.

Inclusion Criteria

All adult patients presenting acutely and belonging to any age group and both sexes and fulfilling the above-mentioned criteria of non-Q wave myocardial infarction were included in the study.

Exclusion Criteria

1. Patients who developed significant Q- waves during their hospital stay.
2. Patients with history of recent intramuscular injections.
3. Patients with incomplete data.

Treatment Protocol

All patients were admitted to the CCU and advised bed rest for 24-48 hours. Their complete medical history and physical examination was recorded. Items particularly stressed in the history were major and minor risk factors for ischemic heart disease (IHD) such as diabetes mellitus, hypertension, hyperlipidemia, amount and duration of cigarette smoking, family history of IHD etc.

Patients were given one 300mg tablet of acetylsalicylic acid to chew and swallow at the time of admission and then half tablet once daily. All patients were given intravenous isosorbide dinitrate infusion for the first 24 hours unless contraindicated or not tolerated. The dose was adjusted to clinical needs and averaged 2 mg/hour.

Every patient was given sodium heparin as a bolus of 5000 IU intravenously followed by a continuous infusion of 1000 IU/hour for 24-48 hours. This was then replaced with regular bolus therapy in a dose of 5000 IU intravenously every 6 hours. No patient was given streptokinase or any other thrombolytic agent.

Oxygen inhalation at a rate of 4-6 litres/minute was given initially to all patients if tolerated whether or not he was in cardiac failure. Continuous ECG monitoring was done of all patients during their period of stay in the CCU.

Concomitant Therapy

Patients received narcotic analgesics (morphine or pethidine), oral nitrates, beta blockers, calcium channel blockers, angiotensin converting enzyme inhibitors, diuretics, anti-arrhythmics, inotropic agents and insulin as indicated in individual cases.

Electrocardiography

Serial ECGs were done by trained technicians 6 hourly during the first 24 hours and then daily for three to four days and whenever specifically indicated.

The number of leads showing changes of acute myocardial infarction (ST segment elevation, ST segment depression, T wave inversion) at the time of admission was recorded. Evolutionary changes in ST segment and T wave morphology were followed in serial ECGs.

Cardiac Enzymes

Creatinine phosphokinase was measured by CK NAC activated humazym UV test (Human manufacture) 6 hourly during the first 24 hours and then daily until it showed a downward trend. Our normal lab values of CPK are 24-195 U/L for males and 24-170 U/L for females. In addition AST and, when required, CK-MB was also measured.

Clotting Profile

All patients had their baseline platelet count, prothrombin time and activated partial thromboplastin time checked in the laboratory before starting anticoagulant therapy and then repeated once daily.

Echocardiography

Echocardiography was done in all patients and in all types of non- Q myocardial infarctions. The same person carried out all the studies on M-mode + 2D echocardiography machine with doppler facility (model SSH 40 A, Toshiba) on an average day 5-7 of the acute infarction. Standard parasternal long and short axis and subcostal views were used. Apex was studied in apical two and four chamber views with patient in left lateral position with head slightly elevated and tranducer placed at the point of maximal cardiac impulse. Wall motion abnormalities of heart were studied in detail. Symmetric systolic inward motion was defined as normal. Less than normal contractility was termed hypokinesia. Akinesia meant absent systolic inward motion. Dyskinesia meant paradoxical outward systolic motion.

End-systolic and end-diastolic volumes and left ventricular ejection fraction (LVEF) were also recorded carefully. Video recording of the studies were observed independently by one of the consultant cardiologists working in the cardiology department of Sheikh Zayed Hospital,Lahore.

Statistical Analysis

It was done with the help of SPSS package for different variables recorded in the study.

RESULTS

Fifty consecutive patients with non- Q wave myocardial infarction fulfilling the criteria of the study and admitted in the coronary care unit over a period of 10 months were included in the study. All the patients were assessed and treated by the residents, under supervision of consultant cardiologists.

Table 1: Age distribution.

Age	*Number*	*Percent*
< 50 years	20	40
> 50 years	30	60
Total	50	100

Amongst the fifty patients, 20 patients (40%) were less than fifty years of age and 30 patients (60%) were more than fifty years of age (Table 1). Thirty-four patients (68%) were males and only 16 patients (32%) were females, (Table 2).

Table 2: Sex distribution.

Sex	*Number*	*Percent*
Male	34	68
Female	16	32
Total	50	100

Thirty patients (60%) were diabetic while 56% had history of hypertension. 32 patients (64%) had history of ischemic heart disease and 18 patients (36%) had no history of ischemic heart disease (Table 3). At presentation, 30 patients (60%) had history of typical ischemic precordial pain lasting more than half an hour associated with sweating, radiating to left arm, neck or back, compressing in nature and severe in intensity.

Table 3: History of medical illnesses (n=50).

Illness	*Number*	*Percent*
Diabetes mellitus	30	60
Hypertension	28	56
Ischemic heart disease	32	64

Twenty patients (40%) did not have history of typical ischemic central chest pain but had only history of epigastric pain or shortness of breath or sweating or sense of compression in the chest or sinking of heart etc.

Patients were classified according to Killip classification[9]. According to this, patients with no pulmonary rales or third heart sound are in class I and those with pulmonary rales upto halfway up the lungs with or without a third heart sound are in class II. Patients with pulmonary rales more than

halfway up the lungs along with a third heart sound are in class III while those in cardiogenic shock are in class IV.

At admission, 28 patients (56%) were in Killip class I, 21 patients (42%) were in class II, only 1 patient (2%) in class III and none in class IV (Table 4). Most of the patients presenting with Killip class II at admission were having ST segment depression or ST segment depression plus T wave inversion in the ECG.

Table 4: Killip class at admission.

Class	*Number*	*Percent*
I	28	56
II	21	42
III	01	02
IV	00	00
Total	50	100

On admission, 10 patients (20%) had ST segment depression in ECG, 2 patients (4%) had ST segment elevation, 17 patients (34%) had T wave inversion and 21 patients (42%) had mixed pattern which means either ST segment elevation with T wave inversion or ST segment depression with T wave inversion or some leads with ST segment elevation and others with ST segment depression and T wave inversion which was rare. Most common mixed pattern was ST segment depression with T wave inversion (Table 5).

Table 5: ECG patterns of non-Q myocardial infarction.

ECG pattern	*Number*	*Percent*
ST segment depression	10	20
ST segment elevation	02	04
T wave inversion	17	34
Mixed pattern	21	42
Total	50	100

Out of 50 patients, 31 patients (62%) had post-infarct angina. Most of the patients having post-infarct angina were those who had presented with ST segment depression in ECG or mixed pattern with ST segment depression and T wave inversion.

Twenty four patients had sinus tachycardia, 2 patients had atrial fibrillation, 4 patients had unifocal PVCs and one patient who presented with ST segment depression developed ventricular tachycardia and ventricular fibrillation and died (Table 6). Two patients had transient LBBB during the study.

Table 6: Incidence of cardiac arrhythmias in the whole group (n=50).

Arrhythmia	*Number*	*Percent*
Sinus tachycardia	24	48
Atrial fibrillation	02	04
Premature ventricular contractions	04	08
Ventricular tachycardia	01	02

Thirty eight patients (76%) showed segmental wall motion abnormalities on echocardiography after non-Q wave myocardial infarction while forty patients (80%) had left ventricular ejection fraction (LVEF) above 50%. Only 5 patients (10%), had evidence of thrombosis in the left ventricular cavity.

DISCUSSION

In this small study, fifty consecutive patients who fulfilled the criteria of non-Q wave myocardial infarction were followed from admission to discharge. 60% of the patients were more than fifty years of age. This compares with 63% in another study done at the same centre by the author and his colleagues in which all patients with IHD were included.[9] Thus no significant difference was found in the age incidence of acute non-Q wave myocardial infarction. As regards the sex distribution, 68% were males while 32% were females. There was a significantly higher proportion of females suffering from acute, non-Q wave myocardial infarction as compared to the 1:5 female

to male ratio for all patients with IHD found in our previous study.[10]

Diabetes mellitus and hypertension are major risk factors for ischemic heart disease. In this small study, 60% of non-Q wave myocardial infarction patients were diabetics and 56% were hypertensive. These figures are again significantly higher than the 21.5% incidence for both diabetes mellitus and hypertension found in our previous study[10] and indicate that the presence of these conditions makes it more likely for the patient to suffer from a non-Q wave myocardial infarction. 60% of patients had history of ischemic heart disease in the form of angina pectoris, unstable angina or previous myocardial infarction which is in agreement with the study of Ogawa et al.[11]

Sixty percent of patients of non-Q wave myocardial infarction had history of typical ischemic central chest pain but 40% did not. They only had history of shortness of breath or sense of compression in the chest or sinking of heart. Therefore, if chest pain is considered as one of the major diagnostic criteria for myocardial necrosis in non-Q wave myocardial infarction, a number of cases will be missed. Thus, cardiac enzymes become the single most important criteria for the diagnosis of non-Q wave myocardial infarction. Cardiac enzyme CK-MB is more specific for the diagnosis of myocardial infarction.

As regards the subtypes of non-Q wave myocardial infarction on ECG, 20% patients presented with ST segment depression, 4% with ST segment elevation, 34% with T wave inversion and 40% with mixed pattern which compares well with the study of Ogawa et al.[11] There is another type of non-Q wave myocardial infarction in which the ECG may be completely normal but that type was not included in this study.

Regarding the Killip classification of non-Q wave myocardial infarction patients at admission, 56% of patients were in class I, 42% were in class II, only two percent in class III and none was in class IV which is in agreement with the studies of Palagi et al and Berger et al. that the complication of left ventricular failure occurs more commonly in Q wave myocardial infarction patients as compared to non-Q wave myocardial infarction patients.[12,13] In this study, 44% of patients had evidence of cardiac failure during hospital stay which was mostly of Killip class II. Majority of these patients had presented with ST segment depression only or with mixed pattern of ST segment depression and T wave inversion. This was also observed by Ogawa et al and Willich et al in their studies.[11,14]

The incidence of serious cardiac arrhythmias was very low in this study. 48% of patient had sinus tachycardia, 4% had atrial fibrillation, 8% had PVCs which were mostly unifocal and only one patient, who presented with ST segment depression, developed ventricular tachycardia and fibrillation and died on the third day of admission. This is in agreement with the study of Gibson et al in which he observed that patients of non-Q wave myocardial infarction can experience sudden death despite well preserved left ventricular function.[15]

There was a high incidence of post-infarct angina in this study. 62% of the patients suffered from angina during their hospital stay. This has also been found in other studies by Cheitlin et al, Mickley et al, and Gibson et al.[6,7,15] They observed that despite less myocardial necrosis and better left ventricular function, the non-Q wave myocardial infarction patients show significantly lower ischemic threshold, have a higher rate of unstable angina, more reinfarction and a greater likelihood of undergoing subsequent angioplasty or bypass surgery.

In this study, segmental wall motion abnormalities were present in 76% of non-Q wave myocardial infarction patients on echocardiography but LVEF was more than 50% in 80% of patients. These findings are similar to the studies of Koyanagi et al. and Mahias et al. in which they have observed that segmental wall motion abnormalities are very common after myocardial infarction but are significantly greater in patients with Q wave myocardial infarction as compared to non-Q wave myocardial infarction. LVEF is well maintained in non-Q wave myocardial infarction patients and there is also significant spontaneous improvement in wall motion of the infarct areas in non-Q wave but not in Q wave events.[16,17]

Our present strategy of conservative treatment of acute, uncomplicated, non-Q wave myocardial infarction with drugs is quite reasonable as we have observed quite good results in our study by this regimen. However, after completion of the acute phase, these patients should be aggressively investigated as there is jeopardized but viable mocardium which is the hallmark of non-Q wave myocardial infarction. If confirmed, it should be treated by revascularization procedures because this

jeopardized myocardium is the main reason for the high reinfarction and mortality rate in non-Q wave myocardial infarction patients but is potentially treatable.

In summary, non-Q wave myocardial infarction was more common in hypertensives, diabetics and in those with previous history of ischemic heart disease. These patients presented mainly with ST depression or T wave inversion or a mixture of these two abnormalities, were only rarely in Killip III or IV, had a more stable hospital course with only 2% mortality and had generally good LV function but had higher incidence of post-infarct angina. Thus, they need to be investigated more aggressively for purposes of possible revascularization.

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