MYOCARDIAL INFARCTION;
CLINICAL PREDICTORS OF IN HOSPITAL COMPLICATIONS IN PATIENTS PRESENTING WITH ACUTE ST SEGMENT ELEVATION

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ABSTRACT… Objective: To determine clinical predictors of in-hospital complications in patients presenting with acute ST elevation myocardial infarction. **Design:** Descriptive Study. **Period:** from October 2010 to January 2011. **Setting:** Faisalabad Institute of Cardiology, Faisalabad.. **Materials and methods:** A total 342 patients with AMI were recruited in this study. All patients presenting with acute ST elevation myocardial infarction and fulfilling inclusion and exclusion criteria were included in the study. A full history was taken, particularly age, sex, occupation, address, history of smoking, diabetes mellitus, hypertension, ischemic heart disease and family history of ischemic heart disease. Primary end point was death while secondary end point were patients who had mechanical, ischemic or electrical complications or all of them. **Results:** Mean age of the study population was 56.3±12.7 years. There were 255(74.6%) males and 87(25.4%) females. There were 103(30.1%) diabetics, 137(40.1%) hypertensive and 174(50.9%) smokers. Family history of IHD was present in 34(9.9%). Obesity was observed in 60(17.5%). Dyslipidemia was observed in 45(13.2%). Majority of patients 123(36%) presented between 4-8 hours after the onset of symptoms. Only 72(21.1%) patients presented to the hospital within 4 hours of onset of symptoms. Overall 194(56.7%) patients had anterior wall myocardial infarction followed by Inferior wall myocardial infarction 84(24.6%) patients. Streptokinase therapy for thrombolysis was given to 236(69%) patients. Overall in-hospital mortality was 28(8.2%). Most frequent in-hospital complication was cardiogenic shock occurring in 38(11.1%) followed by Ischemic complications (Post MI angina and Re-MI) 37(10.8%), heart failure in 37(10.8%) and 1st and 2nd degree AV blocks in 36(10.5%) patients. In-hospital mortality was most significantly associated with site of MI i.e. anterior wall myocardial infarction (X^2=28.88, p=0.0001) followed by patients not receiving Streptokinase therapy (X^2=18, p=0.001), Age (X^2=10.13, p=0.006). Site of MI had the highest Contingency Coefficient value of 0.279 followed by Streptokinase therapy 0.195 and age 0.170. **Conclusions:** Cardiogenic shock was the most frequent complication. Major predictors of in-hospital mortality were anterior wall myocardial infarction, patients not receiving streptokinase therapy and old age.

Key words: Acute myocardial infarction; Ischemic heart disease; Old age, Streptokinase therapy, Cardiogenic shock.

INTRODUCTION

Myocardial Infarction (MI) due to Coronary Artery Disease is a leading cause of death in the United States, where more than one million peoples have acute myocardial infarction (AMI) each year[1]. With the advent of coronary care units and early re-perfusion therapy (Lytics or PCI), in hospital mortality rates have decreased and has improved the outcome in survivors of the AMI due to circulatory failure either due to severe left ventricular dysfunction (for example arrhythmia) or one of the mechanical complications (for example ventricular rupture, papillary muscle rupture). Complications of MI include arrhythmic, mechanical, inflammatory (Early pericarditis and post MI syndrome) as well as left ventricular mural thrombus (LVMT). In addition to these major complications, right ventricular (RV) infarction and cardiogenic shock are possible complications of AMI.

Re-infarction occurs in 5-30 % of patients after fibrinolytic therapy[2]. It is more common in patients with diabetes or history of previous MI. Re-current MI (infarction in a different artery territory) occurs within 48 hours and it

may occur in up to 40 % of patients and can be difficult to diagnose[2]. Cardiac arrhythmias are not uncommon during and immediately after an AMI. About 90 % of the patients after an AMI develop some form of cardiac arrhythmias[3]. In 25% of patients such rhythm abnormalities manifest within the first 24 hours. The incidence of arrhythmias is more in ST elevation MI as compared to non-ST elevation MI. The clinicians must be aware of these arrhythmias in addition to re-perfusion strategies so we can treat to avoid exacerbation of Ischemia and sub-sequent hemodynamic compromise[3].

There are many studies conducted on the in hospital complications in patients with acute MI. These being conducted on different types of MI including cocaine induced MI, in acute inferior wall MI etc[4,5]. There were very few studies on the frequency of in hospital complications in acute MI patients in our community[5-8]. The present study would determine the more frequent in hospital complications in patients with AMI, so these patients can be closely monitored for these complications. With prompt and in time treatment we can save many lives. From this study we can assess the quality of care provided in our cardiac institute. This study was designed to determine clinical predictors of in-hospital complications in patients presenting with acute myocardial infarction.

MATERIAL AND METHODS

This descriptive study was conducted at the Faisalabad Institute of Cardiology, Faisalabad which is a tertiary care cardiac Institute. A total 342 patients with AMI were recruited in this study from October 2010 to January 2011. Patients presenting with acute myocardial infarction were included in the study on the basis of any two of the following criteria:

Chest pain consistent with acute myocardial infarction,

(1) Electro cardiographic changes; ST segment elevation >0.2mv in at least two contiguous chest leads or > 0.1 mv in at least two contiguous limb leads.

(2) New or presumably new left bundle branch block.

MI occurring after invasive coronary artery procedures such as PCI or coronary artery bypass grafting (CABG) and patients with history of valvular heart disease and primary pericardial disease were excluded. A full history was taken, particularly age, sex, occupation, address, history of smoking, diabetes mellitus, hypertension, ischemic heart disease and family history of ischemic heart disease. Relevant clinical examination of all the patients included in the study was done with emphasis on pulse, blood pressure, precordial examination and signs of congestive cardiac failure. Random blood sugar level taken at the time of admission was noted in diabetic patients. ECG was done once daily in all patients. X-ray chest was done only in those patients having signs of left ventricular failure. Site of myocardial infarction and medication used especially Streptokinase, were noted for all patients. All patients were treated according to latest recommendations. For diabetic patients oral hypoglycemic agents were discontinued and all patients were put on insulin according to random blood sugar level which was checked once daily. Patients were followed up daily and pulse, blood pressure, ECG changes and complications if any were monitored.

Primary end point was death while secondary end point was 1) patients who had mechanical, ischemic or electrical complications and 2) patients who had an uncomplicated course and were discharged.

STATISTICAL ANALYSIS

All the data was analyzed by SPSS (Statistical Package for Social Sciences) Version 12.0 for Windows. Categorical variables were expressed as frequencies and percentages and continues variables were presented as means±SD (standard deviation). Influence of various confounding factors on mortality was investigated using Pearson Chi-square and Contingency Coefficient values. The factors considered were age, sex, smoking, diabetes mellitus, history of ischemic heart disease, family history of ischemic heart disease, time from onset of symptoms till arrival at the hospital and Streptokinase therapy. 5% level of significance was used. All tests applied were two tailed.

RESULTS

After fulfilling the inclusion criteria, 342 patients presenting with new onset acute myocardial infarction were studied. Mean age of the study population was 56.3±12.7 years. There were 255(74.6%) males and 87(25.4%) females. There were 103(30.1%) diabetics, 137(40.1%) hypertensive and 174(50.9%) smokers. Family history of IHD was present in 34(9.9%). Obesity was observed in 60(17.5%). Dyslipidemia was observed in 45(13.2%). Table-I.

Table-I. Epidemiological characteristics.

Characteristics	Numbers (Percentages) n=342
Age mean years	56.3±12.7
Gender	
Male	255 (74.6%)
Female	87 (25.4%)
Diabetes Mellitus	103 (30.1%)
Hypertension	137 (40.1%)
Smoking	174 (50.9%)
FH of ischemic heart disease	34 (9.9%)
Obesity	60 (17.5%)
Dyslipidemia	45 (13.2%)

F/H= Family history; H/O= History of; IHD= Ischemic heart disease. AMI= Acute Myocardial Infarction

Majority of patients 123(36%) presented between 4-8 hours after the onset of symptoms. Only 72(21.1%) patients presented to the hospital within 4 hours of onset of symptoms. Overall 194(56.7%) patients had anterior wall myocardial infarction followed by Inferior wall myocardial infarction 84(24.6%) patients. Streptokinase therapy for thrombolysis was given to 236(69%) patients. Table-II.

Overall in-hospital mortality was 28(8.2%). Table-III. Most frequent in-hospital complication was cardiogenic shock occurring in 38(11.1%) followed by ischemic complications (post MI angina and Re-MI) 37(10.8%) and 1st and 2nd degree AV blocks occurring in 36(10.5%)

Table-II. Presentation characteristics.

Characteristics	Numbers (Percentages) n=342
Time from onset of symptoms till arrival	
<4 hours	72 (21.1%)
4-8 hours	123 (36%)
8-12 hours	51 (14.9%)
12-16 hours	46 (13.5%)
16-20 hours	29 (8.5%)
> 20 hours	21 (6.1%)
Site of MI	
Anterior wall MI	194 (56.7%)
Inferior wall MI	84 (24.6%)
Inferior wall + RV MI	18 (5.3%)
Inferior wall + Post MI	16 (4.7%)
Lateral MI	30 (8.8%)
Streptokinase therapy	236 (69%)

MI=Myocardial infarction

patients. Heart failure occurred in 37(10.8%) patients. Pericarditis occurred in 17(5%) patients followed by complete heart block in 14(4.1%) patients.

In order to see the independent clinical predictors of in-hospital mortality all variables were analyzed by using Chi-square and their association was checked by using Contingency Coefficient (Table-IV). In-hospital mortality was most significantly associated with site of MI i.e. anterior wall myocardial infarction (X^2=28.88, p=0.0001) followed by patients not receiving Streptokinase therapy (X^2=18, p=0.001), Age (X^2=10.13, p=0.006. Furthermore Contingency Coefficient value for all variables in relation to in-hospital mortality was noted. Site of MI had the highest value of 0.279 followed by Streptokinase therapy 0.195 and age 0.170.

DISCUSSION

Myocardial Infarction is a leading cause of death in the United States, where more than one million peoples have acute myocardial infarction (AMI) each year[1]. With the advent of coronary care units and early re-perfusion therapy (Lytics or PCI) in hospital mortality rates have decreased and has improved the outcome in survivors of the acute MI due to circulatory failure either due to severe left ventricular dysfunction (for example arrhythmia) or

Table-III. Outcome of study population.

Characteristics	Outcome (n=342)
In-hospital mortality	28 (8.2%)
Complications	
Post MI angina	37 (10.8%)
Shock	38 (11.1%)
AF	5 (1.5%)
VF	5 (1.5%)
VT	8 (2.3%)
1st 2nd 0 AV blocks	36 (10.5%)
CHB	14 (4.1%)
Hear failure	37 (10.8%)
Pericarditis	17 (5%)
VSR	9 (2.6%)

AV=Atrioventricular; AF=Atrial Fibrillation; CHB=Complete heart block; VF=Ventricular fibrillation; VT=Ventricular tachycardia; VSR=Ventricular septal rupture

Table-IV. Independent clinical predictors of in-hospital mortality.

Variable	Chi-square value	p-value (2-sided)	Contingency Coefficient
Age	10.13	0.006	0.170
Sex	0.173	0.677	0.023
Diabetes Mellitus	0.084	0.772	0.016
Smoking	2.56	0.464	0.086
Hypertension	0.834	0.361	0.049
Obesity	0.015	0.902	0.007
F/H of IHD	2.0	0.157	0.076
Time from onset till arrival	5.88	0.317	0.130
Site of MI	28.88	0.0001	0.279
No streptokinase therapy	18	0.001	0.195

one of the mechanical complications (for example ventricular septal rupture, papillary muscle rupture). Complications of MI include arrhythmic, mechanical, inflammatory (Early pericarditis and post MI syndrome) as well as left ventricular mural thrombus (LVMT). In addition to these major complications right ventricular (RV) infarction and cardiogenic shock are possible complications of AMI.

Kang et al[9] reported that hypertension at the time of acute myocardial infarction is associated with an increased rate of in-hospital mortality. Hypertensive patients suffered from acute renal failure, shock, and cerebrovascular event more frequently than non hypertensives. During follow-up of one-year, the incidence of major adverse cardiac events were higher in hypertensives. In multi-variate adjustment, old age, Killip class $\geqslant$ III, left ventricular ejection fraction <45%, systolic blood pressure <90 mmHg on admission, post procedural TIMI flow grade $\leqslant$ 2, female sex, and history of hypertension were independent predictors for in-hospital mortality.

Ganova-Iolovska et al[10] reported a relation between time delay and both age and education level. In 134 patients with AMI, 7% presented to a hospital within 59 minutes, and 44% within 4 hours of symptoms onset. The use of heparin was 98%. In the first 24 hours, Aspirin was administrated in 82% and Beta-Blockers in 73% of the cases. At discharge Aspirin, Beta-Blockers, Angiotensin Converting Enzyme Inhibitors (ACEIs) or Angiotensin receptor blockers (ARBs) and statins were used in 85%, 79%, 66%, and 43% of cases respectively. Intravenous fibrinolytic was given in 32% of the eligible patients with ST-segment elevation. Per cutaneous coronary interventions (PCI) were applied in four patients within the first month after AMI. Hospital location in relation to a patient's place of residence and manner of transportation to hospital did not influence the time delay between the onset of symptoms to the start of hospital treatment.

Nijjae et al[11] reported in unadjusted analyses, the composite outcome and its components of mortality and recurrent MI were significantly different between South Asians and Whites. Among those with diabetes, South Asian patients were 20% more likely to experience a recurrent AMI but White patients were 11% more likely to reach the composite outcome and 42% more likely to die. Occurrence of CHF did not differ significantly between South Asian and White patients. With in both ethnic groups, the presence of diabetes was associated with a

higher risk of developing the composite outcome. Compared to South Asian patients without diabetes, unadjusted analyses showed those with diabetes were 28% more likely to reach the composite outcome and 50% more likely to die. Results among White patients showed similar trends with those of South Asian patients. When examining adjusted regression models with in each ethnic group, patients with diabetes had a higher risk of reaching the composite outcome. Short-term mortality was not significantly different in South Asian patients with diabetes when compared to their non-diabetic counterparts. In comparing long-term mortality, both South Asians and Whites with diabetes had a higher risk than patients without diabetes.

Mobitz type-I or Wencheback AV block occurs in approximately in 10% of patients who have an AMI and accounts for 90% of all patients who have an AMI and a second degree AV block. Mobitz type-II AV block accounts for 10% of all second degree AV block (overall rate of less than 1 % in the setting of AMI)[3]. A third degree AV block, or a complete heart block occurs in 5-15% of patients who have an AMI and may occur in patients with anterior or inferior infarction. The mortality rate for patients with inferior wall who develop complete heart block is approximately 15% unless a co-existing RV infarction is present, in which case the mortality rate is higher than this[3]. Isolated left anterior hemiblock occurs in 3-5% of patients with AMI, progression to complete AV block is uncommon. Isolated left posterior hemiblock occurs in only 1-2% of patients who have an AMI. New right bundle branch block is seen in approximately 2% of patients with AMI and suggest a large infracted territory[3]. In 40% of patients a trifasicular block progresses to a complete heart block. Accelerated idioventricular rhythm is seen in as many as 20% of patient who have an AMI. Non sustained VT in the immediate peri-infarction period does not appear to be associated with an increased mortality risk, however non sustained ventricular tachycardia occurring more than 48 hours after infarction in patients with LV systolic dysfunction (LVEF < 40%) posses an increased risk for sudden cardiac death[3]. Sustained polymorphic VT after an AMI is associated with a hospital mortality rate of 20%. Incidence of primary VF is 4.5% and is greatest in the first hour after the onset of acute MI, thereafter the incidence rapidly declines. Approximately 60% episodes occur within 4 hours, and 80% occurs within 12 hours. Secondary or late VF occurs more than 48 hours after MI and is usually associated with pump failure and cardiogenic shock[3]. Factors associated with an increased risk of secondary VF are, a large infract, an intraventricular conduction delay, and an antero septal MI. Secondary VF due to cardiogenic shock is associated with an in hospital mortality rate of 40-60%. Early use of Beta -Blockers in patients with acute MI reduces the incidence of VF as well as death[12]. Re-occlusion of an infarct related artery (IRA) occurs in 5-30% of patients following fibrinolytic therapy. These patients also have a poor outcome[13].

Out of the mechanical complications, free wall rupture is the most serious complication after acute MI and it accounts for 15-30% of the deaths associated with acute MI. The overall incidence of ventricle free wall rupture ranges from 0.8-6.2% and incidence of this complication has declined with better blood pressure control, increased use of re-perfusion therapy, Beta- Blockers, ACEI and decreased use of heparin[14]. Data from the National Registry of Myocardial Infarction (NRMI) showed an elevated incidence of in hospital mortality among patients who received thrombolytic therapy (12.1%) than among patients who did not (6.1%)[15].

In the Thrombolysis in Myocardial Infarction Phase II (TIMI II) trial, 16% of patient died from cardiac rupture within 18 hours of therapy[16]. Chances of mechanical complications increases with advance age (> 70 years), female sex, no previous MI, Q wave on ECG, hypertension during the initial phase of ST elevation MI, corticosteroid or NSAID use and fibrinolytic therapy more than 14 hours after ST elevation MI onset. Patients with a history of angina pectoris, previous MI, multi vessels coronary artery disease, and congestive heart failure are less likely than others to develop free wall rupture of the LV because they develop collaterals and ischemic pre-conditioning[15,17,18].

Ventricular septal rupture (VSR) is an infrequent but life threatening complication of AMI. In pre-thrombolytic era VSR occurred in 1-13% of individuals with AMIs. The incidence declined with thrombolytic therapy (0.2-

0.34%) because of improved re-perfusion and myocardial salvage. The bimodal distribution of VSR is characterized by a high incidence in the first 24 hours, with an other peak on days 3-5 and rarely more than 2 weeks after MI. In patients receiving thrombolytics, the median time from the onset of symptoms of AMI to sepal rupture was one day in the Global Utilization of Steptokinase and TPA (tissue plasminogen activator) for Occluded Coronary arteries (GUSTO-I) trial[19] and 16 hours in the SHOCK trial[20]. Risk factors for septal rupture include advance age (>65 years) female sex, single vessel disease, extensive MI, and poor septal collateral circulation[21,22]. Lemory et al reported a 30 day survival rate of 24% in patients treated medically compared with 47% in those treated surgically[23]. In the SHOCK trial patients with cardiogenic shock due to septal rupture had higher rate of in hospital mortality (87.3%) than rate of all other causes of cardiogenic shock (59.2% with pure LV failure and 55.1% with acute MR)[20].

Mitral regurgitation (MR) is a common complication of AMI that results from local and global LV remodeling and is an independent predictor of heart failure and death. MR typically occurs 7-10 days after an AMI. Papillary muscle rupture resulting in MR occurs within 1-14 days (median 1 day). Mild to moderate MR is often clinically silent and detected only on Doppler during echocardiography. During the GUSTO trial incidence of MR in patients receiving thrombolytic therapy was 1-73%. The SHOCK trial which included MI patients presenting with cardiogenic shock noted a 39.1% incidence of moderate to severe MR[24]. Kinn et al reported that re-perfusion with angioplasty resulted in an 82% decrease in the rate of acute MR, as compared with thrombolytic therapy (0.31% VS 1.73%)[25]. Risk factors for MR are, advanced age, female sex, large infarct, previous MI, recurrent ischemia, multi-vessel coronary artery disease and congestive heart failure (CHF).

Left ventricular mural thrombus is a frequent complication after anterior wall MI and its incidence ranges 20-40% and may reach up to 60% in patients with large anterior wall AMIs who are not treated with anticoagulant therapy. Anticoagulant therapy may substantially decreases the rate of embolic events by 33% compared with no coagulation. Factors contributing to left ventricular mural thrombus (LVMT) formation include LV regional wall akinesia or dyskinesia with blood stasis, injury to and inflammation of the endocardial tissue that provides a thrombogenic surface and a hyper coagulable state.

CONCLUSIONS

Cardiogenic shock was the most frequent complication. Major predictors of in-hospital mortality were anterior wall myocardial infarction, patients not receiving streptokinase therapy and old age.

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Take calculated risks. That is quite different from being rash.

(George S. Patton 1885 - 1945)