COMPLICATIONS AND MORTALITY IN ST-SEGMENT ELEVATION ACUTE MYOCARDIAL INFARCTION IN DIABETIC AND NON-DIABETIC PATIENTS

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SUMMARY: The difference in environmental conditions along with foods in the subcontinent than other parts of the world incited this work to see how complications and outcome differ in diabetic and non-diabetic patients. 240 patients (76 diabetic and 164 non-diabetic) suffering from ST-segment Elevation acute myocardial infarction were included in the study. Complications of Acute Myocardial Infarction (AMI) and the outcome were compared between diabetics and non-diabetic patients. Different complications studied varied significantly (P<0.001) within diabetics, non-diabetics and in overall after controlling for diabetes. Complications showed similar pattern (heterogeneity test P >0.5) in diabetic and non-diabetic patients. The abnormalities including Cardiogenic shock (OR= 1.9; 95%CI = 0.85-4.22), left ventricular failure (OR = 2.5), re-infarction (OR= 2.2), arrhythmia (OR= 2.04) and ventricular septal defect (OR= 2.17) were 4.2, 4.7, 21.3, 4.2 and 85.24 times higher in diabetics, respectively. However, occurrence of post myocardial angina (OR=0.38) was low in diabetics than non-diabetics. Odds of having diastolic dysfunction were 1.8 times higher in diabetic patients. The moderate and severe LV-dysfunction was 3.3 and 2.5 times higher diabetics, while mild LV-dysfunction in was 2.1 times higher in non-diabetics. Mortality due to STEMI in diabetics was 2.3 times higher than in non-diabetics. Mortality varied significantly between different age groups in non-diabetics and in overall after controlling for diabetes. In non-diabetic group, mortality was 8.4 times higher in patients those were not given streptokinase than those were given streptokinase, while in diabetic group it was 2.5 times higher in patients were not given streptokinase than those were given streptokinase. The results indicate that the diabetics have higher risk of mortality. Inferior infarction is more serious in diabetics than non-diabetics and chances of survival in streptokinase treated patients is five times in non-diabetic while it about two times in diabetics. The results suggest the importance of streptokinase treatment in patients having ST-segment Elevation Acute Myocardial Infarction.

Key words: Diabetic, non-diabetic, complications, mortality, streptokinase.

INTRODUCTION

Acute myocardial infarction (AMI) is one of the leading cause of all acute emergencies and is becoming an important public health problem in the developing countries (1). Diabetes is a universal problem and is becoming a major concern at old age especially in obese people and in people with sedentary life style. The risk of AMI is 2-4 times higher in diabetics. The coronary artery disease is much more serious in diabetics with about 4 times higher morbidity/mortality in men, while 8 times in women (2-4). Acute pulmonary edema, cardiogenic shock, arrhythmia, re-infarction and cerebral infarction are serious complications in diabetics (5).

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Table 1: Comparison of in-hospital complications between diabetic and non-diabetic patients admitted with STEMI.

Parameter	Non-diabetic (n = 164) % (No.)	Diabetic (n = 76) % (No.)	Row Statistic	overall (n = 240)
Cardiogenic Shock	9.8 (16)	17.1 (13)	OR = 1.91 95% CI = 0.85 to 4.22	12.1 (29)
Left Ventricular Failure	17.1** (28)	34.2** (26)	OR = 2.5 95% CI = 1.34 to 4.72	22.5** (54)
Post Myocardial Angina	12.8** (21)	5.3* (4)	OR= 0.38 [reciprocal = 2.64] 95% CI = 0.11 to 1.08	10.4 (25)
Re-infarction	1.2* (2)	2.6* (2)	OR = 2.19 95% CI = 0.22 to 21.29	1.6* (4)
Arrhythmias	11.6** (19)	21.1** (16)	OR = 2.04 95% CI = 0.96 to 4.24	14.6** (35)
VSD	0.6* (1)	1.3* (1)	OR = 2.17 95% CI = 0.06 to 85.24	0.8* (2)
Thrombo-embolic Phenomenon	0.0* (0)	1.3* (1)	OR = infinity 95% CI = 0.1136 to infinity	0.5* (1)
Column Statistic	L-chi-sq. P < 0.001	L-chi-sq.P < 0.001		L-chi-sq. P < 0.001
	M-H chi-sq. P < 0.001			
Sub-classification of Arrhythmia				
Ventricular Fibrillation	2.4 (4)	10.5 (8)	OR = 4.71 95% CI = 1.37 to 18.32	5.0 (12)
Ventricular Tachycardia	6.1 (10)	10.5 (8)	OR = 1.81 95% CI = 0.66 to 4.86	7.5* (18)
Complete AV block	3.0 (5)	0.0* (0)	OR = 0.0 95% CI = 0.00 to 1.75	2.1* (5)
Statistic	Fisher's P = 0.26	Fisher's P = 0.004		Fisher's P = 0.018
	M-H chi-sq. P = 0.02			
Diastolic Dysfunction	50.6 (83)	64.5 (49)	OR = 1.77 95% CI = 1.01 to 3.12	71.7 (132)
Systolic Function by Ejection Fraction				
Mild LV dysfunction (>40)	17.7 ** (29)	9.2 * (7)	OR = 0.47 [reciprocal = 2.12] 95% CI = 0.18 to 1.10	15.0 (36)
Moderate LV dysfunction (30 -40)	9.8 (16)	26.3 (20)	OR = 3.30 95% CI = 1.58 to 6.90	15.0 (36)
Severe LV dysfunction(< 30)	10.4 (17)	22.4 (17)	OR = 2.49 95% CI = 1.18 to 5.24	14.2 (34)
Column Statistic	Chi-sq. P = 0.055	Chi-sq. P = 0.020		Chi-sq. P = 0.957
	M-H chi-sq. P = 0.957			

In this Table ** = significantly higher; * = significantly lower; OR = odd ratio; chi-sq. = Chi-square; L-chi-sq. = Likelihood chi-square; H-chi-sq. = Heterogeneity chi-square

In patients with AMI, heart failure is characterized by diastolic dysfunction alone or systolic and diastolic dysfunctions together. About 3% of the adult patients develop systolic dysfunction which recognized by echocardiography and is asymptomatic in about of them (6). Re-infarction is diagnosed by persistent and typical severe chest pain along with re-elevation of ST-segment and increased concentrations of cardiac markers in the blood. AMI remains one of the leading cause of mortality, especially in elderly patients where mortality can be 9 times higher than patients of less than 65 years of age (7).The symptoms of AMI are late in diabetics, thus causes delay in fibrinolytic therapy and PTCA (8). It has been reported that diabetic patients with AMI should be administered thrombolytic agents very early. Such a practice can reduce mortality in them (9-10). Early therapy with Statin has been found to reduce the recurrent ischemia (11).

There have been a number studies on complications of AMI in diabetic and non-diabetic patients in different countries but such studies are lacking in Pakistan. Therefore, the aim of the study was to compare and study the nature of complications in diabetic and non-diabetic patients in Pakistan. The response to treatment in diabetic and non-diabetic patients and the mortality were also compared.

MATERIAL AND METHODS

The study was carried out on 240 consecutive patients (diabetic and non-diabetic) admitted in Cardiac Center, Divisional Headquarter Hospital, Faisalabad having acute ST-segment elevation myocardial infarction (STEMI). Patients included in the study were from 15 to 75 years of age, both male and female, with history of chest pain of more than 30 minutes but less than 24 hours along with ST-segment elevation. Oral permission of each patient was sought to include them in the study. Complete history of each patient was recorded at the time of hospital admission. A 12-lead ECG of each patient was recorded at the time of hospital admission. The patients were divided into four groups on the basis of ST-segment elevation in different leads. ST-segment elevation in leads V1-V6 (anterior AMI), in II, III, aVF (Inferior AMI), in II, III, aVF+ V4R (Inferior + Right ventricular AMI) and in I, aVL, V5, V6 (Lateral AMI).

Blood samples of 5ml were collected from the patients under study. These samples were analyzed for serum CK and CK-MB and Trop-T by using commercially available kits (Human, Randox, respectively). Blood glucose was determined in serum samples obtained from patients suspected for diabetes using commercial kits (Centronic). HbA1c was determined on whole blood samples by using commercially available kits (Bicon). Patients were declared diabetics on the basis of having established diabetes and undergoing treatment for the same or with fasting plasma glucose more than 126 mg/dl (more than 7.0 mmol/L) or two hours postprandial plasma glucose more than 200 mg/dl (11.1 mmol/L) checked at the time of hospital admission (12). All these patients having HbA1c more than 7.0 were taken as diabetic (13). Patients were not included in the study if they had old myocardial infarction, non-STEMI, unstable angina, valvular heart diseases, cardiomyopathy, and any pre-existing systemic end stage disease, e.g., malignancy and associated comorbidity, i.e., chronic renal failure and chronic liver disease.

Patients were thrombolysed (Streptokinase), if there were no contraindication to thrombolysis. Other standard treatment of acute MI was given to all patients. Patients were examined daily, while cardiac monitoring was done for first 72 hours. However, if there was any rhythm disturbance, cardiac monitoring continued as long as the patients stayed in the hospital. All patients underwent 2-D M-mode echocardiography on 2nd or 3rd day when patient became stable and in unstable patients, especially with mechanical complications, it was done immediately. Systolic LV function was assessed by ejection fraction and by Quinon's method on 2-D echocardiography (14). LV systolic dysfunction was graded as sever (EF <30 %), moderate (EF 30 – 39 %), mild (EF 40 – 49 %) and normal (EF >50 %). Diastolic LV dysfunction was assessed by recording "E" and "A" velocity by pulse wave Doppler interrogation. Blood glucose and HbA1c was determined by using commercial kits (Centronic; Bicon).

The treatment given to these patients included aspirin (given to 100% of patients), streptokinase, statins, ACE inhibitors, nitrates and beta blocker (given to 91.2%, 84.2%, 80.4%, 76.7 and 62.9% patients), respectively (Table 6). Other treatment/drugs listed in the Table were given to less than 40% of the patients. All the diabetic patients were given insulin-glucose and insulin therapy as previously described (15).

These data were analyzed by using chi-square test (Fisher, Pearson and Likelihood-ratio). Odd ratios, Attributable fractions, prevented fractions were also worked out to interpret results by using computer software package (16).

RESULTS

In-hospital Complications

Different complications studied varied significantly ($P<0.001$) in diabetics, non-diabetic and after controlling

Table 2: Comparison of mortality between diabetic and non-diabetic patients of different age and site of infarction.

Age Group	Non-diabetic (n = 164) % (n)	Diabetic (n = 76) % (n)	Row Statistic	overall % (n)
<44 years	2.6* (1/38)	11.1 (1/9)	OR = 4.63 95% CI = 0.11 to 184.63	4.3* (2/47)
45 – 54 years	4.3* (2/47)	14.3 (2/14)	OR = 3.75 95% CI = 0.35 to 38.11	6.6* (4/61)
55 – 64 years	20.0 (8/40)	32.4 (11/34)	OR = 1.91 95% CI = 0.65 to 5.68	25.7** (19/74)
65 – 74 years	28.1** (9/32)	28.6 (4/14)	OR = 1.02 95% CI = 0.23 to 4.15	28.3** (13/46)
> 75 years	14.3 (1/7)	20.0 (1/5)	OR = 1.50 95% CI = 0.03 to 67.71	16.7 (2/12)
Column Statistic	Fisher's P = 0.002 M-H trend chi-sq. P = 0.001	Fisher's P = 0.64 MH trend chi-sq. P = 0.314		Fisher's P = 0.0005 MH trend chi-sq. P < 0.001
	M-H chi-sq. P = 0.003 M-H trend chi-sq. P = 0.001			
Overall	**12.8 (21/164)**	**25.0 (19/76)**	**OR = 2.27 95% CI = 1.12 to 4.55**	**16.7 (40/240)**
SITE OF INFARCTION				
Anterior	16.5 (15/91)	23.7 (9/38)	OR = 1.6 95% CI = 0.60 to 3.99	18.6 (24/129)
Inferior	7.8 (4/51)	36.4 (8/22)	OR = 6.7 95% CI = 1.71 to 28.11	16.4 (12/73)
Inferior+ right ventricular	14.3 (2/14)	20.0 (1/5)	OR = 1.50 95% CI = 0.04 to 24.18	15.8 (3/19)
Lateral	0.0 (0/8)	9.1 (1/11)	OR = infinity 95% CI = 0.0383 to infinity	5.3 (1/19)
Column Statistic	Fisher's P= 0.4107	Fisher's P = 0.3933		Fisher's P= 0.623
	Mantel-Haenszel chi-square P = 0.373			

In this Table ** = significantly higher; * = significantly lower; OR = odd ratio; chi-sq. = Chi-square

for diabetes (Table 1). Left ventricular failure and arrhythmia were observed in higher (P<0.001), while post myocardial angina, re-infarction, VSD and thrombo-embolic phenomenon were observed in less (P<0.001) number of diabetic patients and in all patients after controlling for diabetes status. These complications showed similar pattern and differed in similar fashion (heterogeneity test P >0.5) in diabetic and non-diabetic groups with only difference that postmyocardial angina was observed in higher (P<0.001) number of non-diabetic patients (Table 1). Comparison of complications between diabetic and non-diabetic patients showed significant differences. Cardiogenic shock (OR= 1.9; 95%CI = 0.85-4.22), left ventricular failure (OR = 2.5; 95%CI =1.34-4.72), re-infarction (OR= 2.2; 95%CI =0.22-21.29), arrhythmia (OR= 2.04; 95%CI =0.96-4.24) and ventricular septal defect (OR= 2.17; 95%CI =0.06-85.24) were as high as 4.2, 4.7, 21.3, 4.2 and 85.24 times in diabetics, respectively (Table 1). However, occurrence of post myocardial angina (OR=0.38; 95%CI = 0.11-1.08) was low in diabetics.

Table 3: Comparison of mortality between diabetic and non-diabetic patients admitted with STEMI given or not given streptokinase.

	Mortality		Overall mortality
	Non-diabetic	Diabetic	
Streptokinase not given	46.7 (7/15)	42.9 (3/7)	45.45 (10/22)
Streptokinase given	9.4 (14/149)	23.2 (16/69)	13.76 (30/218)
	Chi-sq. P < 0.001 OR = 8.44 95% CI = 2.51 to 27.07	Fisher P = 0.357 OR = 2.48 95% CI = 0.42 to 13.07	chi-sq. P < 0.001 OR = 5.22 95% CI = 2.00 to 13.22
	Mantel-Haenszel chi-square P < 0.001 Heterogeneity of odds ratio chi-sq P = 0.218 Mantel-Haenszel odds ratio = 5.15		

In this Table OR = odd ratio; chi-sq. = Chi-square

The distribution of type of arrhythmia showed significant difference in diabetics (P<0.05) and after controlling for diabetes (P<0.05), while this was non-significant in non-diabetic group (Table 1). A significant heterogeneity test indicates difference (P<0.05) in types of arrhythmia between non-diabetic and diabetic patients and suggests that there is significant interaction present between diabetes and arrhythmia. In diabetic group, complete AV block was observed in significantly (P<0.005) less number of patients. Similarly complete AV block was observed in significantly (P<0.05) less number of all patients combined, while ventricular tachycardia was observed in significantly (P<0.05) higher number (Table 1). Among the types of arrhythmia, ventricular fibrillation was 4.7 times higher in diabetics (OR = 4.71).

Odds of having diastolic dysfunction were 1.8 times in diabetic patients (Table 1). Systolic dysfunction was classified into mild, moderate and severe. The systolic LV dysfunction did not differ in non-diabetics and after controlling for diabetes. However, among diabetics, mild LV dysfunction was observed in significantly less (P<0.05) number of patients. A significant (P<0.05) heterogeneity test indicates difference in types of LV dysfunction between non-diabetic and diabetic patients and suggest a significant interaction of diabetes with left ventricular systolic dysfunction.

Among systolic dysfunction, moderate and severe LV-dysfunction was observed in 3.3 and 2.5 times higher in diabetics, while mild LV-dysfunction was 2.1 times higher in non-diabetics (Table 1).

Mortality in Diabetics and Non-diabetics

Mortality due to STEMI in diabetics was 2.3 times (CI = 1.12-4.55) higher than in non-diabetics (Table 2).

Mortality varied significantly between different age groups in non-diabetics and after controlling for diabetes but showed non-significant difference in diabetics. Mortality increased significantly with the increase in age (upto the age 74 years) in non-diabetics and after controlling for diabetes and peaked in patients of 65-74 years, while lower in patients of <54 years of age (Table 2). A non-significant test of heterogeneity showed no interaction of mortality with diabetes and age (Table 2). The mortality in diabetics was 4.6 and 3.8 times higher at age below 44 and 45-54 years and was higher in other age groups also (Table 2).

Mortality in diabetics, non-diabetics and after controlling for diabetes showed non-significant difference between infarction sites (Table 2). However, in inferior infarction group (row comparison), 6.7 times higher mortality was observed in diabetics than non-diabetics (Table 2). A non-significant test of heterogeneity showed no interaction of mortality with diabetes and site of infarction.

A total of 149 (91%) non-diabetic patients and 69 (91%) diabetic patients received the streptokinase therapy (Table 3). In non-diabetic group, mortality was 8.4

times higher (P<0.001) in patients not given streptokinase (CI = 2.51-27.07) than those given streptokinase, while in diabetic group it was 2.5 times higher in patients not given streptokinase than those given streptokinase. After controlling for diabetes, mortality was 5.2 times (P<0.0001) higher in patients not given streptokinase than those given streptokinase (Table 3).

Table 6: Treatment given to patient included in the study admitted with STEMI.

Treatment Given	Total Patients	
	No.	%
Streptokinase	218	90.83
Dispirin / Aspirin	240	100
Nitrates	184	76.7
Beta-blockers	151	62.9
ACE inhibitors	193	80.4
Calcium Channel Blockers (Diltiazem, Amlodipine)	43	17.9
Lipid Lowering Agents (Statins)	202	84.2
Inotropic Support (Dobutamine, Dopamine)	46	19.2
Diuretics (Furosemide, Spironolactone)	54	22.5

DISCUSSION

Among patients admitted with STEMI 31.7% were diabetic. This number is about 10% lower than patients admitted with AMI in Karachi (43%) (17). However, number of diabetics in STEMI observed in our study were almost similar to another study abroad (27%) (18). This difference in percentage compared with a local study from Karachi can be related with the status of diabetes and its management in different areas of the country.

The most significant complications observed during present study were left ventricular failure and arrhythmia, while less significant were post myocardial angina, re-infarction, ventricular septal defect and thrombo-embolic phenomenon. These complications showed similar patterns in diabetic and non-diabetic patients as heterogeneity test was non-significant. These results of ventricular failure and ventricular tachycardia were almost similar to earlier study from Pakistan suggesting their significance in people of Pakistan (19). During present study, post MI angina did not vary between diabetic and non-diabetic suggesting less occurrence of angina among diabetics is probably not appropriate. Therefore, it needs further studies to clarify about the rate of occurrence of post myocardial angina in diabetics and non-diabetics in different geo-environmental conditions and genetics of people. Results of present study suggest higher chances of left ventricular failure and arrhythmia in diabetic than non-diabetic patients. Results also showed a significant interaction of arrhythmia and diabetes. These findings are further strengthened by the fact that diastolic dysfunction and moderate to sever LV systolic dysfunctions were the significant problems in diabetics that suggest a poor ventricular function in them and thus agrees with other reports of impaired LV systolic and diastolic function (20). It has been stated that in diabetics substances from endothelium may affects the myocardium. The endothelin and agniotensin II have been found responsible in causing myocardial hypertrophy and increase in interstitial connective tissue (20). It has been reported that left ventricular ejection fraction is reduced in patients with diabetes (21). Results also revealed that among different types of arrhythmia, about 77% of diabetics (OR = 4.7) suffer from ventricular fibrillation.

The mortality in diabetics was two times higher than in non-diabetics with an overall mortality of 16.7%. These results of higher mortality in diabetics was not different than earlier reports. A study from Greece reported higher in-hospital mortality in diabetics (3). Another study had similar results of higher mortality in diabetic patients (22). It shows that diabetic patients are difficult to deal once they are hospitalized and have relatively poor prognosis. It has been reported that atherosclerosis can develop at higher rate in diabetics that can contribute to significant mortality due to involvement of multiple vessels (23).

Our results showed that mortality can be 4.6 and 3.8 times higher in diabetics at age <44 and 45-54 years, respectively which suggests a higher mortality at young age than reported from other parts of the world. This could be related with the degree of awareness among the people about the cardiac problems. Most people in Pak-

istan rush to hospital only when they already had second or third heart attack. Young people don't consider that they may be suffering from cardiac problem and thus ignore it when they first time experience the signs of heart disease. In most cases, the history shows that patients had first signs of heart disease one-two days earlier and they ignored it and did not consider that they can be victim of heart disease at this age. It has been reported that in-hospital mortality increases with age, i.e., 2.1% at age <55 years to 26.3% at age 85 years or higher (24). Another study reported 1.5, 2.4, 3.4, 11.1 and 19.2% in-hospital mortality in patients of 34-44, 45-54, 55-64, 65-74 or 75 years of age, respectively (25). Mortality did not show statistical difference between diabetic and non-diabetic after 70 years, while a significant and four fold difference in subjects of less than 60 years old and 2.5 fold in subjects aged 61-70 years has been recorded (26). We observed higher mortality in diabetic than non-diabetic at young age which suggests that as the age grows the diabetic and non-diabetic have almost equal chances of expiry but at younger age, it is the diabetic those are at higher risk.

Our results revealed non-significant difference in mortality in diabetics, non-diabetics and after controlling for diabetes due to infarction at different sites. However, we found 6.7 times higher mortality in diabetics in inferior infarction group. Incidently, most of the patients in inferior infarction group presented with serious complications like cardiogenic shock, ventricular tachyarhythmia and left ventricular failure that probably contributed to higher mortality in diabetic patients of this group. However, it may require more studies to clarify the facts.

Studies before the introduction of fibrinolytic therapy have shown 1.5 to 2 times higher mortality in diabetic patients (27). Streptokinase is a fibrinolytic agent and has shown a greater promise in controlling the outcome of AMI. In our study, we found that streptokinase treatment reduced mortality in both diabetic (about 2.5 times) and non-diabetic (about 8.4 times) patients. This reduction in mortality was significant in non-diabetic but not in diabetic group. This suggests the usefulness of streptokinase and has more promising results in non-diabetic subjects. We also found that in streptokinase group, the mortality was 2.9 times higher in diabetics than non-diabetics. This ratio of mortality is lower than recorded in another study (3.2% higher mortality in diabetics) in patients receiving thrombolysis treatment (8). It has been reported that the recovery after intravenous thrombolysis is less frequent in diabetic than in non-diabetic subjects (28). This might be due to the diffuse coronary artery disease, metabolic derangements, complexity of the atherosclerotic plaque, microangiopathy, endothelial dysfunction, impaired fibrinolysis, increased platelet aggregatory activity, and diminished flow reserve in diabetics (29-30).

CONCLUSIONS

Among the patients admitted with STEMI, about 32% were diabetic. In overall, left ventricular failure and arrhythmia were the significant complications. Mortality at young age was significantly higher in diabetics than non-diabetics. Streptokinase reduced mortality in both diabetic (about 2.5 times) and non-diabetic (about 8.4 times) patients.

REFERENCES

1. Hampton J, Gray A: The future of General Medicine; Lesson from an admission ward. J Roy Coll Physcian, 32: 39-43, 1998.

2. Dirkali A, van der Ploeg T, Nangrahary M, Cornel JH, Umans VA. The impact of admission plasma glucose on long-term mortality after STEMI and NSTEMI myocardial infarction. Int J Cardiol,121: 215-7, 2007.

3. Pitsavos C, Kourlaba G, Panagiotakos DB, Stefanadis C: Characteristics and in-hospital mortality of diabetics and nondiabetics with an acute coronary syndrome; the GREECS study. Clin Cardiol, 30: 239-44, 2007.

4. Laing SP, Swerdlow AJ, Slater SD, Bothat JL, Burden AC, Waugh NR, Smith AW, Hill RD, Bingley PJ, Patterson CC, Qiao Z, Keen H: The British Diabetic Association Cohort Study II cause-specific mortality in patients with insulin-treated diabetes mellitus. Diabet Med, 16: 466–471, 1999.

5. Lindeman RD, Romero LJ, Hundley R, Allen AS, Liang HC, Baumgartner RN, et al: Prevalence of type 2 diabetes, the insulin resistance syndrome and coronary heart disease in an elderly, biethnic population. Diabetes Care, 21: 959-966, 1998.

6. Nielsen OW, Hansen JF, Hilden J, Larsen CT, Svanegaard J. Risk assessment of left ventricular systolic dysfunction in primary care cross sectional study evaluating a range of diagnostic tests. BMJ, 320: 220-4, 2000.

7. Maggioni AP, Zuanetti G, Franzosi MG et al: Prevalence and prognostic significance of ventricular arrhythmias after acute

myocardial infarction in the fibrinolytic era. GISSI-2 results. Circulation, 87: 312–22, 1993.

8. Mak KH, Moliterno DJ, Granger CB, Miller DP, White HD, Wilcox RG, et al: Influence of diabetes mellitus on clinical outcome in thrombolytic era of acute MI. Gusto-1 Investigator Global utilization of streptokinase and TPA for occluded coronary arteries. J Am Coll Cardial, 30: 171-179, 1997.

9. Brandle MM, Amann FW, Soloman F: Diabetes mellitus and Coronary Heart Disease. Abteilung for Endorcrinologic / Diabetologic, universitatsspital. Zurich, 129: 700-706, 1999.

10. Tjandrawidjaja MC, Y Fu, Goodman SG, Van De, Werf F, Granger CB: The impact of gender on the treatment and outcomes of patients with early reinfarction after fibrinolysis insights from ASSENT-2. Eur Heart J, 24: 1024-34, 2003.

11. Serruys PW, de Feyter P, Macaya C, Kokott N, Puel J, Vrolix M, et al: Fluvastatin for prevention of cardiac events following successful first percutaneous coronary intervention A randomized controlled trial. JAMA, 287: 3215-3222, 2002.

12. Iqbal MJ, Azhar M, Javed MT, Tahira I. Study on St-segment Elevation Acute Myocardial Infarction (Stemi) in Diabetic and Non-diabetic Patients. Pak J Med Sci, 24: 786-791, 2008.

13. Davidsona MB, Schrigerb DL. Effect of age and race/ethnicity on HbA1c levels in people without known diabetes mellitus: Implications for the diagnosis of diabetes. Diabetes Res Clin Pract, 87: 415-421, 2010.

14. Quinones MA, Waggoner AD, Reduto LA, et al: A new, simplified and accurate method for determining ejection fraction with two dimensional echocardiography. Circulation, 64: 744-53, 1981.

15. Kirk A, Mutrie N, MacIntyre P, Fisher M: Effects of a 12-month physical activity counselling intervention on glycaemic control and on the status of cardiovascular risk factors in people with Type 2 diabetes. Diabetologia, 47: 821-832, 2004.

16. Abramson, JH: WINPEPI (PEPI-for-Windows): computer programs for epidemiologists. Epid Persp and Innov 1, 6, 2004.

17. Tipoo FA, Quraishi AR, Najaf SM, Kazmi KA, Jafary F, Dhakam S, Shafquat A: Outcome of cardiogenic shock complicating acute myocardial infarction. J Coll Physicians Surg Pak, 14: 6-9, 2004.

18.Peterson PN, Spertus JA, Magid DJ, Masoudi FA, Reid K, Hamman RF, Rumsfeld JS: The impact of diabetes on one-year health status outcomes following acute coronary syndrome. BMC Cardiovascular Disorders, 6: 41, 2006.

19. Abid AR, Malick NH , Shahbaz A, Tarin SMA: In-hospital outcome of acute myocardial infarction (ST Segment elevation type) in diabetics and non-diabetics. J Col Phy Sur Pak, 15: 524-527, 2005.

20. Huey BL, Beller GA, Kaiser DL, Gibson RL: A comprehensive analysis of myocardial infarction due to left circumflex artery occlusion: comparison with infarction due to right coronary artery and left anterior descending artery occlusion. J Am Coll Cardiol, 12: 1156–66, 1988.

21. Ishihara M, Sato H, Kawagoe T, Shimatani Y, Kurisu S, Nishioka K, Kouno Y, Umemura T, Nakamura S. Impact of diabetes mellitus on long term survival after acute myocardial infarction in patients with single vessel disease. Heart, 86: 133-138, 2001.

22. Atmaca A, Gogan S, Dagdele S, Kabakci G, Kes S, Nazli N, Erbas T: Management and in-hospital outcome of patients with first episode of acute myocardial infarction: impact of diabetes mellitus. Journal National Med Assoc, 98: 1752-1757, 2006.

23. Jaski BE, Cohen JD, Trausch J et al: Outcome of urgent percutaneous transluminal coronary angioplasty in acute myocardial infarction: comparison of single-vessel versus multivessel coronary artery disease. Am Heart J, 124: 1427–1433, 1999.

24. Boucher JM, Normand Racine, Thao Huynh Thanh, Elham Rahme, James Brophy, Jacques LeLorier, Pierre Théroux: on behalf of the Quebec Acute Coronary Care Working Group Age-related differences in in-hospital mortality and the use of thrombolytic therapy for acute myocardial infarction CMAJ,164: 1285-90, 2001.

25. Polanczyk CA, Johnson PA, Cook EF, Lee TH: A proposed strategy for utilization of creatine kinase-MB and troponin I in the evaluation of acute chest pain. Am J Cardiol, 83: 1175-9, 1999.

26. Donnan PT, Boyle DI, Broomhall J, Hunter K, MacDonald TM, Newton RW, Morris AD: Prognosis following first acute myocardial infarction in Type 2 diabetes: a comparative population study. Diabet Med, 19: 448-55, 2002.

27. Stone PH, Muller JE, Hartwell T, York BJ, Rutherford JD, Parker CB, Turi ZG, Strauss HW, Willerson JT, Robertson T: The effect of diabetes mellitus on prognosis and serial left ventricular function after acute myocardial infarction: contribution of both coronary disease and diastolic left ventricular dysfunction to the adverse prognosis. J Am Coll Cardiol, 14: 49–57, 1989.

28. Angeja BG, Gunda M, Murphy SA, et al: TIMI myocardial perfusion grade and ST segment resolution: association with infarct size as assessed by single photon emission computed tomography imaging. Circulation, 105: 282-5, 2002.

29. Aronson D, Rayfield E, Cheseboro J: Mechanisms determining course and outcome of diabetic patients who have had acute myocardial infarction. Ann Intern Med, 126: 296–306, 1997.

30. Beckman JA, Creager MA, Libby P: Diabetes and atherosclerosis epidemiology, pathophysiology and management. JAMA, 287: 2570-81, 2002.

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