FREQUENCY AND CLINICAL OUTCOME IN CONDUCTION DEFECTS IN ACUTE MYOCARDIAL INFARCTION

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Background: Conduction defects complicating acute myocardial infarction (MI) are frequent and associated with increased mortality and complications. Common conduction defects after acute MI are atrioventricular nodal blocks (1st, 2nd and 3rd degree) and intraventricular conduction defects (right or left bundle branch blocks and hemiblocks). In myocardial infarction occlusion of coronary arteries at different levels affects the conduction system of heart leading to various types of blocks. Conduction defects usually reflect extensive damage to the myocardium. **Methods:** In this descriptive case series with non-probability purposive sampling, 345 cases of acute ST elevation myocardial Infarction were studied at Armed Forces Institute of Cardiology/National Institute of Heart Disease, Rawalpindi from May 2007 to May 2008. ECG was continuously observed in CCU and daily ECGs were done. Conduction defects whether transient or persistent were recorded in pre-designed proforma in addition to other clinical features and associated complications during hospital stay. **Results:** Out of 345 patients, 251 (72.8%) patients received thrombolytic therapy and 61 (17.6%) developed various types of conduction defects (Group A) and 284 had no significant conduction defects (Group B). Isolated complete atrioventricular block (AVB) at the node level occurred in 28 patients (8.1%) mainly in inferior MI. Bundle branches Blocks occurred in 32 (9.2%) patients mostly in Anterior MI. One patient (0.6%) had complete heart block at bundle branch level. All patients with complete atrioventricular block reverted to sinus rhythm except one who required permanent pacemaker. Mortality rate and clinical complications were higher in group A as compared to group B. **Conclusion:** Conduction defects are common even in this thrombolytic era. Patients with conduction defects are at high risk of inhospital complications and mortality. They need close monitoring and optimum clinical care to reduce mortality and morbidity.

Keywords: Conduction defects, Acute myocardial infarction, Bundle branch blocks

INTRODUCTION

The presence of conduction defects complicating acute myocardial infarction (MI) is relatively frequent and is associated with increased short and long term mortality rates.[1–3] Thrombolytic therapy, although, has been established to reduce the mortality in acute myocardial infarction, however its role in reducing the incidence of conduction defects is less clearly defined. Mortality and morbidity associated with conduction defects also remain unchanged.[4] The most commonly observed conduction defects after acute MI include atrioventricular nodal blocks (1st, 2nd and 3rd degree) and intraventricular conduction defects (right or left bundle branch block). In some cases fascicular blocks (anterior and posterior hemiblocks) alone or in combination with right bundle branch block (bifascicular blocks) are also observed.

The atrioventricular (AV) conduction system has a major blood supply from the right coronary artery. The AV node is supplied by the right coronary artery in 90% of cases and by the left circumflex artery in 10%. The bundle of His is supplied mainly by the AV nodal branch of the right coronary artery.[5] The right bundle is a narrow structure, relatively vulnerable to focal ischemia. The blood supply to the proximal segment of the right bundle is derived from the AV nodal artery, whereas the remaining two-thirds of the right bundle is supplied from the septal branches of the left anterior descending artery.[6,7] Therefore both anterior or inferior MI may be complicated by right bundle branch block (BBB) and associated high mortality.[8] The less frequent compromise of the left bundle during acute MI may reflect its diffuse structure. The left anterior fascicle is supplied by the septal branches from the left anterior descending artery and is particularly susceptible to ischemia or infarction. The main left bundle and its posterior fascicle receive a dual blood supply both from septal branches of the left anterior descending artery and the AV nodal artery.[6] Thus, only extensive damage that includes most of the ventricular septum and the anterior wall may interrupt the conduction of the left bundle.[9]

If conduction block occurs at AV node, the escape rhythm originates in AV junction and the QRS is of normal duration but when block occurs distal to AV node, the escape site is ventricular and rhythm is unstable. QRS configuration is abnormal and its duration is prolonged. Patients with the combination of right bundle branch block and either left anterior or left posterior hemiblock along with prolonged PR interval have a particularly high risk of progression to complete heart block.

The prognostic significance and management of these disturbances may vary with the location of the infarction, the type of conduction disturbance, associated clinical features and the extent of hemodynamic compromise.[10] Conduction disturbances resulting in severe bradycardia may simply be managed with intravenous atropine. Complete AV nodal block or compete heart block at bundle branch level with hemodynamic compromise require support of transvenous temporary pacemaker but they usually are transient rarely requiring permanent pacemaker insertion.

The purpose of this study was to document the frequency of various conduction defects in cases of acute myocardial infarction undergoing thrombolytic therapy who were observed in coronary care unit, and to know about their relationship with complications and inhospital clinical outcome.

PATIENTS AND METHODS

From May 2007 to May 2008, 345 patients with acute myocardial infarction reporting to Armed Forces Institute of Cardiology and National Institute of Heart Diseases, Rawalpindi, a tertiary cardiac care centre, were included in this study. It was a descriptive case series. All patients were explained the study in detail in the language they understood and a written informed consent was taken from them. This study was approved by ethical committee of Armed Forces Institute of Cardiology and National Institute of Heart Diseases, Rawalpindi

All patients of both sexes sustaining acute ST elevation myocardial infarction were included in this study and non-probability purposive sampling technique was used. Patients with old established conduction defects based upon their old medical record, patients with advanced heart failure, renal failure and prior coronary artery bypass surgery were excluded from this study.

Acute Myocardial Infarction (AMI) was diagnosed on the basis of recently adopted definition of AMI by ACC/AHA/ESC/WHF task force.[11] ST elevation Myocardial Infarction was defined as typical rise and fall in CK-MB (usually twice the level of upper reference limit) and at least one mm ST rise in two contiguous limb leads or 2 mm rise in two contiguous chest leads. LBBB was defined as the QRS duration of ≥ 0.12 s; a QS or rS complex in lead V1 or an R-wave peak time of ≥ 0.06 s (often with a notched R-wave) in lead I, AVL, V5, or V6 associated with the absence of a Q-wave in the same lead.[12] RBBB was defined as a prolonged QRS duration of ≥ 0.12 s or an rsr', rsR', or rSR' pattern in lead V1 or V2. If this was not present, the R-wave in lead V1 had to be notched with a prolonged R-wave peak time of >0.05 s in lead V1 and a normal peak

time in leads V5 and V6. Leads V6 and I had to show a QRS complex with a wide S-wave (S duration$>$R duration or >0.04 s.[13] Left anterior hemiblock required a leftward shift of the QRS axis $\leq -30°$ and left posterior hemiblock required a rightward shift to $\geq 120°$.[14] Complete AVB was defined as complete dissociation between atrial and ventricular rates with the atrial rate greater than the ventricular rate and narrow escape rhythm.[15] Compete heart block at the bundle branch level was diagnosed when AV dissociation was noted with slow ventricular escape rhythm. The conduction defect was classified as transient when it was not present at the time of discharge and permanent when it was there at the time of discharge or the patient died.

At Emergency Department, a brief history was obtained from each patient presenting with chest pain including presence of risk factors like diabetes, smoking and hypertension and previous history of Ischemic heart disease (IHD). A brief clinical examination was done with emphasis on signs of cardiac failure. Standard 12 leads Electrocardiogram (ECG) was done at Emergency and blood samples were sent to laboratory for cardiac enzymes and baseline biochemical profile. All patients were considered for thrombolytic therapy (Injection Streptokinase 1.5 million units over one hour) in the absence of any contraindications and managed according to standard treatment protocols. All patients underwent continuous ECG monitoring for at least 48 hours on admission to coronary care unit (CCU). ECG was immediately done after thrombolytic therapy, daily during hospital stay and prior to discharge (usually 5–7 days). Additional recordings were made in the event of any arrhythmia or occurrence of any conduction defect or recurrent chest pain. In patients who presented with or developed conduction defects, the site of defect was recorded and defect was classified as AV nodal block, bundle branch block (right or left), bifascicular block, trifascicular block or complete heart block. In case of fluctuating block, the highest (most severe) degree of block was recorded. Patients who developed haemodynamic instability or severe bradycardia (<40 bpm) refractory to atropine were considered for temporary pacemaker support with VVI pacing mode. Permanent pacemaker implantation was considered only if complete AV block persisted after two weeks of observation. Baseline clinical data including all study variables were recorded on a detailed pre-designed research proforma.

SPSS-12 was used to analyse data. Frequency and percentages were calculated for categorical variables like gender, risk factors, types of myocardial infarction and conduction defects.

Mean and standard deviation were calculated for quantitative data like age, blood pressures, Ejection fraction (EF), Cardiac enzymes and other biochemical parameters. The association of certain variables like types of acute MI, types of conduction defects, diabetes and hypertension along with statistical significance were calculated using Pearson Chi Square tests. Means of quantitative data were compared wherever required and *p*-values calculated using Student's *t*-test.

RESULTS

The study included 345 patients whose baseline characteristics are shown in Table-1. Out of 345 patients, 303 (87.8%) patients were males. Hypertensive were 107 (31.01%), diabetics 71 (20.5%), smokers 119 (34.5%), and 55 (15.9%) patients were aged above 70 years. Only 251 (72.8%) patients received thrombolytic therapy rest being late for it or having contraindications. Acute anterior MI was present in 174 (50.4%), acute inferior MI 153 (44.4%) and others 18 (5.2%). Mean hospital stay was 5.7 days. Renal failure occurred in 29 (8.4%) patients and 31 (8.9%) died during their stay in the hospital, main cause being cardiogenic shock and arrhythmia.

Out of 345 patients, 61 (17.6 %) developed various types of conduction defects (Group A) and 284 had no significant conduction defects (Group B). Isolated complete atrioventricular block (AVB) at the node level occurred in 28 patients (8.1%). Block involving the bundle branches occurred in 32 (9.2 %) patients. Among whom 8 (2.3%) had Left bundle branch, 13 (3.7%) had isolated right bundle branch block, and bifascicular block was seen in 5 (1.4%) patients. Bundle branch block in association with complete AV block was present in 6 (1.7%) patients among whom 4 (1.1%) had right BBB, 1 (0.2%) left BBB and 1 (0.2%) had bifascicular block (Table-2). Complete heart block at the bundle branch level occurred only in one (0.2%) patient.

In group A, 32 (52.4%) conduction defects occurred in inferior MI while 27 (44.2%) defects were associated with anterior MI and only 2 (3.3%) defects occurred in anteroinferior MI (Table-3).

Out of 28 cases of complete AV nodal block, 23 had inferior MI, 3 anterior and 2 had combined anterior and inferior infarction. Majority of the conduction defects (24 out of 32) at bundle branch level persisted even at discharge. Except one, all complete AV blocks reverted to sinus rhythm within a mean duration of 3.8 days (range 1-13 days), majority reverting to sinus rhythm within first 48 hours of onset of the complete AV block. Only 23 patients required the support of temporary pacemaker

due to severe bradycardia and haemodynamic compromise.

Among group A, 44 (72.1%) patients were given Injection Streptokinase while 17 patients were not given thrombolytic therapy as being late for it. Only one patient with complete AV block out of 34, required permanent pacemaker which was implanted after 21 days of observation. In associated morbidity, 9 (14.7%) patients developed acute renal failure. Arrhythmias were noted in 09 (14.8%) patients during their hospital course. Fourteen (22.9%) patients among group A died during hospital stay (Table-4).

Table-1: Baseline Characteristics of Patients with Acute Myocardial Infarction (n=345)

Variables	Values
Age(yrs)	59±14.2
>70 years	55 (15.9%)
Males	303 (87.8%)
Diabetics	71 (20.5%)
Hypertensive	107 (31.01%)
Smokers	119 (34.5%)
Previous history of MI	69 (20%)
Hospital stay (days)	5.7±3.92
Heart rate (bpm)	71.43±32.04
Systolic BP (mmHg)	102.95±24.266
Diastolic BP	66.98±15.046
Killip Class	
I	180 (52.17%)
II	83 (24.06%)
III	55 (15.94%)
IV	27 (7.83%)
Thrombolysis eligible	251 (72.8%)
Ejection fraction	37.95±9.24
Blood Sugar Random	189.52±151.73
Types of Myocardial Infarction	
Anterior	174 (50.4%)
Inferior	153 (44.4%)
Anteroinferior	6 (1.7%)
Inferoposterior	12 (3.5%)
Ventricular Fibrillation	29 (8.4%)
Ventricular tachycardia	45 (13%)
Renal Failure	29 (8.4%)
In- hospital deaths	31 (8.9%)

Data presented is either Mean±SD or numbers along with their percentages depending on the type of variable.

Table-2: Types of Conduction Defects in Acute MI

Types of conduction defects	No	Total Percentage (n=345)	Group Percentage (n=61)
Complete AV block	28	8.1	45.9
Bundle Branch Block	32	9.2	52.4
Right Bundle Branch Block	13	3.7	21.3
Left Bundle Branch Block	8	2.3	13.1
Bifascicular Block	5	1.4	8.2
RBBB plus complete AVB	4	1.1	6.5
LBBB plus complete AVB	1	0.2	1.6
Bifascicular with complete AVB	1	0.2	1.6
Complete heart block (at bundle branch level)	1	0.2	1.6

MI= Myocardial Infarction, RBBB= Right bundle branch block, LBBB= left bundle branch block, AVB= Atrioventricular Block

Table-3: Conduction Defects and Types of Myocardial Infarction

Conduction defects	Types of Myocardial Infarction			
	Anterior (n=27)	ant. inf (n=2)	Inferior (n=32)	Total (n=61)
Bifascicular Block	5 (18.5)	0	0	5 (8.2)
Bifascicular plus complete AVB	0	0	1 (3.1)	1 (1.6)
Complete AVB	3 (11)	2 (100)	23 (71.8)	28 (45.9)
complete AVB plus LBBB	0	0	1 (3.1)	1 (1.6
complete AVB plus RBBB	1 (3.7)	0	3 (9.3)	4 (6.5)
Complete heart block at bundle branch level	1 (3.7)	0	0	1 (1.6)
Left Bundle Branch Block	7 (26)	0	1 (3.1)	8 (13.1)
Right Bundle Branch Block	10 (37)	0	3 (9.3)	13 (21.3)

Data presented shows their numbers along with their percentages, RBBB= Right bundle branch block, LBBB= left bundle branch block, AVB= Atrioventricular Block, Ant. Inf= Anterior inferior

Table-4: Comparison of Group A (conduction defects) and Group B (no conduction defects)

Variable	Group A (n=61)	Group B (n=284)	p-value
C K (u/l)	2840±2625	1540±1320	<0.001
CK- MB (u/l)	256± 143	167± 75	<0.001
Renal Failure	9 (14.7)	20 (7.0)	=0.05
Ventricular Fibrillation	9(14.7)	17 (5.9)	<0.001
Deaths	14 (22.9)	17 (5.9)	<0.001

Data presented is either Mean±SD or numbers along with their percentages depending on the type of variable, CK= Creatinine Kinase, CK-MB = Creatinine Kinase-Myocardial Band U/l= units per litre

Table-5: Characteristics of Patients with and without Conduction after Myocardial Infarction

Variable	Group	Normal (n=284)	AV node block (n=28)	BBB (n=32)	p-value
Smoking	Smokers	96 (80.6)	11 (9.2)	12 (10)	
	Non smokers	188 (83.1)	17 (7.5)	20 (8.8)	=0.7
Age	>70	40(72.2)	5 (9.0)	10 (18.2)	
	< 70	244(84.4)	23 (7.9)	22 (7.6)	=0.04
Diabetes	Diabetic	40 (56.3)	8 (11.3)	23 (32.4)	
	Non-diabetics	248 (89.5)	20 (7.2)	09 (3.3)	<0.001
Past h/o MI	past MI	39 (56.5)	8 (11.6)	22 (31.9)	
	no past MI	245 (89.1)	20 (7.3)	12 (3.7)	<0.001
Infarct type	Ant MI	148 (85)	03 (1.7)	23 (13.2)	
	Inf MI	125 (83.3)	20 (13.3)	5 (3.3)	
	Others	11 (55)	5 (25)	4 (20)	<0.001
LVF	LVF present	56 (68.3)	6 (7.3)	20 (24.3)	
	LVF absent	228 (87)	22 (8.4)	12 (4.6)	<0.001
Outcome	Deaths	16 (53.3)	4 (13.3)	10 (33.3)	
	No mortality	268 (85.3)	24 (7.6)	22 (7)	<0.001

AV= atrioventricular , Ant= Anterior, Inf= inferior, LVF = left ventricular failure, MI= Myocardial Infarction, BBB= bundle branch block

DISCUSSION

This case series studied the frequency of conduction defects and their influence on inhospital outcome in cases of acute MI, in whom about 70% received thrombolytic therapy. Our finding of having 8.1% cases of complete AV nodal block and 9.2% cases of bundle branch blocks is lower as compared to previous studies which have reported 9.1–12.8% rate of complete AV nodal block,[10,16] and 10–12% rate of bundle branch block.[1,9] Jones et al studied 556 cases of acute MI and published his study in 1977.[17] He found complete AV Block in 9.15% case, bundle branch block (BBB) in 5.5%, and bifascicular block in 10.6%. Similarly in another study done by Woo on 636 patients of acute MI in Hong Kong, a higher rate of conduction defects was found.[18] Complete AV block was present in 11.3% cases while right BBB was in 12.7% cases and left BBB in 3.3%.These studies were done in the pre-thrombolytic era. This decrease in the frequency of conduction defects in our study is due to reduction in the extent of myocardial injury with the use of early thrombolytic therapy. Our results are comparable with Archbold et al who studied 1225 cases of acute MI and documented

the frequency of cases of various conduction defects. In his study AV nodal blocks were 5.3%, bundle branch block 8.9%, and complete heart block (at bundle branch level) were 1.6%.[19] In a meta analysis by Menie et al comprising of 75993 patients, incidence of AV block was 6.9% and it was associated with significantly high mortality.[20]

In a local study by Bilal et al, 54 cases of acute MI were studied.[21] Complete Av block was present in 11.8%, left BBB in 14.7%, and right BBB 8.8%. All patients with complete block died and mortality was significantly higher in patients with heart blocks. As compared to our study this study showed higher rate conduction defects as only cases with anterior MI were included. First degree heart block was also documented as conduction defect which comprised 26.5% cases against 42.6% total cases of heart blocks.

In our study blocks involving the bundle branches were more common in anterior infarction but blocks at the atrioventricular node occurred almost exclusively in inferior infarction (Table-3). This association was statistically significant (p<0.001). Overall conduction defects (34 out of 61) were more common (55.7%) with inferior myocardial infarction or

its associated combined variants. This finding in our study is consistent with a study by Majumder *et al* carried out in Bangladesh. He found strong association of AV blocks with inferior MI and that of bundle branch blocks with anterior MI and concluded that conduction defects were associated with increased rate of complications and death.[22] Our data in this respect is also very closely similar to the findings of Egcosteguy *et al* who studied 929 cases of acute MI in Brazil and found significant association of AV blocks and BBB with inferior and anterior MI.[23] Similar association were also reported by a previously mentioned study by Archbold *et al*.[19]

Pateints in group A had much higher levels of creatinine kinase (CK) and creatinine kinase – myocardial band (CK-MB) as compared group B (Table-4). This difference was statistically significant (p<0.001). This significantly higher levels of enzymes in group A suggest extensive damage to myocardial tissue involving conduction system. Higher number of patients had renal failure in group A as compared to group B. Our data in this respect is consistent with other studies by Archbold *et al*[19], Thompson *et al*[24] and Opolski *et al*[25].

There was no significant difference between group A and group B in frequency of conduction defects as far as smoking was concerned. In our study bundle branch blocks were more common in patients age more than 70 years, diabetics, previous infarction, and left ventricular failure. Patients with conduction defects (Group A) had a higher mortality as compared to group B (Table-4). This risk was increased for all types of conduction defects being more significant for bundle branch blocks (Table-4). It was due to extensive myocardial tissue damage in such cases. Most of the deaths were due to ventricular arrhythmia and cardiogenic shock. In this respect our data is consistent with other studies which have shown increased mortality in bundle branch blocks and complete heart blocks.[19,22,26,27]

CONCLUSION

Conduction defects complicating acute myocardial infarction are common in this thrombolytic era in our population. Such patients should be closely observed and monitored as they have got higher rate of complications and mortality during hospital course. This study shows a small decline in frequency of conduction defects which reflects beneficial effect of thrombolytic therapy as compared with previous studies which were done in pre-thrombolytic era.

REFERENCES

1. Dubois, C, Picrard LA, Smeets JP, Foidart C, Legrand V, Kulbertus HE. Short & long term prognostic importance of complete bundle branch block complicating acute myocardial infarction. Clin Cardiol 1988;11:292–6.
2. Klein RC, Vera Z, Mason DT. Intraventricular conduction defects in acute myocardial infarction: Incidence, prognosis, and therapy. Am Heart J 1984;108:1007–1.
3. Godman MJ, Lasers BW, Julian DG. Complete bundle branch block complicating acute myocardial infarction. N Engl J Med 1970;282:237–40.
4. Newby KH, Pisano E, Krucoff MW, Green C, Natale A. Incidence and clinical relevance of the occurrence of bundle branch block in patients treated with thrombolytic therapy. Circulation 1996;94:2424–8.
5. Matetzky S, Novikov M, Gruberg L, Freimark D, Feinberg M, Elian D, *et al*. The significance of persistent ST elevation versus early resolution of ST segment elevation after primary PTCA. J Am Coll Cardiol 1999;34:1932-8.
6. Gann D, Balachandran PK, El-Sherif N, Samet P. Prognostic significance of chronic versus acute bundle branch block in acute myocardial infarction. Chest 1975;67:298–303.
7. James TN, Burch GE. Blood supply of the human intraventricular septum. Circulation 1958;17:391– 6.
8. Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheiman MM, DeSanctis RW *et al*. The clinical significance of bundle branch block complicating acute myocardial infarction. II. Indications for temporary and permanent pacemaker insertion. Circulation 1978;58:689–99.
9. Scheinman M, Brenman B. Clinical and anatomic implications of intraventricular conduction block in acute myocardial infarction. Circulation 1972;46:753–60.
10. Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheiman MM, DeSanctis RW *et al*,. The clinical significance of bundle branch block complicating acute myocardial infarction, 1: clinical characteristics, hospital mortality, and one-year follow-up. Circulation. 1978;58:679–88.
11. Thygesen K, Alpert JS, White HD. Universal definition of Myocardial Infarction.J A Coll Cardiol 2007;50:2173–95.
12. Sgarbossa EB, Pinski SL, Barbagelata A, Underwood DA, Gates KB, Topol EJ *et al*, for the GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. N Engl J Med 1996;334:481–7.
13. Willems JL, Robles de Medina EO, Bernard R, Coumel P, Fisch C, Krikler D, *et al*. Criteria for intraventricular conduction disturbances and pre-excitation. World Health Organizational/International Society and Federation for Cardiology Task Force Ad Hoc. J Am Coll Cardiol 1985;5:1261–1275.
14. Rosenbaum MB. The hemiblock, diagnostic criteria and clinical significance. Mod Concepts Cardiovasc Dis 1970;39:141–6.
15. Clemmenson P , Bates ER, Callif RM , Hlatky MA, Aronson L, George BS, Lee KL *et al*. Complete atrioventricular block complicating inferior wall acute myocardial infarction treated with reperfusion therapy. Am J Cardiol. 1991;64:224–30
16. Kostuk WJ, Beanlands DS. Complete heart block associated with acute myocardial infarction. Am J Cardial 1970;26:380–4.
17. Jones ME, Terry G, Kenmure AC. Frequency and significance of conduction defects in acute myocardial infarction. Am Heart J 1977;94:163–7.
18. Woo KS. Conduction defects in acute myocardial infarction in the Chinese in Hong Kong. Int J Cardiol 1990;26:325–34.
19. Archbold RA, Sayer JW, Ray S, Wilkinson P, Ranjadayalan K, Timmis AD. Frequency and prognostic implications of conduction defects in acute myocardial infarction since the introduction of thrombolytic therapy. Eur Heart J 1998;19:893–8.
20. Meine TJ, Al-Khatib SM, Alexander JH, Granger CB, White HD, Kilaru R *et al*. Incidence, predictors, and outcomes of high-degree atrioventricular block complicating acute

myocardial infarction treated with thrombolytic therapy. Am Heart J 2005;149:670–4.

21. Bilal HB, Sultan J, Hassan K, Ovais K, Majeed I. Heart blocks as predictors of Mortality in Acute Myocardial Infarction . J Rawal Med Coll 1999;3(1–2):13–6.

22. Majumder AA, Malik A, Zafar A. Conduction disturbances in acute myocardial infarction: Incidence, site-wise relationship and the influence on in-hospital prognosis. Bangladesh Med Res Counc Bull.1996;22:74–80.

23. Escosteguy CC, Carvalho Mde A, Medronho Rde A, Abreu LM, Monteiro Filho MY. Bundle branch and atrioventricular block as complications of acute myocardial infarction in the thrombolytic era. Arq Bras Cardiol 2001;76:291–6.

24. Thompson PL, Fletcher EE, Katavatis V. Enzymatic indices of myocardial necrosis: influence on short- and long-term prognosis after myocardial infarction. Circulation 1979;59:113–19.

25. Opolski G, Kraska T, Ostrzycki A, Zieliński T, Korewicki J. The effect of infarct size on atrioventricular and intraventricular conduction disturbances in acute myocardial infarction. Int J Cardiol.1986;10:141–7.

26. Nicod P, Gilpin E, Dittrich H, Polikar R, Henning H, Ross J. Long-term outcome in patients with inferior myocardial infarction and complete atrioventricular block. J Am Coll Cardiol 1988;12:589–94.

27. Coll JJ, Weinberg SL. The incidence and mortality of intraventricular conduction defects in acute myocardial infarction. Am J Cardiol 1972;29:344–50.

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