Journal of Cardiovascular Diseases & Diagnosis

Khaznadar and Mahmood, J Cardiovasc Dis Diagn 2019, 7:4

Research Article Open Access

Prevalence of Conduction Defects in Patients with Acute ST Elevation Myocardial Infarction

Khaznadar AAJ[1*] **and Mahmood KH**[2]

[1]*Department of Medicine, College of Medicine, University of Sulaimanyia, Iraq*

[2]*General Teaching Hospital, Sulaymaniyah, Iraq*

[*]**Corresponding author:** Khaznadar AAJ, Department of Medicine, College of Medicine, University of Sulaimanyia, Iraq, Tel: + 9647701577711; E-mail: amanjxaznadar@yahoo.com

Received: June 14, 2019; **Accepted:** July 02, 2019; **Published:** July 10, 2019

Abstract

Background: Conduction defects including various types of atrioventricular blocks and bundle branch blocks, may occur as complications of acute ST elevation myocardial infarction, and are associated with increased short- and long-term mortality rates.

Objectives: To determine the prevalence of conduction defects in patients with STEMI during hospital stay.

Materials and Methods: This prospective study included 100 consecutive cases (72 males, 28 females) of STEMI at Slemani cardiac center, with mean age of (60.06±12.86 years). The initial ECG was done immediately after the patient's admission, observation of patients performed in CCU and daily ECGs were done. Conduction defects whither transient or present on discharge were recorded.

Results: Out of 100 patients, 78% of patients had no significant conduction defects, 22% of patients developed various types of conduction defects, of which 10 patients (45.45%) had transient conduction defects, and 12 patients (54.5%) had permanent conduction defects. 50% of conduction defects were atrioventricular blocks all with inferior MI, the other 50% were intraventricular blocks. patients with conduction defects had significant higher mortality rates. the mean age of patients with conduction defects (mean=65.1year) was higher than those without conduction defects (mean=58.6 year).

Conclusion: Conduction defects are frequent complications of myocardial infarction in Slemani, even with reperfusion therapy, associated with high mortality, and their prevalence is increased with increasing age. All atrioventricular blocks had occurred with inferior STEMI while intraventricular blocks had occurred with variable types of STEMI.

Keywords: STEMI; Conduction defects; Sulaimanyia, Iraq

Introduction

Wilhelm His had suggested in1899 that a lesion of the bundle he had described might be the cause of Adams-Stokes disease. Evidence accumulated slowly until 1930 that the errors was finally corrected [1]. The presence of conduction defects complicating acute myocardial infarction (MI) is relatively frequent and is associated with increased short- and long-term mortality rates [2-4]. The important finding of heart block in patients with acute MI has some remarkable prognostic implications [5]. The initial data concerning the frequency of heart block after acute MI were largely derived from studies performed before the development of acute revascularization strategies in the 1990 [2,4,6-12].

Thus, only extensive damage that includes most of the ventricular septum and the anterior wall may interrupt the conduction of the left bundle [13]. Regarding conduction disturbances and infarct location, the clinical management of patients with conduction abnormalities after an MI depends in part upon the location of the infarct. Second- or third-degree AV block associated with inferior wall MI is located above the His bundle in 90 percent of patients [12,14]. AV block associated with anterior MI is more often located below the AV node [12].

Mobitz type I (Wenckebach), and complete heart block are commonly seen, since the SA node, AV node, and His bundle are primarily supplied by the RCA [14]. Less commonly, anterior MI produces first degree AV block below the level of the AV node, a situation that should be presence of a widened QRS complex [15]. High degree AV block: High (second or third) degree AV block occurs in approximately 9.8 percent of patients with an inferior MI who receive thrombolytic therapy [15]. Approximately one-half of cases are present on admission and one-half develop in the 24 hours after thrombolytic therapy is given [16]. Inferior MI is typically associated with the more benign second-degree AV block of the Wenckebach type (Mobitz type 1); Mobitz type II is uncommon in this setting, generally occurring with anterior MI. Mobitz type I block is usually transient, resolving in most cases within five days. Rarely, RCA occlusion produces complete heart block (CHB) that is usually transient but may persist. The latter finding suggests concurrent involvement of the left coronary system, resulting in poor collateral flow [15].

Anterior MI

Serious conduction disturbances more commonly occur with anteroseptal MI, and the degree of arrhythmic complications is usually directly related to the extent of infarction [17]. High degree AV block: High (second or third) degree AV block occurs in approximately 3.2 percent of patients with an anterior MI who receive thrombolytic therapy [15]. Second degree AV block with anterior MI is usually at the level of the AV node or below and is almost exclusively a Mobitz type II block. The clinical course may be unpredictable, with CHB developing with little warning. AV nodal Wenckebach can occur with occlusion of the left circumflex artery in the 10 percent of individuals whose AV node is supplied by this artery [9].

Problems with the diagnosis of acute STEMI and conduction defects

The electrocardiographic (ECG) diagnosis of MI is more difficult when the baseline ECG shows a bundle branch block pattern that may precede or be a complication of the infarct or the patient has a paced rhythm [18-22]. With incomplete blocks, the QRS interval is between 100-120 msec. Partial fascicular blocks in the left bundle system (left anterior or posterior fascicular blocks) associated with shifts in the frontal plane QRS axis (leftward or rightward, respectively). The presence of a prolonged PR interval and bi-fascicular block does not necessarily indicate tri-fascicular involvement [23].

Materials and Methods

This prospective study carried out in Slemani cardiac center in Slemani during 1st January 2015 to 1st November 2015. A total number of 100 patients (72 males and 28 females) admitted to the coronary care unit (CCU) of Slemani cardiac center were included in the study after obtaining an informed consent, all cases were diagnosed with ST Elevation Myocardial Infarction (STEMI), a brief history was obtained from each patient presenting with chest pain including presence of risk factors like diabetes, smoking, hypertension and previous history of Ischemic heart disease (IHD). A brief clinical examination was done.

Inclusion criteria

All patients of both genders sustaining acute ST elevation myocardial infarction were included in this study. Patients with old established conduction defects based upon their old medical record, patients with advanced heart failure, renal failure, prior coronary artery bypass surgery, patients who was died on arrival, previous LBBB, paced rhythm and Brugada syndrome where excluded.

Statistical Analysis

All data were entered to SPSS-21, frequency and percentages were calculated for categorical variables like gender, risk factors, types of myocardial infarction, and conduction defects. Descriptive statistics presented as (mean ± standard deviation). Analytic analysis was conducted to association and differences between compared variables by using t test, bivariate, and chi square test, and fisher's exact test, which was used if more than 20% expected variables were less than 5. The $p \leq 0.05$ was considered significant.

Results

One hundred patients 72 male and 28 females with STEMI were included in the final analysis. With mean age 60.06 ± 12.86 years (range 35-88), 60 patients (60%) were aged above 55 years, and the most prevalent age group was 60-69 years (30%) (Table 1).

Variable	No.	%
Age mean ± SD (60 ± 12.86 years)		
30-39	2	2
40-49	23	23
50-59	21	21
60-69	30	30
70-79	15	15
80-89	9	9
Total	100	100
Gender		
Male	72	72
Female	28	28
Total	100	100

Table 1: Demographic characteristics of STEMI patients.

Number of smokers was 51 (51%), of which (82.35%) current smoker, (17.65%) ex-smoker, Mean random blood sugar (RBS) of the patients was 202 ± 101 mg/dl, 32 (32%) of them with previous history of diabetes mellitus, Mean blood pressure of the patients was133 /83 ± 30/19 mm Hg, 37 (37%) of them were hypertensive, 16 patients (16%) with family history of IHD, 13 patients (13%) with previous history of IHD, 17 patients (17%) with past drug history of beta blockers, and 40 patients (40%) with more than one risk factor (Table 2).

Types of conduction defects	No. of patients (%) (N=100)	Permanent/Transient	% With in defects (N=22)
2nd degree block, Mobitz type 1	1 (1)	0 / 1	4.5
2nd degree block, Mobitz type 2	1 (1)	0 / 1	4.5
CHB (3rd degree AV block, narrow QRS)	5 (5)	0 / 5	22.7

CHB (wide QRS)	1 (1)	0 / 1	4.5
RBBB	4 (4)	4 / 0	18.2
LBBB	1 (1)	1 / 0	4.5
Bifascicular block	5 (5)		22.7
Total	22 (22)	12 / 100	100

Table 2: The frequency of different types of conduction defects.

Hospital stays of patient 1.95 ± 1 day, with a range of 1-6 days, most of the cases stayed 1day (38%) followed by 2 days (37%) and 3 days (22%) respectively. Mean heart rate (HR) of patients on admission was 83 ± 24 beat/minute with a range of 32-166 beat/minute.

Out of hundred patients 41 (41%) had inferior, 32 (32%) anterior, 13 (13%) anteroseptal, 11 (11%) anterolateral and 3 (3%) had lateral ST Elevation Myocardial Infarction (STEMI) (Figure 1).

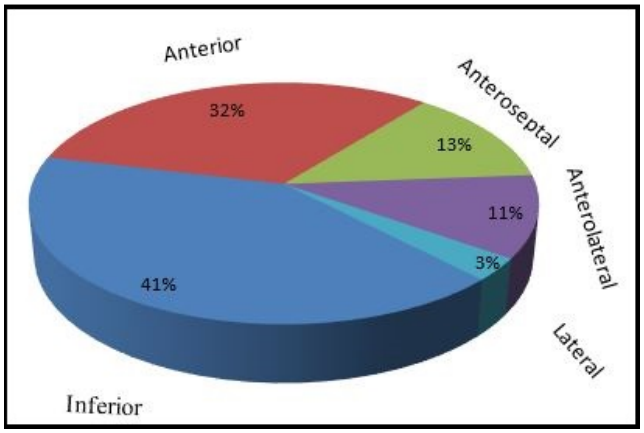

Figure 1: Types of STEMI.

Out of 100 patients, 78 (78%) of patients had no significant conduction defects, 22 (22%) of patients developed various types of conduction defects, 10 patients (45.45%) of conduction defects were transient 5 of them resolved after PCI, 1 of these 5 patient resolved spontaneously before PCI, and 5 conduction defects resolved after thrombolytic, only 12 (54.54%) of conduction defects were persistent.

Out of 22 (22%) conduction defects 11 (50%) had atrioventricular block 4 (18.18%) of them had 1st degree block 3 were present on discharge one of them was died, 1 (4.5%) had 2nd degree block Mobitz type 1 which was transient resolved after thrombolytic, 1 (4.5%) had 2nd degree block Mobitz type 2 which was transient resolved spontaneously, and 5 (22.7%) had 3rd degree AV block (narrow QRS) all were transient resolved after PCI and thrombolytic.

The other 11 (50%) of patients with conduction defects had intraventricular block, 1 (4.5%) had CHB (wide QRS) resolved after thrombolytic, 4 (18.18%) had isolated RBBB two of them were present on discharge one of them was died only one of them resolved after thrombolytic, 1 (4.5%) had LBBB which was present on discharge and 5 (22.7%) had bi-fascicular block all with (RBBB+ LAHB), three of them were present on discharge one of them was died only one of them resolved after thrombolytic (Table 2). All atrioventricular blocks had occurred with inferior STEMI while intraventricular blocks had occurred with variable types of STEMI (Table 3). The only patient with CHB (wide QRS) developed in inferior STEMI, RBBB developed in (1 anterior, 1 anteroseptal. 1 inferior and 1 anterolateral), LBBB developed in lateral STEMI, and bi-fascicular blocks developed in (1 Anterior, 1 Anteroseptal, and 3 Anterolateral) (Table 3).

Types of conduction defects	Types of STEMI					Total
	Anterior	Anteroseptal	Inferior	Anterolateral	Lateral	
1st degree block	0	0	4	0	0	4
2nd degree block, mobitz type 1	0	0	1	0	0	1
2nd degree block, mobitz type 2	0	0	1	0	0	1
CHB (3rd degree AV block, narrow QRS)	0	0	5	0	0	5
CHB (wide QRS)	0	0	1	0	0	1
RBBB	1	1	1	1	0	4
LBBB	0	0	0	0	1	1
Bifascicular block	1	1	0	3	0	5

Table 3: Conduction Defects VS Types of STEMI.

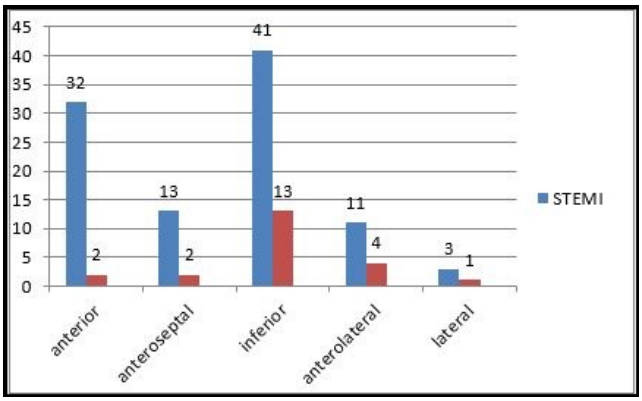

Figure 2: Types of STEMI VS conduction defects.

A significant association was observed between STEMI types and atrioventricular and intraventricular blocks (p=0.004) all atrioventricular blocks developed with inferior STEMI, while most of the intraventricular blocks developed with anterior, anteroseptal, and anterolateral STEMI (Table 4). There was no statistically significant association between duration of conduction defects and reperfusion therapy (p>0.05) (Table 5).

	Types of STEMI					p value
	Anterior	Anteroseptal	Inferior	Anterolateral	Lateral	
Atrioventricular block (N=11)	0	0	11	0	0	0.004*
Intraventricular block (N=11)	2	2	2	4	1	
*Fishers' exact test						

Table 4: Distribution of atrioventricular and intraventricular conduction blocks with STEMI types.

Variable		Group	Total (n=100)	Conduction block (n=22)	Permanent / Transient	p value
Reperfusion	PCI	Yes	46	10	5 / 5	0.09
		No	54	12	7 / 5	
	Thrombolytic	Yes	58	11	6 / 5	
		No	42	11	6 / 5	

Table 5: Distribution of duration of conduction defects in relation to reperfusion therapy.

There was significant association between STEMI types and presence or absence of conduction defects (p=0.005), 59% of conduction defects developed with inferior STEMI (Table 6). The conduction defects were higher among patients with age >55 years (p=0.04) the mean age of patients with conduction defects was 65.1year, it was higher than those without conduction defects (mean=58.6 year) with mean difference of 6.5 years (p=0.035) (Table 7). The risk of conduction defects was higher in females with OR: 1.29, CI 95%:(0.5-3.0), but there was no significant association between gender of patients and conduction defects (p>0.05). The risk of conduction defects is higher among smokers with OR: 1.5, CI 95%: (0.8-2.7), but there was no significant association between conduction defects and smoking history (p>0.05). There was no significant association between conduction defects and history of diabetes mellitus, hypertension, family history of IHD and previous history of IHD (p>0.05) (Table 6).

The risk of conduction defects is higher among those patients who had drug history of B-blockers with OR: 1.3, CI 95%:(0.4-4.1), but there was no significant association between conduction defects and drug history of beta-blockers. The risk of conduction defects is higher among those patients not treated by PCI or thrombolytic with OR: 1.3, CI 95 %:(0.4-4.0), but there was no statistically significant association between presence of conduction defects between those who treated and those who don't treated by PCI and thrombolytic probably due to that conduction defects was present before doing PCI (p>0.05).

The intra hospital death was 4 (4%) of patients 3 of them has conduction defects one with 1st degree block, one RBBB, and one with bi-fascicular block, there was significant association between conduction defects and intra hospital death (p=0.03). Five patients developed ventricular fibrillation (VF) were successfully treated by DC shock three of them had no conduction defects, two of them had conduction defects both had CHB narrow QRS.

Variable	Group	Total (n=100)	Conduction block (n=22)	p value
Age	>55	60	19	0.004
	≤ 55	40	3	
Gender	Male	72	17	0.5
	Female	28	5	
Smoking	Smokers	51	8	0.12
	Non-smokers	49	14	
Diabetes	Diabetic	32	7	0.98
	Non-diabetics	68	15	
Hypertension	h/o hypertension	37	8	0.94
	No h/o hypertension	63	14	
Family h/o IHD	Family h/o IHD	16	1	0.09
	No family h/o IHD	84	21	
h/o IHD	h/o IHD	13	4	0.4
	no h/o IHD	87	18	
h/o B-blocker	h/o B-locker	13	3	0.6
	No h/o B-blocker	87	19	
Types of STEMI	Anterior	32	2	0.005*
	Inferior	41	13	
	Anteroseptal	13	2	
	Anterolateral	11	4	
	Lateral	3	1	
PCI	Yes	46	10	0.95
	No	54	12	
Thrombolytic	Yes	58	11	0.39
	No	42	11	
Death	Intra-hospital death	4	3	0.03*
	Discharged alive	96	19	

Table 6: Distribution of Conduction defects between different variables.

There was no statistically significant difference between mean of hospital stay of patients with and without conduction defects (mean difference=0.6 day) (p>0.05) (Table 7). There was no statistically significant difference between mean of blood pressure and random blood sugar on admission of patients with and without conduction defects (p>0.05) (Table 7). The mean heart rate of patient with conduction defects on admission was 67 beats/minute, but without conduction defects was 87 beats/minute, there was statistically

significant difference between them (p<0.001) (mean difference=20) CI 95%:(9-32) (Table 7).

Variables	Conduction defects	N	Mean	Mean difference	p value	t-test	95% CI of the difference	
							Lower	Upper
Age of patients/years	Absent	78	58.6	6.5	0.035	2.13	12.5	0.45
	Present	22	65.1					
Hospital stay of patients/ days	Absent	78	1.8	0.6	0.07	2.74	1.1	0.17
	Present	22	2.4					
SBP on admission mm-hg	Absent	78	137	20	0.07	2.78	5.8	35.2
	Present	22	117					
DBP on admission by mm-hg	Absent	78	85	13	0.06	2.78	3.7	22.3
	Present	22	72					
RBS on admission mg/dl	Absent	78	196	29	0.34	0.95	89.9	31.6
	Present	22	225					
HR on admission bpm	Absent	78	87	20	<0.001	3.63	9	32
	Present	22	67					

Table 7: The collective effect of age and symptoms on ACS.

Discussion

In the present study we realized the frequency and fate of conduction defects and their influences on in-hospital outcome in cases of acute STEMI. About 83% of patients received reperfusion therapy (PCI and thrombolytic). 22% of patients developed various types of conduction defects, compared to the previous study which has been done in Tehran by A. Shirafkan et al. [24] reported a lower prevalence of conduction defects 15.8% than our study, probably due to that we took only STEMI patients but in their study only 41.5% of patients had STEMI the remainder had non STEMI, another study has been done in Pakistan by M. A. Bhalli et al. [25] found 17.6% which was also lower than our results, but another study done by K. S. Woo [26] on 636 patients of acute MI in Hong Kong, a higher rate of conduction defects 27.1% was found.

In this study 54.5% of conduction defects were present on discharge but 45.5% of patients with conduction defects were transient, resolved in hospital, most of them after reperfusion therapy, this is comparable to a study which has been done by A. Shirafkan et al. [24] who found a similar result 52.4% permanent and 47.6% transient. Out of 22 (22%) conduction defects 11 (11%) had atrioventricular block in our study, this is comparable to the study which was done by K.S. Woo [26] who found that the incidence of atrioventricular block was 11.3%, and also a similar result 11.5% was found by R. W. Brown et al. [27] A. Shirafkan et al. [24] reported a lower prevalence of atrioventricular blocks 5.5%.

In the present study the remainder 11 (11%) of patients with conduction defects had intraventricular block, was higher as compared to a study which was done in Pakistan by M. A. Bhalli et al. [25] which have reported the 9.2%, and is lower as compared to a study of K. S. Woo [26] which have reported 15.8% and is comparable to 10% of the study of Dubois et al. [2]. In this study, out of 100 STEMI patients 4% of them had 1st degree block, while a study has been done by A. Shirafkan et al. [24] reported relatively a similar result of 1st degree block 4.5%, and a lower prevalence of complete heart block (CHB) 0.75% but all of them were permanent and they didn't mentioned any transient CHB, this is opposite to our study that all of CHBs 5% was transient. Nguyen et al. [28] found that overall proportion of patients with AMI who developed CHB was 4.1% (relatively similar to our findings). M. A. Bhalli et al. [25] reported a higher prevalence of CHB 8.1% than our findings.

We found that 1% of STEMI patients had CHB (wide QRS), 4% had isolated RBBB, 1% had LBBB, and 5% had bi-fascicular block all with RBBB+LAHB. Shirafkan et al. [24] found that the prevalence of LBBB was 3% which was higher than our results and they found the prevalence of RBBB was 1.5% which was significantly lower than our results this might be due to different ethnicity. M. A. Bhalli et al. [25] found that the prevalence of RBBB was 4.8% relatively similar to our findings; LBBB was 2.5% higher than our results, and bi-fascicular block 1.4% lower than our result.

This study was supported by a previous study which was done in Italy by Rizzon et al. [29] which was studied 325 cases and they reported the prevalence of RBBB, LBBB and bi-fascicular block was 3.5%, 1.8% and 5.5% respectively which was relatively near to our results. In the present study bundle branches blocks were more common in anterior, anteroseptal, and anterolateral infarction, while blocks at the atrioventricular node occurred almost exclusively in inferior infarction, this association was statistically significant (p=0.004), the same result was reported by M. A. Bhalli et al. [25].

As per our observation, there was significant association between STEMI types and presence of conduction defects (p=0.005), overall conduction defects (13 out of 22) were more common (59%) with

inferior myocardial infarction, this is consistent with a study done by M. A. Bhalli et al. [25] which reported conduction defects (34 out of 61) were more common (55.7%) with inferior myocardial infarction or its associated combined variants, the findings of our study are also in accordance with those of Majumder et al. [30] carried out in Bangladesh, they found strong association of AV blocks with inferior MI and that of bundle branch blocks with anterior MI.

The conduction defects were higher among patients with age >55 years, this is consistent with a study by M. A. Bhalli et al. [25] which reported that bundle branch blocks were more common in patients' age more than 70 years, and also similar to a study done by J. J. Col et al. [31] who found that the mean age of patients with intraventricular conduction defects was higher statistically than that of patients without such defects.

In this study there were no significant association between conduction defects and gender of patients, smoking, presence of diabetes and hypertension, a similar result was found in the study done by A. Shirafkan et al. [24]. The present study showed the risk of conduction defects is higher among those patients not treated by PCI or thrombolytic with OR:1.3, CI 95 %:(0.4-4.0), but there was no statistically significant association between presence of conduction defects between those who were treated and those who were not treated by PCI and thrombolytic ($p>0.05$). This inversely correlated to what's found by A. Shirafkan et al. [24] in which they found the development of heart blocks was more common among those patients treated with thrombolytic therapy (21.1% vs. 12%, $p=0.01$). Our explanation is that, in current practice, thrombolytic and PCI are more frequently used for STEMI patients like our study, and less frequently used for those patients who generally suffer from less extensive infarctions like non STEMI like the study of A. Shirafkan et al. in which 58.5% of their patients had non STEMI; therefore, the association between thrombolytic therapy and heart blocks in AMI patients in their study is secondary to their more extensive lesions.

We found that The intra-hospital death was 4 (4%) of patients 3 of them has conduction defects one with 1st degree block, one RBBB, and one with bi-fascicular block, there was significant association between conduction defects and intra hospital death ($p=0.03$), this is comparable to the study of A. Shirafkan et al. [24] which was found that 36 (9%) of patients was died, 9 (25%) of those patients who died following AMI had experienced heart blocks, compared with only 63/364 (13.6%) of those who survived, the difference was statistically significant ($p<0.01$), this is higher than mortality rate in our study probably due to that (5%) of our patients had VF successfully treated by DC shock.

Conclusion

1.Conduction defects are 22% in patients with ST elevation myocardial infarction in Slemani, even with reperfusion therapy, and are associated with high mortality.

2.All atrioventricular blocks had occurred with inferior STEMI while intraventricular blocks had occurred with variable types of STEMI. All first-degree heart blocks had occurred with inferior STEMI, and bi-fascicular more in anterolateral STEMI.

3.There were no significant statistical associations between conduction defects and gender, smoking habit, being diabetic, drug history of beta blockers, treatment by PCI or thrombolytic, and presence of hypertension.

4.Older patients are more prone to develop conduction defects and its statistically significant, and this is true for patients who had slower heart rates.

Conflicts of Interest

There are no conflicts of interest for the present study.

References

1. Fleming PR (1997) A short history of cardiology (1st ed). Amsterdam-Atlanta. Rodopi.
2. Dubois C, Piarard LA, Smeets JP, Smeets JP, Legrand V, et al. (1988) Short - and long - term prognostic importance of complete bundle - branch block complicating acute myocardial infarction. Clin Cardiol 11: 292-296.
3. Klein RC, Vera Z, Mason DT (1984) Intraventricular conduction defects in acute myocardial infarction: Incidence, prognosis, and therapy. Am Heart J 108: 1007-1013.
4. Godman MJ, Lassers BW, Julian DG (1970) Complete bundle-branch block complicating acute myocardial infarction. N Engl J Med 282: 237-240.
5. Rathore SS, Gersh BJ, Berger PB, Weinfurt KP, Oetgen WJ, et al. (2001) Acute myocardial infarction complicated by heart block in the elderly: Prevalence and outcomes. Am Heart J 141: 47-54.
6. Gupta PK, Lichstein E, Chadda KD (1976) Heart block complicating acute inferior wall myocardial infarction. Chest 69: 599-604.
7. Sugiura T, Iwasaka T, Hasegawa T, Matsutani M, Takahashi N, et al. (1989) Factors associated with persistent and transient fascicular blocks in anterior wall acute myocardial infarction. Am J Cardiol 63: 784-787.
8. Goldberg RJ, Zevallos JC, Yarzebski J, Alpert JS, Gore JM, et al. (1992) Prognosis of acute myocardial infarction complicated by complete heart block (the Worcester Heart Attack Study). Am J Cardiol 69: 1135-1141.
9. Hindman MC, Wagner GS, JaRo MA, Atkins JM, Scheinman MM, et al. (1978) The clinical significance of bundle branch block complicating acute myocardial infarction. 1. Clinical characteristics, hospital mortality, and one-year follow-up. Circulation 58: 679-688.
10. Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheinman MM, et al. (1978) The clinical significance of bundle branch block complicating acute myocardial infarction. 2. Indications for temporary and permanent pacemaker insertion. Circulation 58: 689-699.
11. Scheinman MM, Gonzalez RP (1980) Fascicular block and acute myocardial infarction. JAMA 244: 2646-2649.
12. Zimetbaum PJ, Josephson ME (2003) Use of the electrocardiogram in acute myocardial infarction. N Engl J Med 348: 933-940.
13. Scheinman M, Brenman B (1972) Clinical and anatomic implications of intraventricular conduction blocks in acute myocardial infarction. Circulation 46: 753-760.
14. Feigl D, Ashkenazy J, Kishon Y (1984) Early and late atrioventricular block in acute inferior myocardial infarction. J Am Coll Cardiol 4: 35-38.
15. Meine TJ, Al-Khatib SM, Alexander JH, Granger CB, White HD, et al. (2005) Incidence, predictors, and outcomes of high-degree atrioventricular block complicating acute myocardial infarction treated with thrombolytic therapy. Am Heart J 149: 670-674.
16. Berger PB, Ruocco NA, Ryan TJ, Frederick MM, Jacobs AK, et al. (1992) Incidence and prognostic implications of heart block complicating inferior myocardial infarction treated with thrombolytic therapy: Results from TIMI II. J Am Coll Cardiol 20: 533-540.
17. Aplin M, Engstrøm T, Vejlstrup NG, Clemmensen P, Torp-Pedersen C, et al. (2003) Prognostic importance of complete atrioventricular block complicating acute myocardial infarction. Am J Cardiol 92: 853-856.
18. Goldberger AL (1991) Myocardial infarction: Electrocardiographic differential diagnosis. St. Louis: Mosby YearBook.
19. Frans JT (1987) The diagnosis of myocardial infarction in the presence of left bundle branch block. Cardiol Clin 5: 393-401.

20. Sgarbossa EB, Pinski SL, Barbagelata A, Underwood DA, Gates KB, et al. (1996) Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. N Engl J Med 334: 481-487.
21. Wellens HJ (1996) Acute myocardial infarction and left bundle-branch block—can we lift the veil?. N Engl J Med 334: 528-529.
22. Sgarbossa EB, Pinski SL, Gates KB, Wagner GS, Gusto-1 Investigators (1996) Early electrocardiographic diagnosis of acute myocardial infarction in the presence of ventricular paced rhythm. Am J Cardiol 77: 423-424.
23. Goldberger AL (2015) Electrocardiography. In: Dennis L. Kasper, Stephen L. Hauser, J. Larry Jameson, Anthony S. Fauci, Dan L. Longo, Joseph Localzo (eds.). Harrison's Principles of Internal Medicine. 19th ed. New York: Mc Graw Hill.
24. Shirafkan A, Mehrad M, Gholamrezanezhad A, Shirafkan A (2009) Conduction disturbances in acute myocardial infarction: A clinical study and brief review of the literature. Hellenic J Cardiol 50: 179-184.
25. Bhalli MA, Khan MQ, Samore NA, Mehreen S (2009) Frequency and clinical outcome in conduction defects in acute myocardial infarction. Journal of Ayub Medical College Abbottabad 21: 32-37.
26. Woo KS (1990) Conduction defects in acute myocardial infarction in the Chinese in Hong Kong. Int J Cardiol 26: 325-334.
27. Brown RW, Hunt D, Sloman JG (1969) The natural history of atrioventricular conduction defects in acute myocardial infarction. American Heart J 78: 460-466.
28. Nguyen HL, Lessard D, Spencer FA, Yarzebski J, Zevallos JC, et al. (2008) Thirty-year trends (1975-2005) in the magnitude and hospital death rates associated with complete heart block in patients with acute myocardial infarction: A population-based perspective. Am Heart J 156: 227-233.
29. Rizzon P, Di Biase M, Baissus C (1974) Intraventricular conduction defects in acute myocardial infarction. Br Heart J 36: 660-668.
30. Majumder AA, Malik A, Zafar A (1996) Conduction disturbances in acute myocardial infarction: Incidence, site-wise relationship and the influence on in-hospital prognosis. Bangladesh Med Res Counc Bull 22: 74-80.
31. Col JJ, Weinberg SL (1972) The incidence and mortality of intraventricular conduction defects in acute myocardial infarction. Am J Cardiol 29: 344-350.