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Original Article

Conduction Blocks in Acute Myocardial Infarction: A Prospective Study

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Abstract

Introduction: Coronary artery disease is a worldwide disease. Ischemic heart disease is the cause of 25-30% of deaths in most industrialized countries. Electrocardiogram is the most useful and feasible diagnostic tool for the initial evaluation, early risk stratification triage, and guidance of therapy in patients who have chest pain.

Materials and Methods: This is a prospective hospital-based study, comprising of 232 patients diagnosed with acute ST-elevation myocardial infarction (STEMI) admitted to ICCU in the Mahatma Gandhi Memorial (MGM) Hospital, Warangal, who developed conduction disturbances.

Results: This is a prospective hospital-based study, comprising of 232 patients diagnosed with acute STEMI admitted to the ICCU in MGM Hospital, Warangal, who presented from November 2012 to October 2013. Out of them, 36 (15.5%) patients were noted to have conduction blocks.

Conclusion: Developing heart block is an indirect measure of the severity and extension of the disease in patients with acute myocardial infarction.

Key words: Acute Myocardial Infarction, Blocks, Conduction

INTRODUCTION

Coronary artery disease (CAD) is a worldwide disease. Ischemic heart disease is the cause of 25-30% of deaths in most industrialized countries.

Electrocardiogram (ECG) is the most useful and feasible diagnostic tool for the initial evaluation, early risk stratification triage, and guidance of therapy in patients who have chest pain. Patients with ST elevation or new left bundle branch block (LBBB) are considered to have acute myocardial infarction (AMI) and are usually referred for immediate reperfusion therapy, whereas those without ST elevation or those with predominantly ST depression are usually treated conservatively initially.

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Bradyarrhythmias and conduction blocks (CB) are well-recognized complications of AMI. They are induced by either autonomic imbalance or ischemia and necrosis of the conduction system. It is important to recognize that bradyarrhythmias are transient and that are likely to progress to irreversible and symptomatic high-degree block. ECG reflects the physiology of the myocardium during acute ischemia. Various types of CB develop following AMI. First-degree atrioventricular block (AVB) occurs in 4-14% of patients with AMI; Mobitz Type I second-degree AVB is observed in up to 10% of patients with AMI and is usually transient. Mobitz Type II second-degree AVB occurs in >1% of patients with AMI.[1] Third-degree or complete AVB occurs in about 5-8% of patients.[2] The development of BBB, complete AVB is associated with poor prognosis likely owing to the extensive nature of the infarction.[1]

Defining the incidence and prognostic significance of new conduction abnormalities associated with AMI is complicated for several reasons. Data are most commonly generated from retrospective reviews or sub-analyses of clinical trial data. Much of the data on bradyarrhythmias and BBB predate the development of primary reperfusion

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therapies (thrombolysis and primary percutaneous coronary intervention). By reducing infarct size, these therapies may also reduce the incidence of new conduction abnormalities, although the prognostic significance of new conduction abnormalities, when they occur, may be similar.[3]

The presence or absence of BBB is usually determined on the initial ECG. Uncertainty regarding the age of BBB also impacts the interpretation of prognostic data. Chronic and new conduction abnormalities may both predict poorer outcomes but for different reasons: The former due to more extensive underlying cardiac disease and the latter due to the association with larger infarctions.

In the present day scenario, especially in our subset of patients, the risk factor profiles have changed. Furthermore, the age of onset of CAD has decreased with more number of patients having diffuse disease including multivessel disease (interheart study).

Furthermore, no study has been done in our subset of patients on CB and their prognostic implications. The present study is aimed at observing patterns of various CB and their prognostic implications in AMI.

MATERIALS AND METHODS

This is a prospective hospital-based study, comprising of 232 patients diagnosed with acute STEMI admitted to the ICCU in Mahatma Gandhi Memorial (MGM) Hospital, Warangal, who developed conduction disturbances.

Place of Study

ICCU, MGM Hospital, Warangal, Telangana, India.

Study Population

Conduction abnormalities in Patients presenting with AMI.

Period of Study

The study period is from November 2012 to October 2013.

Before the commencement of the study, permission was obtained from the department with an approval of the protocol of the study. All enrolled patients were informed about the nature of the study and their rights to refuse. Their written consent was taken before including them in the study.

A detailed history was taken about the chest pain, the presence of risk factors and duration of risk factors as appropriate. A detailed history was also obtained about the use of different medications. Random venous blood sample was obtained for analysis of blood glucose, lipid profile, renal function test, and routine blood investigations.

A diagnosis of AMI was made on the basis of chest pain lasting >30 min; ST-segment elevation ≥1 mm in at least two of the limb leads and elevation of creatine kinase enzyme and its myocardial band (MB) fraction to more than twice the upper limit of normal or troponins.

Following admission into ICCU, all the patients were followed up, and special attention was paid to detect the occurrence of CB. Continuous electrocardiographic monitoring was performed for an average of 48 h. Standard 12-lead ECG was taken on admission into ICCU, at a paper speed of 25 mm/s and an amplification of 10 mm/mV.

In an acute phase, the repeat ECG was recorded whenever the clinical condition required and every 12 h thereafter for the first 2 days and then once daily and more frequently if a change in rhythm or conduction was noted. The isoelectric line defined as the level of the preceding TP segment. The degree of ST segment elevation and depression was measured to the nearest 0.5 mm at the J-point in each of the 12 standard leads.

ECG criteria for the diagnosis of STEMI: New ST elevation at J-point in two contiguous leads with cut points: ≥0.1 mv in all leads other than leads V2-V3 where the following cut points apply: ≥0.2 mv in men ≥40 years, ≥0.25 mv in men < 40 years, ≥0.15 mv in women.

The diagnosis of various CB was made based on the following ECG features:

- First-degree AVB: PR interval of more than 0.20 s
- Second-degree AVB: Intermittent failure of AV conduction.
  - Mobitz Type I: Characterized by Wenckebach cycle, beginning with normal or prolonged PR interval and, with each successive beat, the PR interval lengthens until the block of the supraventricular impulse occurs and a beat is dropped. The pause is shorter than the PR interval of any two consecutively conducted beats. The shortest PR interval follows and the longest PR interval precedes the ventricular pause.[4,5]
  - Mobitz Type II: There is an intermittent failure of AV conduction, but the PR intervals of all the conducted supraventricular impulses are constant.
- Third-degree or complete AVB:[4] It is characterized by:
  1. AV dissociation: "P" waves bear no relationship to QRS complexes.
  2. Slow ventricular rate: Usually in the range of 30-35 bpm if subsidiary pacemaker is situated in ventricles and in the range of 35-40 bpm if subsidiary pacemaker is situated in the lower AV node (i.e., below the block) or in the bundle of His.

3. QRS configuration: If subsidiary pacemaker is situated in the lower AV node (i.e., below the block) or in the bundle of His, QRS configuration is normal or near normal and it is abnormal, being broad, notched, slurred, and bizarre if the pacemaker is situated in the ventricular musculature.

- Left anterior hemiblock (LAHB)[5-7]
  - Frontal plane mean QRS axis of −45° to −90°
  - QRS duration less than 120 m
  - qR pattern in leads I and aVL
  - Late intrinsicoid deflection in aVL (>0.45 s)
  - RS pattern in leads II, III, and aVF.
- Left posterior hemiblock (LPHB)[5-7]
  - Frontal plane mean QRS of ≥+120°
  - QRS duration of <120 ms
  - Small initial "r" wave and prominent "S" wave in leads I and aVL
  - qR pattern in leads II, III, and aVF
  - Late intrinsicoid deflection in aVF (>0.045 s)
  - Exclusion of other causes of right axis deviation.
- LBBB

Features of complete LBBB:[5]
- QRS duration ≥ 120 ms
- Broad, notched "R" waves in lateral precordial leads (V5 and V6) and usually in leads I and aVL
- Absent septal "q" waves in left-sided leads
- Small or absent initial "r" waves in right precordial leads (VI and V2) followed by deep "S" waves
- Prolonged intrinsicoid deflection (>60 ms) in V5 and V6.

Features of incomplete LBBB:[5]
- Loss of septal "q" waves
- Slurring and notching of the upstroke of "R" waves
- Modest prolongation of the QRS complex (between 100 and 120 ms)
- Right bundle branch block (RBBB).

Features of complete RBBB:[5]
- QRS duration ≥120 ms
- Broad, notched "R" waves ("rsr," "rsR," or "rSR" pattern) in right precordial leads(VI and V2)
- Wide and deep "S" waves in left precordial leads (V5 and V6).

Incomplete RBBB:[5]
- "RSr" pattern in lead V1 with a QRS duration between 100 and 120 ms.

RBBB plus LAHB:[5]
- Characterized by ECG pattern of RBBB plus left axis deviation beyond −45°.

RBBB plus LPHB:[5]
- Characterized by ECG pattern of RBBB plus a mean QRS axis deviation to the right of +120°.

Cardiac enzymes:
- CPK-MB
- Troponin I-positive/negative
- The biochemistry tests were done in MGM Hospital, Warangal.

Statistical Analysis

1. Continuous variables are presented as mean ± standard deviation and frequency variables as percentages
2. Chi-square and Fisher's exact test were performed for statistical significance. $P < 0.05$ was considered for statistical significance
3. SPSS software version 20.0 was used for statistical analysis.

Inclusion Criteria

Patients having AMI as per the WHO criteria[8] that is at least two of the following three elements be present:
1. History of ischemic type of chest discomfort
2. Evolutionary changes on serially obtained electrocardiograph tracings
3. A rise and fall in serum cardiac markers.

Exclusion Criteria

1. Patients with previous CB
2. Patients with cardiomyopathy
3. Patients with congenital or rheumatic heart disease
4. Patients with history of intake of drugs causing CB such as clonidine, methyldopa, verapamil, and digoxin.

RESULTS

This is a prospective hospital-based study, comprising of 232 patients diagnosed with acute STEMI admitted to the ICCU in MGM Hospital, Warangal, who presented from November 2012 to October 2013. Out Of them, 36 (15.5%) patients were noted to have CB.

The age of the study group ranged from 25 to 90 years (54.78 ± 13.27). The majority of the patients belonged to age group of 50-69 years (51.28%). Among male patients, majority belonged to the age group 50-59 years, compared to the females majority of whom were in the age group of 60-69 years.

Smoking was the most common risk factor in males (53.1%), followed by hypertension and diabetes. In female patients, hypertension and diabetes were the commonest risk factors (52.5%).

Smoking is the most common risk factor seen in 79 (40.3%) patients without blocks and 12 (33.3%) patients with blocks followed by hypertension in 62 (31.6%) without blocks and 10 (27.7%) patients with blocks. Diabetics had a significantly higher rate of occurrence of CB. No much difference was noted between the two groups overall.

Chest pain was the most common symptom overall and was noted in 193 (98.4%) patients without blocks and 29 (80.5%) patients with blocks. Breathlessness, palpitations, vomitings, and giddiness were more common in patients with CB compared to those without CB, and this was statistically significant.

Bradycardia, hypotension, and raised JVP are more common in patients with CB as compared to patients without CB.

Anterior or lateral wall AMI accounted for 56.8% of all cases, whereas inferior or posterior wall accounted for 43.2% of all cases. First-degree heart block was the most common of all (25%), followed next by LBBB (19.4%). BBB accounted for 69.4% of all conduction abnormalities noted. The incidence of CB was higher among patients with anterior or lateral wall AMI than the posterior or inferior wall AMI which was statistically insignificant ($P = 0.35$). 72.7% of AVB occurred in inferior wall AMI, whereas 76% of bundle branch blocks occurred in anterior wall AMI, which was statistically significant ($P = 0.04$).

Cardiogenic shock was the most common complication noted, seen in 17.2% of all cases. All cases with third-degree heart block and RBBB eventually died mean heart rate, systolic and diastolic blood pressures were lower in patients who had CB. In addition, CB occurred in slightly higher age group of patients.

About 33.3% and 10.2% of patients with and without CB, respectively, died, projecting the higher mortality associated with the development of CB. Elderly patients, diabetics, who are not thrombolysed were associated with the development of CB significantly. Similarly, CB were associated with higher risk of development of acute kidney injury and outcome death.

Advanced age, occurrence of inferior or posterior wall AMI, failure to receive thrombolytic therapy, and development of acute kidney injury during hospital stay are found as independent predictors of in-hospital mortality in my study.

DISCUSSION

232 patients of AMI were selected from cases admitted in ICCU, MGM Hospital, Warangal from November 2012 to October 2013. Out of 232 cases taken into the study 36, (15.5%) patients were noted to have CB.

Age

In the present study, the mean age of study population was 54.78 years. The subgroup with CB had a mean age of 55.55 years and the subgroup without CB had a mean age 54.34 years. The patients with CB were slightly elder than those without CB; it was statistically significant. Eriksson *et al.* reported the similar finding, as did other studies. One recent study by Shirafkan *et al.*[9] failed to show any correlation between age and incidence of CB. This could be attributed to the difference in demographic profile as well as racial, genetical disparities and also changing patterns of the population at risk.

Various registries across the world showed the mean ages of the person developing STEMI to be around 63-69 years. In another recent study from India (Create Registry[10]), the mean age was 57 years. Another similar study done by Shirafkan *et al.*[9] had a mean age group of 65.8 years.

On comparison with other studies, patients in our study were younger, corroborating with the emerging evidence from various recent studies, that CAD occurs a decade earlier in Indian population.[11] This also emphasizes the growing prevalence of CAD as well as a change of the population at risk.

Sex

In the present study group, 68.9% were males and 31.1% were females with male to female ratio of 2.2:1. Our figures are not very much different in comparison with study done by Newby *et al.* and Shirafkan *et al.*[9]

In various other studies the reported M: F ratio varied from 3.6:1 (Vakil, 1962) to 2.4:1 (Singh *et al.*, 1977).[12] The decreasing sex ratio when compared to the older studies could be due to the increasing incidence and prevalence of CAD in Indian females.

Among males, majority belonged to the age group 50-59 years, compared to the females, majority of whom were in the age group of 60-69 years. Mean age of presentation in females was more than in males (60.9 vs. 52.07 years).

Risk Factors

Smoking was the most common risk factor in males (53.1%), followed by hypertension and Diabetes. In female patients, hypertension and diabetes were the commonest risk factors (52.5%). Apart from smoking, other risk factors were not much different between male and females. In the present study, smoking was the most common risk factor in cases with and without blocks, followed by hypertension and diabetes.

Of 232 patients in the study, 132 (56.9%) had features suggestive of anterior wall AMI and 100 (43.1%) had inferior wall AMI. This was nearly similar to the previous studies reported. In a study by Shirafkan *et al.*,[9] of 400 patients, 255 (63.75%) showed electrocardiographic and echocardiographic evidence of anterior AMI, whereas 145 (36.5%) had inferior AMI.

Most of AVBs (72.7%) were seen in inferior/posterior wall AMI, whereas 76% of intraventricular blocks were associated with anterior/lateral wall AMI. These results were concurrent with the study done by Majumdar *et al.,*[13] which are 92% and 72%, respectively. In a study by Shirafkan *et al.*,[9] 79.5% of the blocks in patients with anterior AMI were of intraventricular type, whereas 68.4% of the blocks which happened after inferior AMI were AV type blocks.

The first-degree AVB was the most common as well as the most common AVB noted in the present study accounting for 25% of the total conduction defects. Third-degree AVB (complete heart block [CHB]) was seen in 5.5% which was near similar to that of study by Shirafkan *et al.*[9] (4.8%).

Another study by Nguyen *et al.*[14] found that the overall proportion of patients with AMI who develop CHB is 4.1% and emphasized that the incidence of CHB complicating AMI has declined appreciably over time, with the greatest decline in these incidence rates occurring during the most recent years (2.0% of patients hospitalized with AMI in 2005 vs. 5.1% in 1975).

LBBB was the most frequent type of intraventricular block, unlike the study by Shirafkan *et al.*[9] where LAHB was the most common. This was followed by LAHB which accounted for 16.6% of the total blocks. In a study by Shirafkan *et al.*,[9] LAHB was the most common followed by LBBB and RBBB which accounted for 30.2%, 19%, and 9.5% of the cases, respectively. Elizari *et al.*[15] also noted the presence of a frequent association between anteroseptal myocardial infarction and LAHB.

LPHFB was the least common similar to the earlier studies. As stated by Basualdo *et al.*,[16] the posterior division of the left bundle is relatively short and thick and hence is less exposed to mechanical trauma than its anterior counterpart. In addition, the posterior division of the left bundle probably receives a double blood supply from both the left anterior descending and the right coronary arteries. These anatomical considerations explain the fact that LPHB is an infrequent complication of AMI and that it is commonly associated with other conduction disturbances.

The reasons for the differences from the previous studies could be probably explained by the demographic, regional, and cultural variations between the earlier studies and the present study. Moreover, the Indian population is said to have an aggressive variety of CAD, with earliest onset, multivessel involvement, and greater myocardial damage for the extent of disease.

Symptoms and Signs

Chest pain was the most common symptom overall and was noted in 193 (98.4%) patients without blocks and 29 (80.5%) patients with blocks. Vomiting and giddiness are the next two common symptoms. Breathlessness, palpitations, vomitings, and giddiness were more common in patients with CB compared to those without CB which was statistically significant.

The probable reason for more breathlessness and other symptoms could be explained by the severity of the nature of disease. Patients with CB predominantly had anterior wall involvement (22/36, 61%). Therefore, these patients are bound to have large areas of myocardium at risk, more significant LV dysfunction. Hence, these patients were likely to have symptoms of low cardiac output such as breathlessness, palpitations, vomitings, and giddiness. Furthermore, the patients were much sicker with significant mortality and renal dysfunction.

Cardiogenic Shock

Cardiogenic shock was more common in patients with CB (38.8%) than in those without in the present study (13.2%). The incidence of cardiogenic shock was 100% in patients with CHB in the present study, which was higher on comparison with other studies.

Shirafkan *et al.*in their study showed that all patients with first-degree AVB were discharged alive, whereas 2/3 of patients who developed CHB died. Similarly, Nguyen *et al.,* in their study, showed that patients with AMI who developed third-degree heart block had greater in-hospital mortality than did those who did not develop CHB (43.2% vs. 13.0%). This shows that patients with CHB are quite sick and are bound to have significant mortality and morbidity and hence the significant amount of hypotension

Mortality

In patients with CB, the mortality rate was higher (33.3%) than in those without (10.2%). Values obtained are higher in the present study on comparison with all other studies. This could be due to the most severe nature of CAD which is usually seen in Indian patients.

In Indians, the CAD is considered to start a decade earlier, and the incidence of multivessel disease is also high. Moreover, the Indian patients are known to have much severe disease compared to lesion severity, in the form of significant myocardial damage, lower ejection fractions, higher morbidity and mortality for the same amount of disease. This could very well explain the higher mortality rates in the present study.

Furthermore, factors relating to delay in coming to medical attention, as well as a delay in treatments and lack of awareness among patients, could also have resulted in higher mortality.

CONCLUSION

1. Developing heart block is an indirect measure of the severity and extension of the disease in patients with AMI.
2. CB are associated with higher in-hospital mortality rate and are important predictors of poor outcome in patients with AMI.

LIMITATIONS

1. One of the main drawbacks of our study was the fact that there was approximately 2.5 h lag between the onset of the patients' symptoms and the recording of the baseline ECG. There is a strong possibility of developing heart block during this lag period, and such heart blocks would have been considered as "old" heart blocks and excluded from the study.
2. The other drawback of the study was the fact that due to certain limitations the patients were not followed up after discharge from the hospital.

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