ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 01 | January - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

"CLINICAL PROFILE OF BRADYARRHYTHMIA IN ACUTE MYOCARDIAL INFARCTION PATIENTS : A PROSPECTIVE OBSERVATIONAL STUDY"

General Medicine

Dr Kadamduke Neha Assistant Professor, Department Of General Medicine, GMC Miraj
Dr Somnath Magdum Associate Professor, Department Of General Medicine, GMC Miraj
Dr Anita Basavaraj Professor And Head Department Of General Medicine, GMC Miraj
Dr Ajit Hange* Assistant Professor, Department Of General Medicine, GMC Miraj *Corresponding Author

ABSTRACT

Coronary artery disease, it remains most and important cause of death all over the world. Many of these deaths are attributed to the development of arrhythmias. Arrhythmias are key events before, during or after the occurrence of acute MI. Bradyarrhythmias occur more frequently in inferior than anterior wall myocardial infarction. Sinus bradycardia is the most common bradyarrhythmia throughout acute myocardial infarction, seen frequently in first 4-6 hours of infarction. Block of early onset is generally of short duration. First degree atrioventricular block has no hemodynamic effects and needs no intervention. Treatment required only if symptoms developed. **Methods** A prospective observational study conducted at a tertiary care centre with approval from Ethics Committee for Academic Research projects, Postgraduate Academic Committee. A total of 60 patients over a period of 1 year admitted to the ICU for Acute myocardial infarction having bradyarrhythmia are included in study. **Results** Mean age group is 65 ± 14. All females are of post menopausal age group. Hypertension is the most common risk factor. It is present in (48.33%) patients. Diabetes mellitus is second most common risk factor in present study. It is present in (38.33%) patients. Smoking is present in (26.67%) patients. Total no of anterior wall MI with bradyarrhythmia is (26.67%) while no of inferior wall MI with bradyarrhythmia is (71.67%). In this study, no of cases discharged is (90%) while mortality is seen in (10%) patients. **Conclusion** It is also seen that conduction defects are most commonly associated with inferior wall myocardial infarction and interventricular defects are commonly associated with anterior wall myocardial infarction patient. Maximum bradyarrhythmia are seen in 1st hour of presentation.

KEYWORDS

Myocardial infarction, Arrhythmia, Conduction blocks, Bradyarrhythmias.

INTRODUCTION

Almost any rhythm disturbance can be associated with acute myocardial infarction, including bradyarrhythmias, supraventricular tachyarrhythmias, ventricular arrhythmias and atrioventricular block (1). Arrhythmias are key events before, during or after the occurrence of acute MI. At least 75% of patients with acute myocardial infarction (AMI) have an arrhythmia during the periinfarct period (2). Sudden cardiac death (SCD) is most often due to this pathophysiology and around one-half of death occurs before patient reach hospital. Prophylactic antiarrhythmic management strategies have largely been not used.

In acute MI, AV block transiently develops in 10-25% patient; most commonly first and second degree but complete heart block may also occur (3). A second degree and high grade AV block tends to occur more often in inferior than in anterior acute MI. In anterior infarction, however, AV block may reflect extensive septal necrosis and the prognosis is poor despite pacing (4). In anterior MI, AV block mostly located below the AVN and is usually associated with ischemia or infarction of the bundle branches.

In the first 48 to 72 hours after the onset of symptoms, most arrhythmias are observed during the pre- hospital and coronary intensive care unit phase. Reperfusion of the right coronary rhythm disturbances in patients with acute myocardial infarction may be related to coronary artery can also lead to sinus bradycardia or heart block that is due to accumulation of adenosine in nodal tissue (5). Conduction abnormalities after acute MI result from autonomic disturbances or interruption of blood supply to conduction system (6).

The mechanism for these arrhythmias is multifactorial and includes ongoing ischemia, hemodynamic abnormalities and electrolyte abnormalities (hypokalemia, hypomagnesemia) metabolic abnormalities (acidosis, hypoxia), re-entry, and enhanced automaticity. Acute myocardial ischemia leads to ATP deficiency lead to anaerobic glycolysis causing acidosis and elevation of extracellular potassium and lysophosphatidylcholine accumulation.

Bradyarrhythmias occur more frequently in inferior than anterior wall myocardial infarction. Sinus bradycardia is the most common bradyarrhythmia throughout acute myocardial infarction, seen frequently in first 4-6 hours of infarction. Block of early onset is generally of short duration. First degree atrioventricular block has no hemodynamic effects and needs no intervention. Treatment required only if symptoms developed.

Intravenous atropine usually restores AV conduction temporarily, but if escape complex is wide, temporary pacing is indicated.

Conduction system of heart

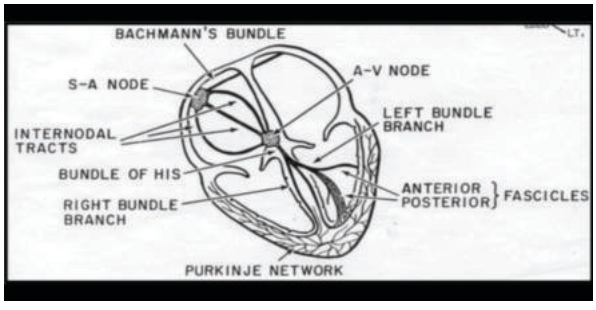

Fig no- 1

Blood supply to heart-

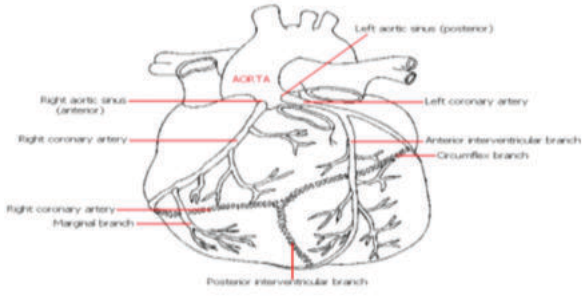

Fig no- 2

Electrophysiology Of The Heart

Myocardial cell in the resting state has membrane potential of approximately "-90 mv" called as polarized state. Influx and efflux of Na, K+ ,Ca++ ions in and out of the cells result in depolarization and repolarization thereby produce characteristic action potential curve , which has 5 phase (7) .

1 Phase 0 : Depolarization – Rapid influx of sodium
2 Phase 1 : Early rapid repolarization – Closure of sodium channels
3 Phase 2 : Plateau phase – Efflux of potassium, chloride and influx of calcium ions
4 Phase 3 : Final rapid repolarization – Efflux of potassium ions
5 Phase 4 :Resting membrane potential

Atrial and ventricular myocytes as well as purkinje fibres have the above said type of action potential called as fast channel action potential. Genesis of this type of action potential requires stimulation and can not occur spontaneously.

Bradyarrhythmias-

Suppression of automaticity of SA node or impairment of conduction in any one component of the conductive system, either due to enhanced vagal activity or ischemic damage to the conductive tissues leads to Bradyarrhythmias.

Sinus Bradycardia-

The most frequent manifestation of sinus node dysfunction is sinus bradycardia. Sinus bradycardia is defined as a rhythm with a rate of less than 60 beats/min, in which the electrocardiogram displays P waves with normal contour. If the degree of slowing is sufficient, an escape rhythm usually occurs from A-V junctional tissue, and if the junctional escape beats continue for a period of time an A-V junctional rhythm develops (8, 9). Sinus bradycardia with a subsequent escape rhythm is usually a consequence of inferior myocardial infarction.

Mechanisms for bradycardia-

Ischemia and/or infarction of the sinus node which is produced by occlusion of either the right coronary or left circumflex artery proximal to the origin of the sinus-node artery .The sinus node might be slowed indirectly as metabolites (ATP, amino acids, and other products of tissue break down) released from infarcted cells in closely adjacent regions reach the sinus node. There are large cholinergic ganglia and postganglionic fibers in the posterior basal portion of the interatrial septum. These structures, when made ischemic by right coronary artery occlusion and inferior infarction, may cause sinus bradycardia and peripheral vasodilation .The vagus nerve is considered the common pathway for mediation of reflexes. In addition, Constantine has found that coronary occlusion can produce a reflex decrease of sympathetic nerve discharge to both the heart and to peripheral vessels. Stretch receptors in the vicinity of the acutely ischemic area of myocardium were implicated in initiating this reflex. The presence of extreme pain and fear has been shown to be a factor in producing reflex sinus slowing. The influence of these reflexes could cause sustained sinus slowing and must certainly be involved in production of the bradycardia hypotension syndrome. Many drugs used for treatment of the patient with acute myocardial infarction have the potential for slowing of impulse formation. Morphine, with its vagomimetic action, the tranquilizers with their central depression, and the antiarrhythmic with their local anaesthetic effects may all exert a negative chronotropic influence on the sinus node (10).

METHODOLOGY

This study is a prospective observational study conducted at a tertiary care centre with approval from Ethics Committee for Academic Research projects, Postgraduate Academic Committee. The study is conducted in Government Medical College, Miraj hospital during period of one year. A total of 60 patients admitted to the ICU for Acute myocardial infarction having bradyarrhythmia are included in study.

- A total of 60 patients admitted to intensive care unit in Government medical college and hospital were included in study. They include 42 male and 18 female patients.
- Patient with confirmed diagnosis of acute myocardial infarction with bradyarrhythmia and satisfying the inclusion and exclusion criteria are included in the study group.
- The diagnosis of acute myocardial infarction is done as follows:-
- We used following as criteria for acute myocardial infarction-
- Symptoms of ischemia and ECG changes –
- New or presumed new significant ST segment changes or new left bundle branch block.
- Development of pathological Q waves in ECG.

Informed consent was taken of all patients admitted in ICU for acute myocardial infarction with bradyarrhythmia. A detailed history with special reference to cardiovascular system were taken. A thorough physical examination is done with emphasis on cardiovascular system. After confirmed diagnosis of acute myocardial infarcton with bradyarrhythmia, written informed consent of either patient (clinically stable) or close relative (clinically unstable) is taken.

Statistical analysis -

Chi square test and Fisher's exact test used for data analysis.

Inclusion criteria-

1 Patient in hyper acute to acute phase of myocardial infarction and developed bradyarrhythmia
2 Age more than 19 years

Exclusion criteria-

1 Patients having only acute myocardial infarction but not associated with bradyarrhythmia
2 Patient having bradyarrhythmia but not associated with myocardial infarction
3 Patient admitted with previous history of myocardial infarction
4 A known case of valvular heart disease
5 A known case of thyroid disorder
6 Patient on medications like antiarrhythmics, beta blocker.
7 Age less than 19 years.

RESULTS-

1) Age Distribution-

The age of the study group ranged from 23 to 90. Majority of the patients belonged to age group of 61 to 70.

2) Risk factors-

- Hypertension is the most common risk factor.
- It is present in 29 (48.33%) patients.
- Diabetes mellitus is second most common risk factor in present study.
- It is present in 23 (38.33%) patients.

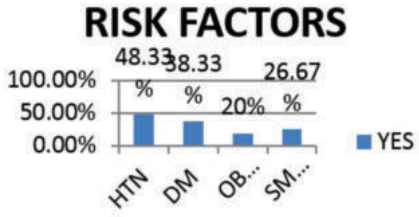

Chart No-1

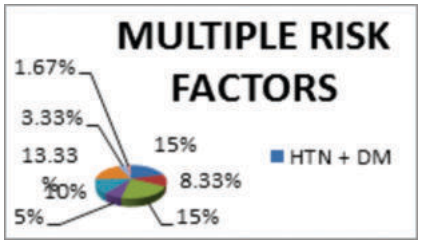

Chart No- 2

- 43 patients have more than one risk factor.
- 39 patients have 2 risk factors while 4 patients have 3 risk factors.

3) Symptoms -

Angina is the most common symptom in present in 51 (85%) patients. Vommiting, breathlessness and sweating are the other two common symptoms. Sweating is complaint of 22 (36.67%) patients. Nausea and vomiting is complaint of 17 (28.33%) patient. Dyspnoea is seen in 17 (28.33%) patient.

4) Vitals-

Bradycardia is seen in 45 patients (75%) while heart rate more than 60 is seen in 15 patients (25%). Hypertension is seen in 17(28.33%)

patients. Hypotension is seen in 22(36.67%) patients.

5) Thrombolysis-
Out of 60 patients, total 53(88.33%) patients were thrombolysed.

6) Time of appearance of bradyarrhythmia-
Table no - 1

Time of bradyarrhythmia	No of cases	Percentage
Within 1 hr	39	65%
1-12 hrs	15	25%
12 – 24 hrs	4	6.66%
> 24 hrs	2	3.33%
Total	60	100

7) Type of MI associated with Bradyarrhythmia -
Total no of anterior wall MI with bradyarrhythmia is 16 (26.67%) while no of inferior wall MI with bradyarrhythmia is 43(71.67%).

8) Type of Bradyarrhythmia-
First degree heart block (38.33%) is most common bradyarrhythmia seen in acute myocardial infarction patient followed by sinus bradycardia (26.67%). Complete heart block is seen in 7(11.67%) patient. LBBB is seen in 6 (10%) while RBBB is seen in 5(8.33%) patients. 2nd degree heart block type I seen in one (1.37%) patient and type II in 2 (3.33%) patients.

9) 2D Echo Ejection Fraction-
Out of 60 patients, 47 (78.33%) patients of myocardial infarction with bradyarrhythmia have ejection fraction >40% and 13(21.67%) patients of myocardial infarction with bradyarrhythmia have ejection fraction <40%.

10) Complications-
Cardiogenic shock is seen in 9(15%) patients having acute myocardial infarction with bradyarrhythmia. Heart failure is seen in 5(8.33%) patients having acute myocardial infarction with bradyarrhythmia.

11) Outcome of Bradyarrhythmia-

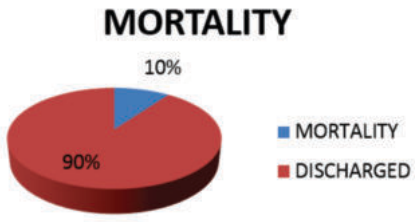

Chart no 2

Table no – 2

Persistence of bradyarrhythmia	No of Cases	Percentage of Cases
Yes	12	20%
No	48	80%
Total	60	100%

Limitations-
This study involve only hospitalised patients in Tertiary care hospital, so analysis may not reflect actual distribution of cases at population level. Sample size was small as selection was confined to patients who are admitted in this Tertiary care hospital and with only ST segment elevation acute myocardial infarction. (ACS and NSTEMI were not included.) Due to limited availability of resources, we were not able to do Troponin T , Troponin I and CK-MB of each patient in the study. So, these tests were not included in our study. Also some data variables are not available for all patients .

CONCLUSION

In this study, most common bradyarrhythmia associated with acute myocardial infarction was first degree heart block. Occurrence of bradyarrhythmia with acute myocardial infarction is seen more commonly in elderly people. In females, mostly in post menopausal age group.

It is also seen that conduction defects are most commonly associated with inferior wall myocardial infarction and interventricular defects are commonly associated with anterior wall myocardial infarction patient. Maximum bradyarrhythmia are seen in first hour of presentation.

It is also seen that patients with complete heart block have higher incidence of cardiogenic shock and hence, mortality rate is higher in patients with acute myocardial infarction having complete heart block than any other bradyarrhythmia.

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