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Conduction Blocks in Myocardial Infarcts in Tertiary Care

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ABSTRACT
ST segment elevation myocardial infarction is important cardiac disease in present days. Patients of ST segment elevation myocardial infarction can develop different complications like conduction blocks, ventricular dysfunction,cardiogenic shock, mechanical complications, ventricular arrhythmias.[1] Prognosis of ST segment elevation myocardial infarction patients developing these complications is poor.

INTRODUCTION
Cardiac conduction block is one of the important complication of ST segment elevation myocardial infarction. Cardiac conduction block is delay or interruption of the cardiac impulse. Cardiac conduction block in ST segment elevation myocardial infarction patients is because of the following physiological changes.

1. Ischemia causing temporary or permanent structural changes of the tissuessurrounding the sinoatrial node and AV junctions.
2. An increase in parasympathetic tone commonly associated with an inferior wall myocardial infarction.
3. An increase in extracellular potassium, which causes slowing of cardiac impulse conduction.
4. Local release and formation of adenosine a metabolite of adenosine triphosphate breakdown, which leads to slowing of velocity of impulseconduction through the AV node.[2]

Various types of conduction blocks develop following ST segment elevation myocardial infarction. First-degree AV block occurs in 4 to 14% of patients withST segment elevation myocardial infarction, Mobitz type I second-degree AV block is observed in around 10% of patients with ST segment elevation myocardial infarction and which is transient in nature. Mobitz type II second-degree AV block observed in <1% of patients with ST segment elevation myocardial infarction[2]. Third-degree or complete heart block occurs in about 5-8% of patients. [3] The development of complete AV block is associated with poor prognosis because of its extensive nature of the infarction.[2,3]

Bundle branch block in ST segment elevation myocardial infarction have poor prognosis. This is related both to the extent of myocardial damage [(4)] and to thefrequency of ventricular asystole.[5] Development of conduction blocks worsensthe outcome of ST segment elevation myocardial infarction. Knowing various types of conduction blocks occurring in ST segment elevation myocardialinfarction help out to recognise conduction blocks at an early stage, so that appropriate treatment including temporary or permanent pacing can be instituted at an

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early stage.

This study is undertaken to understand various patterns of conduction blocks occurring in various ST segment elevation myocardial infarction patients and itsprognostic implications at tertiary care hospital.

OBJECTIVES

- To study various patterns of conduction blocks occurring in ST elevation myocardial infarction.
- To study the prognostic implications of conduction blocks occurring in ST elevation myocardial infarction.
- To study the relation of conduction blocks with ST elevation Myocardial Infarction and implementing it to detect morbidity or mortality associated with it.

Review of Literature

Morgagni, Spens, Burnett, Adams, Mayo, Gibson, Holbertson and finally Stokes all contributed to characterization of the Adams–Stokes syndrome[8]. Mackenzie described sinoatrial block in 1902 during an epidemic of influenza[8]. Lown coined the term Sick Sinus Syndrome in 1907[8].

Moe first demonstrated dual pathways in the AV node of animals in 1956[8]. Kaufmann and Rothberger, Singer and Winterberg independently developed the concept of exit block[8].

Anatomy of Conducting System of Heart:-

Conducting system of heart is made of specialized myocardial cells and conducting fibers, capable of initiating and conducting electrical impulses. “The functioning of conducting system should be regular and rhythmic for effective Synchronization of cardiac Events, so that heart can effectively receive and pump out blood”. [8,9]

Conducting System is comprised of:-

- Sinoatrial node.
- Interatrial and Internodal Pathways.
- Anterior (Bachman), Middle, Posterior
- ➢ AV Node
- ➢ Bundle of His
- ➢ Bundle Branches
- ➢ Purkinje fibers

Figure 1: Cardiac Conduction System

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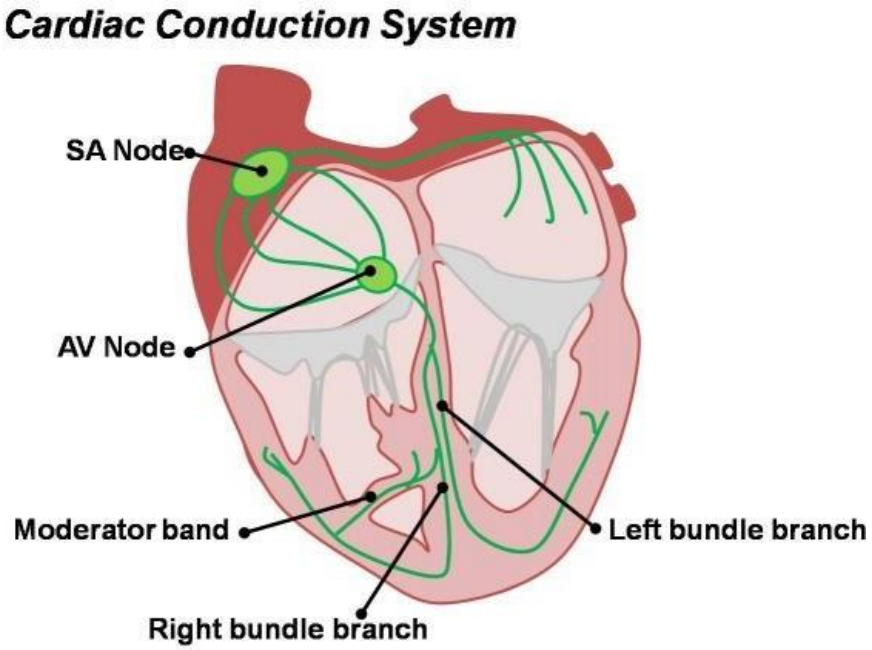

SINO ATRIAL NODE [10-16]

It consist of spindle shaped cells. It is 20 mm long 2-3 mm breadth, locatedjust 1 mm beneath the epicardial surface, at the junction of superior vena cavawith the Right atrium.

BLOOD SUPPLY: RCA in 55-60%

LCA in 40-45%

INNERVATION: - Densely innervated 3 times more than Atrial tissue by sympathetic and parasympathetic Nerves through B1 and B2, muscuranic receptors.[10]

INTERNODAL PATHWAYS:-

Anterior internodal path is the one which start in anterior margin of sinoatrial node to end in superior margin of left Atrium. Middle starts from both superior and posterior margin via intermodal septum to end in superior margin of AV Node. Posterior tract extends from posterior margin to travel posteriorly throughto interatrial septum and joins AV node. They continue as transitional fibers to end in AV node.

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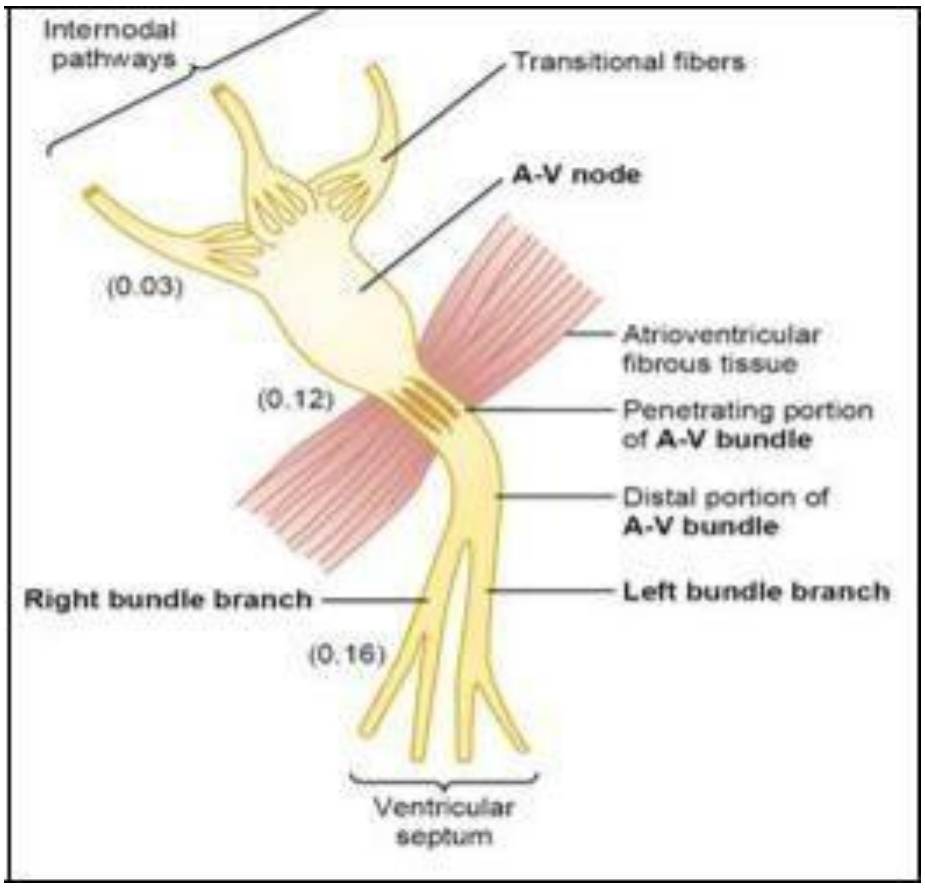
Figure 2: Cardiac Conduction System



ATRIOVENTRICULAR NODE:-

LOCATION: RA endocardium at the apex of Koch's triangle formed
ABOVE: Tendon of todaro
BELOW: Tricuspid valve septal leafletBASE: Opening of coronary sinus
BLOOD SUPPLY: Right coronary artery =85-90%Left circumflex coronary artery=10-15%
SIGNIFICANCE: The distal part of AV node is capable of automaticity. "AV node allows for travel of electrical impulse from the atria to the ventrical, giving time for emptying atrial blood in to ventrical and coordinating contractions.[11,12]

HIS BUNDLE:

LOCATION: Begins in AV node then penetrates central fibrous Body to end inmembranous septum.
BLOOD SUPPLY: Both Anterior and Posterior descending Coronary artery.
SIGNIFICANCE**:** "His bundle: Ischemia is rare unless it is widespread due to dual supply."[13]

BUNDLE BRANCH:

LOCATON: Begins in upper margin of muscular Portion of the interventriclar septum dividing into

i) Left Bundle Branch
ii) Right Bundle Branch

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Left pass through interventricular septum to divide into anterior and posteriorfascicle. Right bundle pass intramyocardially to supply Right Ventricle.

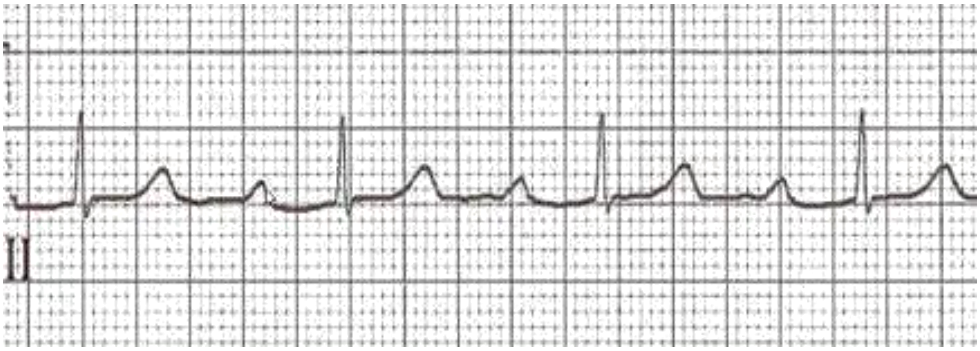
HEART BLOCK: [36-38]

"Delay or interruption in conduction of electrical impulse from atrium down to ventricle leading to dyssynchronisation of both atria and ventricular depolarization".

FIRST DEGREE HEART BLOCK:

Delay in the form of prolonged duration of PR without block in conduction.

Figure 9: First degree heart block



ETIOLOGY:

1) **Increase in vagal tone**: Either physiologic or pathologic.

i) Physiological as in athlete

ii) Pathologic as in autonomic dysfunction or drugs like Digoxin which is vagotonic.

2) **Myocardial infarction:**

i) In IWMI AV node delay is because of blood supply (i.e) both are supplied byRCA.

ii) In AWMI it is associated with BBB resulting in wide "QRS" complex when compared to IWMI which results in narrow "QRS" complex.

3) **Structural defect in AV node**

4) **Drugs like:** Non dihydropyridine ca^{2+} blockers (interferes with depolarizing current), Digoxin, Na^{2+} channel blockers causing block in Bundle of his.

5) **Dilated Cardiomyopathy (DCM)**

6) **Lyme disease**

7) **Lev disease:** It is usually asymptomatic, diagnosed using-
(i) Routine ECG (ii) EPS

Criteria :-

(i) PR duration >200ms in ECG

(ii) Prolonged AH duration>300ms.

(iii) Infranodal: HV interval>100ms.

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History of cardiac disease, drug intake, lifestyle (athlete) should be considered. Atropine causes rapid conduction in AV node and also SA node. It thereby reverts AV node delay but worsens the block in infranodal delay due to increased duration of refractory period, because of increased rate of SA nodal discharge.

Usually benign, not associated with increased mortality in Acute MI. In some conditions there is need for pacemaker as follows: [39]

i. Pacemaker syndrome: it occurs due to decreased time available for atrium to fill as it immediately follows ventricular contraction, leading to "awareness of one own heart beat".[40]
ii. In neuromuscular disease, as it may progress to high degree.
iii. Wide QRS complex (HV>100ms) in infranodal delay.
iv. First degree heart block with AV dissociation.

II DEGREE AV BLOCK:- [41,42]

This is due to intermittent block of electric impulse through AV node, which maybe of

I. Regular pattern: The block is variable, may be of 2:1, 3:1 due to one non-conducted „P" at a time.
II. Mobitz type I: PR duration is prolonged in progressive way ultimately resulting in a drop of beat then conducting normally followed by similar cycle.
III. Mobitz type II: PR interval is not changed, before and after a non-conducted "P" wave.
IV. High grade: in which more than one „P" wave are not conducted

Figure 10: Mobitz type I or Wenckebach

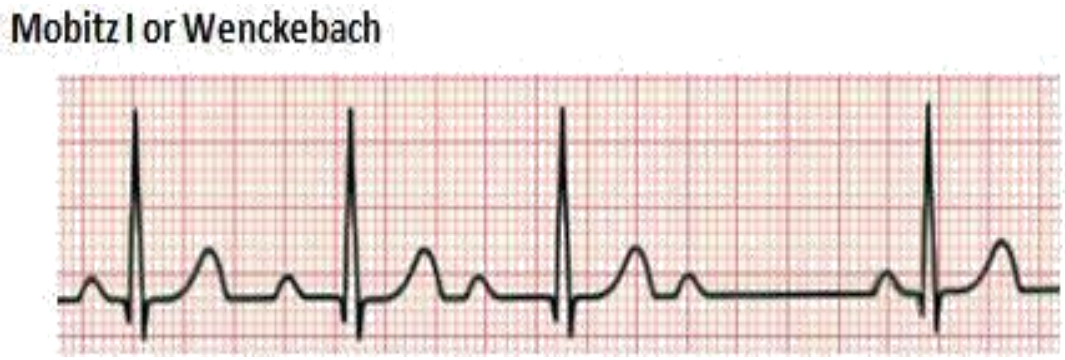
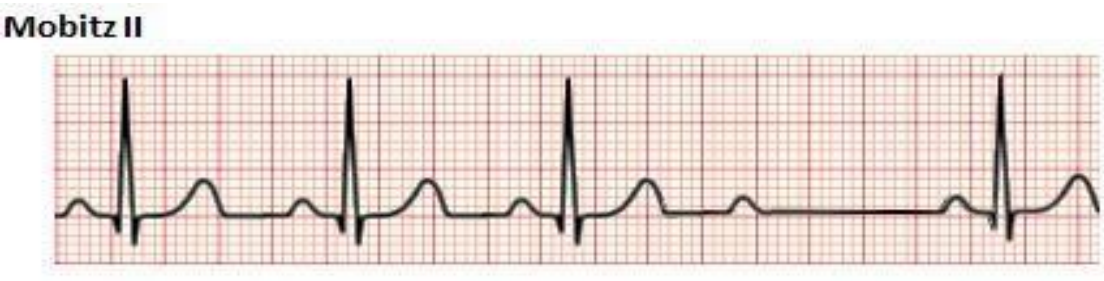


Figure 11: Mobitz Type II



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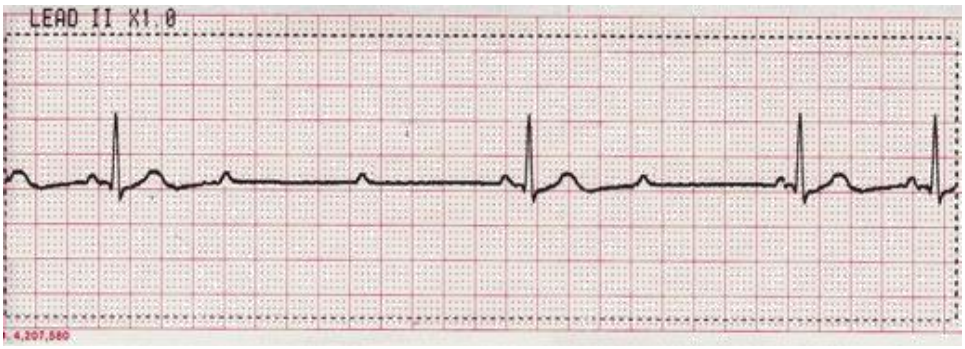
High grade:



CAUSE OF II DEGREE BLOCK :[43]

I. Physiologic
II. Increased vagal tone
III. Myocardial infarction
IV. Myocarditis
V. Endocarditis
VI. Hyperkalemia

Type 1 is usually benign, may cause irregular pulse, syncope and in acute MI it may lead to decreased cardiac output. It responds to Atropine and is worsened by carotid sinus massage. Diagnosed by EPS, which is indicated in syncope, the findings are progressive A-H and constant H-V, finally no H-V electrogram,

THERAPY:

(i) Atropine (in hemodynamically unstable)
(ii) Temporary pacemaker (in Acute MI)
(iii) Permanent pacemaker (in hemodynamically stable).

Materials and Methods

Type of study: This was prospective, observational cohort study done in patients admitted in wards and ICU who diagnosed of having ST segment elevation myocardial infarction and developed conduction blocks.

Total Patients enrolled: A total of seventy patients admitted in wards and ICUwere enrolled in the present study.

Duration of study: This study was conducted over period of 18 months. (1st December 2017 to 31st May 2019)

Study setting: This study was carried out in patients admitted in wards and ICU who fulfils the W.H.O. criteria OF ST segment elevation myocardial infarction at Krishna Hospital and Medical Research Centre, Karad.

Inclusion criteria

1. Patients diagnosed with ST segment elevation myocardial infarction (STEMI) as per W.H.O criteria that is at least two of the following three elements be present:

- Typical history of chest pain presenting for > 30 min
- Classical ECG changes indicating ACUTE MI.

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- Elevated cardiac enzymes levels CPK MB and troponin I

Exclusion criteria

- Patients with old bundle branch block.
- Patients with cardiomyopathy.
- Patients with congenital or Rheumatic heart disease.
- Patients with history of intake of drugs causing conduction blocks like, clonidine, methyl dopa, verapamil, digoxin etc. All the patients included in the study was explained about the procedure in detail and issued Patient Information Sheet. Informed and written consent was taken in each case.

All the investigations and interventions (if necessary) was done under the direct supervision and guidance of our guide.

This study was approved by Institutional Ethics and protocol committee. Informed and written consent from patients were taken before enrolling in 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 cardiac enzymes, blood glucose, lipid profile, renal function test, and routine blood investigations.

A diagnosis of STEMI 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 ICU, all the patients were followed up, and special attention was paid to detect the occurrence of conduction block. Continuous electrocardiographic monitoring was performed for an average of 48 hr . Standard 12-lead ECG was taken on admission in to ICU, at a paper speed of 25 mm/s and an amplification of 10 mm/mV.
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
Diagnosis of various conduction block was made based on characteristic ECG changes as follow:

- First-degree AV block.
- Second-degree AV block: Intermittent failure of AV conduction.
- Mobitz Type I.
- Mobitz Type II.
- Third-degree or complete AV block.
- Left anterior Hemi block (LAHB).
- Left posterior Hemi block (LPHB).
- LBBB.
- RBBB.

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Other investigations:

- CPK-MB by CK-MB ELISA kit on EM360 analyser
- Troponin I by Eurolyser troponin I smart kit on EM360 analyser
- Serum Lipid level
- 2 D-ECHO(WIPRO GE-95, Reg.no.: MH/STR/0376)
- ECG (12 lead ECG machine serial number:DUTB5C3153,ID number: KIMSDU/KH/W.NO.3/ECG-1)

STATISTICAL ANALYSIS

The statistical analyses performed using the Statistical Package for Social Science (SPSS) version21 for Windows. Data were expressed as mean values ± standard deviations (SD) for continuous variables. Frequency and proportions were reported for categorical variables. The p-value of < 0.05 was considered statistically significant.

Observations and Results

Frequency distribution of ST segment elevation myocardial infarction patients according to gender:

In the present study total seventy (n=70) ST segment elevation myocardial infarction patients were enrolled. Out of the seventy ST segment elevation myocardial infarction patients forty nine (70%) patients were male and twenty one (30%) patients were female. Prevalence of ST segment elevation myocardial infarction was significantly (p=0.001) more in males as compared tofemales.

Frequency distribution of ST segment elevation MI patients according togender is depicted in table no.1.

Table 1: Frequency distribution of ST segment elevation myocardial infarction patients according to gender

Gender	Number (n=70)	Percentage
Male	49	70
Female	21	30
Total	70	100
(„*p*' value =0.001)		

Table 3: Distribution of ST segment elevation myocardial infarction patients according to symptoms

Symptoms	Sex		Total	Percentage
	Male	Female		
Chest pain	49	20	69	98.57
Vomiting	20	6	26	37.14
Sweating	48	19	67	95.71
Dyspnea	25	10	35	50.00
Palpitation	11	8	19	27.14

Distribution of conduction blocks among various sites of ST segment elevation myocardial infarction:

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In the present study distribution of conduction blocks among various sites of ST segment elevation myocardial infarction was studied. Most common site of ST segment elevation myocardial infarction was anterior wall myocardial infarction (n=27). Out of these twenty seven patients, nine (n=9) patients were having first degree heart block, seven (n=7) patients were having Mobitz type 2 AV heart block, six (n=6) patients were having right bundle branch block, three (n=3) patients were having complete heart block, one patient having left bundle branch block another one patient having Mobitz type 1 AV heart block.

Number of ST segment elevation myocardial infarction patients having inferior wall myocardial infarction was twenty four (n=24). Out of these twenty four patients nine (n=9) patients were having first degree heart block, five (n=5) patients were having Mobitz type 1 AV heart block, four (n=4) patients were having complete heart block, two (n=2) patients were having left bundle branch block another two (n=2) patients were having Mobitz type 2 heart block.

Number of ST segment elevation myocardial infarction patients having lateral wall myocardial infarction was eight (n=8). Out of these eight patients three (n=3) patients were having left bundle branch block, two (n=2) patients were having first degree heart block and one patient of each complete heart block, right bundle branch block , left anterior hemi block. Number of ST segment elevation myocardial infarction patients having anterolateral wall myocardial infarction were five (n=5). Out of those five patients three (n=3) were having Mobitz type 2 heart block and one patient of each having left bundle branch block & right bundle branch block.

Number of ST segment elevation myocardial infarction patients having anteroseptal wall myocardial infarction were three (n=3). Out of those three there was one patient of each complete heart block, Mobitz type1 AV and Mobitz type 2 AV heart block.

Number of ST segment elevation myocardial infarction patients having inferoposterior wall myocardial infarction was two (n=2). Out of those two patients there was one patient of complete heart block and one patient of Mobitz type 1 AV heart block.

Number of ST segment elevation myocardial infarction patient having anteroinferior wall myocardial infarction was one (n=1) who had complete heartblock.

Table 8: Distribution of conduction blocks among various sites of ST segment elevation myocardial infarction

	Total no. of conduc	**Types of conduction block**

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Site		CHB	FIRST DEGREE HB	LAHB	LBBB	MOBITZ TYPE 1	MOBITZ TYPE 2	RBBB	TRIFASCICULAR BLOCK
Anterior Wall MI	27	3	9	-	1	1	7	6	-
Anteroinferior wall MI	1	1	-	-	-	-	-	-	-
Anterolateral wall MI	5	-	-	-	1	-	3	1	-
Anteroseptal wall MI	3	1	-	-	-	1	1	-	-
Inferoposterior wall MI	2	1	-	-	-	1	-	-	-
Inferior wall MI	24	4	9	-	2	5	2	0	1
Lateral wall MI	8	1	2	1	3	-	1	1	-

DISCUSSION

CLINICAL PRESENTATION

Different symptoms observed among the ST segment elevation myocardial infarction patients in the current study was chest pain (98.57%), sweating (95.71%), dyspnea (50%), vomiting (37.14%) and palpitation (27.14%). The most common symptoms was chest pain followed by sweating.

In the study done by **Chandrashekar and Path et al**[61], in their study observed that, 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 was the next two common symptoms. Breathlessness, palpitations, vomiting, and giddiness were more common in patients with CB compared to those without CB which was statistically significant.

RISK FACTORS

In the present study hypertension was the most common risk factor being present in twenty seven (38.57%) of ST segment elevation myocardial infarction patients. In the hypertensive patients male (30%) were more than female (8%). Second risk factor was Diabetes mellitus being present in 27.14%(n=19) of ST segment elevation myocardial infarction patients. Third risk factor was smoking present in 21.43% (n=15) of ST segment elevation myocardial infarction patients. All of the smoking patients were male. Only one (n=1) of theST segment elevation myocardial infarction patient was having cerebrovascular accident as a risk factor which was present in female patient.

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In the study by **Ratan Ram et al[56],** Various risk factors such as hypertension were present in 27% of cases, diabetes in 25% of cases, IHD in 13% of cases,and smoking in 30% of cases. A similar finding was observed in the study by **Chavda et al[57]**.where smoking (72.0%) was the most common risk factor followed by IHD in 14% of cases and 10% had DM. The prevalence of hypertension and diabetes mellitus in the study by **Hreybe and Sab[62]** was 22.3% and 20.2%, respectively. On comparing between males and females, hypertension, IHD, and smoking were more among males, but diabetes was more among females.

Summary and Conclusions

1. In the present study prevalence of ST segment elevation myocardial infarction was significantly (P=0.001) more in males as compared to females. Age of patients ranged from 32 to 110 years with mean age of 60.69 (± 13.41) years.
2. Different symptoms observed among the ST segment elevation myocardial infarction patients in the current study was chest pain (98.57%), sweating (95.71%), dyspnea (50%), vomiting (37.14%) and palpitation (27.14%). The most common symptoms was chest painfollowed by sweating.
3. In the present study hypertension was the most common risk factor being present in twenty seven (38.57%) of ST segment elevation myocardial infarction patients. In the hypertensive patients male (30%) were more than female (8%). Second risk factor was Diabetes mellitus being present in 27.14% (n=19) of ST segment elevation myocardial infarction patients. Third risk factor was smoking present in 21.43% (n=15) of ST segment elevation myocardial infarction patients. All of the smoking patients were male. Only one (n=1) of the ST segment elevation myocardial infarction patient was having cerebrovascular accident as a risk factor which was present in female patient.
4. In the present study among the seventy (n=70) ST segment elevation myocardial infarction patients studied we found different sites of myocardial infarction. Anterior wall myocardial infarction present in 38.57% (n=27) patients, Inferior wall myocardial infarction present in 34.29% (n=24) patients, Lateral wall myocardial infarction present in 11.43% (n=8) patients, Anterolateral wall myocardial infarction present in 7.14% (n=5) patients, Anteroseptal wall myocardial infarction present in 4.28% (n=3) patients, Inferior Posterior wall myocardial infarction present in 2.86%(n=2) patients, anteroinferior wall myocardial infarctionin 1.43%(n=1) patients. The most prevalent sites was anterior wallmyocardial infarction followed by Inferior wall myocardial infarction.
5. In the seventy (n=70) ST segment elevation myocardial infarction patients studied there were eight different types of conduction blocks observed. First Degree heart block present in 28.57% (n=20) patients, Mobitz type 2 AV block present in 20% (n=14) patients, complete heart block present in 17.14% (n=12) patients , Mobitz type 1 AV block present in 11.43% (n=8) patients, Right bundle branch block present in 10% (n=7) patients, Left bundle branch block present in 10% (n=7) patients, Left anterior hemi block present in 1.43% (n=1) patients, andTrifascicular block present in 1.43% (n=1) patients. The most prevalent conduction block was first degree heart block followed by Mobitz type 2AV heart block and complete heart block.
6. In the present study distribution of conduction block was studied according to gender. We found no significant difference in the numbers of each conduction block compared to gender. Though there was no significant difference, the higher number of

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patients among allconduction blocks were of male gender.

7. In the present study mortality among the ST segment elevation myocardial infarction patients was studied according to type of conduction block. The mortality was only observed in patients with complete heart block (n=8) and first degree heart block (n=2). While in all other patients with other conduction blocks improvement was noted. The rate of mortality of patients with complete heart block when compared with the mortality rate of patients with first degree heart blocks, there was significantly (p=0.0031) higher mortality rate observedin patients with complete heart block than first degree heart block.
8. In the present study, we compared the mortality according to Kilipclassification. We observed that there was 100% mortality among Kilip class 3 anOur study concludes that the prevalence of ST segment elevation myocardial infarction was significantly more in male. The most common symptom in ST segment elevation myocardial infarction patients was chest pain followed by sweating. In ST segment elevation myocardial infarction patient‟s the most common site of myocardial infarction was anterior wall myocardial infarction followed by inferior wall myocardial infarction. In the present study most prevalent conduction block was first degree heart block followed by Mobitz type 2 AV block. High prevalence of mortality was seen in the patients with complete heart block.Thus severity of conduction block is predictor of poor outcome in the ST segment elevation myocardial infarction patients. All patients with ST segment elevation myocardial infarction should be monitored for early recognition of conduction block and appropriate treatment should be started to improve the outcome of patient.

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