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A COMPARATIVE STUDY ON PREVALENCE, CLINICAL SCENARIO, TREATMENT PATTERNS AND QUALITY OF LIFE IN STEMI AND NSTEMI PATIENTS

Oruganti Sai Vinod Kumar*[1], Gowri Ganesh[2], Dr. C. Venkatesh[3], Dr. Raghavendra Cheruku[4], Dr. N. M. Vageesh[5]

[1*,2]Department of Pharmacy Practice, St. Johns College of Pharmaceutical Sciences, Yerrakota, Kurnool District, Andhra Pradesh.

[3]Assistant Professor, Department of Pharmacy Practice, St. Johns College of Pharmaceutical Sciences, Yerrakota, Kurnool district, Andhra Pradesh.

[4]Department of Cardiology, Chief Consultant Interventional Cardiologist, HOD & Director of Cath Lab at Gem Care Kamineni Hospital, Kurnool, Andhra Pradesh.

[5]Department of Pharmaceutics, St. Johns College of Pharmaceutical Sciences, Yerrakota, Kurnool district, Andhra Pradesh.

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***Corresponding Author**
Oruganti Sai Vinod Kumar
Department of Pharmacy Practice, St. Johns College of Pharmaceutical Sciences, Yerrakota, Kurnool District, Andhra Pradesh.

ABSTRACT

Aim: The aim of this study to compare the prevalence, clinical scenario, treatment patterns and quality of life in STEMI and NSTEMI patients. **Objectives:** To determine the prevalence of STEMI and NSTEMI in the target population over a defined period. To describe the clinical scenario & treatment patterns of STEMI & NSTEMI patients. To assess the quality of life in STEMI & NSTEMI patients using validated WHOQOL-BREF tools. **Methodology:** A Prospective observational study was conducted over a period of 6 months in department of cardiology in Gemcare kamineni hospital, Kurnool. Patients were enrolled based on inclusion and exclusion criteria. The patient data collected with the standard proforma. The patient's data collected was categorized into two groups according to the study. **Results:** STEMI was more prevalent than NSTEMI, especially in younger males, while NSTEMI was common in older patients with comorbidities. STEMI patients presented with more typical and severe symptoms, leading to quicker hospital arrival and more immediate interventions like PCI. NSTEMI patients often

had delayed presentation and were managed more conservatively at first. Both groups received similar medications, but STEMI patients showed better quality of life at follow-up due to faster treatment and fewer underlying conditions. **Conclusion:** STEMI and NSTEMI are both serious acute coronary syndromes but differ significantly in patient demographics, treatment approaches, and post-recovery outcomes. STEMI patients, typically better quality of life. In contrast, NSTEMI patients, often older with more comorbidities, require a more cautious treatment approach and tend to report lower physical and emotional health outcomes post-discharge.

KEYWORDS: To describe the clinical scenario & treatment patterns of STEMI & NSTEMI patients.

INTRODUCTION

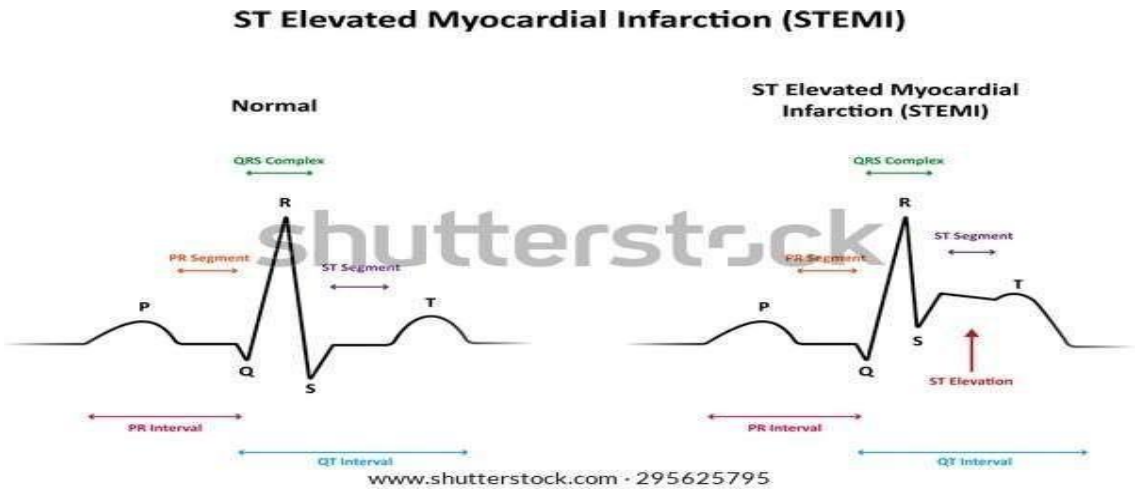
Definition: Myocardial infarction (MI), commonly known as a heart attack, is defined pathologically as the irreversible death of myocardial cells caused by ischemia. Clinically, MI is a syndrome that can be recognized by a set of symptoms, chest pain being the hallmark of these symptoms in most cases, supported by biochemical laboratory changes, electrocardiographic (ECG) changes, or findings on imaging modalities able to detect myocardial injury and necrosis. (Placeholder5)

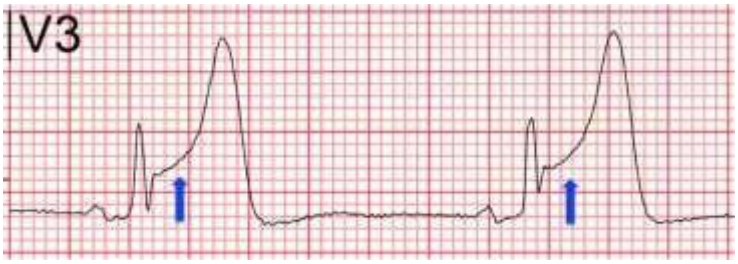
Types of MI

MI is divided into two main categories, non–ST elevation MI (NSTEMI) and ST-elevation MI (STEMI)

STEMI (ST-Elevation Myocardial Infarction)

A complete blockage of a coronary artery generally characterizes STEMI. It is leading to significant damage to the heart muscle. This blockage is typically evidenced by persistent ST-segment elevation on an electrocardiogram (ECG). STEMI is generally considered a medical emergency. Also, it requires immediate reperfusion healing. This is thrombolytic therapy or percutaneous coronary intervention (PCI). It restores blood flow to the affected area of the heart.





The STEMI is further divided into

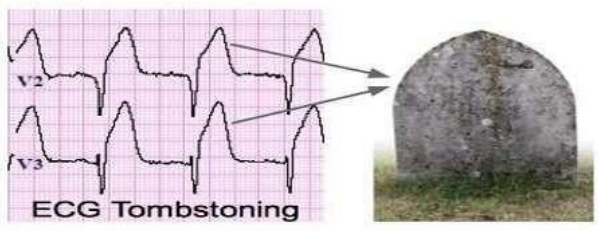
TYPE 1: Anterior Wall Myocardial Infarction

- It is also known as anterior wall MI, or AWMI, or anterior ST segment elevation MI, or anterior STEMI.
- It occurs when anterior myocardial tissue usually supplied by the left anterior descending coronary artery suffers injury due to lack of blood supply. When an AWMI extends to the septal and lateral regions as well, the culprit lesion is usually more proximal in the LAD or even in the left main coronary artery.
- This large anterior myocardial infarction is termed an extensive anterior.

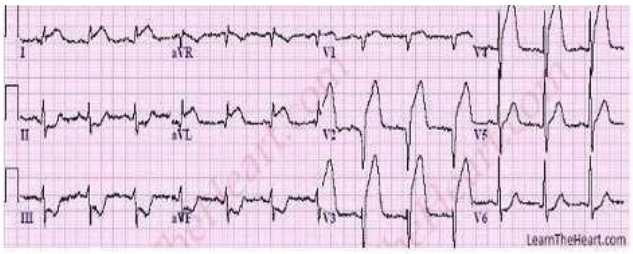
The ECG findings of an acute anterior myocardial infarction wall include

- ST segment elevation in the anterior leads (V3 and V4) at the J point and sometimes in the septal or lateral leads, depending on the extent of the MI. This ST segment elevation is concave downward and frequently overwhelms the T wave. This is called “tombstoning” and the shape is similar to that of a tombstone.

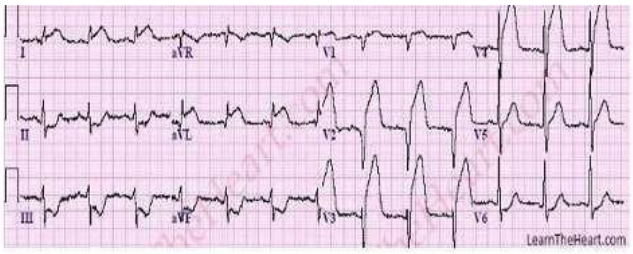
Patterns of revascularization and its impact in Patients



- Reciprocal ST Segment depression in the inferior leads (II, III and aVF).
- According to the American College of Cardiology/American Heart Association guidelines for STEMI, there must be ―new ST segment elevation at the J point in at least two contiguous leads of ≥ 2 mm (0.2 mV) in men or 1.5 mm (0.15 mV) in women in leads V2-V3 and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads.‖ This means 1 millimeter in any two contiguous leads, except leads V2 or V3, where the elevation must be 2 mm in men or 1.5 mm in women.



- The ECG findings of an old anterior wall MI include the loss of anterior forces, leaving Q waves in leads V1 and V2. This is a cause of poor R wave progression, or PRWP.



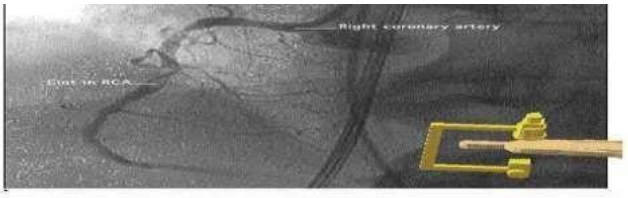
Type 2: Inferior St Segment Elevation Myocardial Infarction

It is also known as IWMI, or inferior MI, or inferior ST segment elevation MI, or inferior STEMI

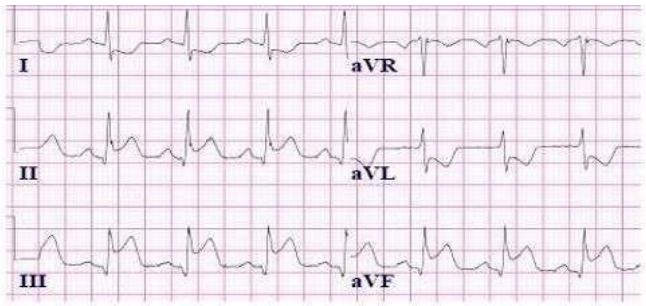
- It occurs when inferior myocardial tissue supplied by the right coronary artery, or RCA, is injured due to thrombosis of that vessel.
- When an inferior MI extends to posterior regions as well, an associated posterior wall MI may occur.

The ECG findings of an acute inferior myocardial infarction include the following

- ST segment elevation in the inferior leads (II, III and aVF).
- Reciprocal ST Segment depression in the lateral and/or high lateral leads (I, aVL, V5 and V6).



Inferior STEMI

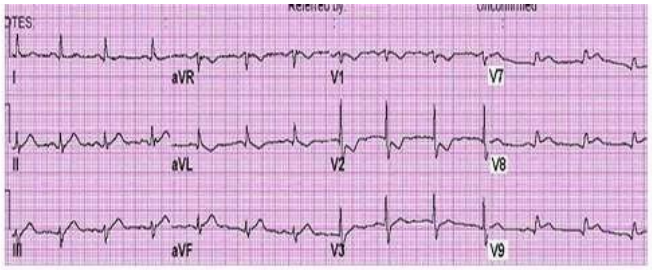


Type 3: Posterior St Segment Elevation Myocardial Infarction

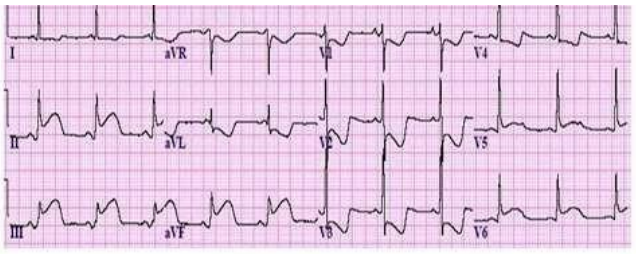
The ECG findings of a posterior wall myocardial infarction are different than the typical ST segment elevation seen in other myocardial infarctions. A posterior wall MI occurs when posterior myocardial tissue (now termed inferobasilar), usually supplied by the posterior descending artery — a branch of the right coronary artery in 80% of individuals — acutely loses blood supply due to intracoronary thrombosis in that vessel. This frequently coincides with an inferior wall MI due to the shared blood supply.

The ECG findings of an acute posterior wall MI include the following

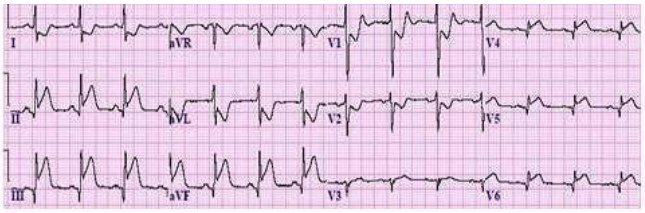
- ST segment depression (not elevation) in the septal and anterior precordial leads (V1-V4). This occurs because these ECG leads will see the MI backwards; the leads are placed anteriorly, but the myocardial injury is posterior.
- An R/S wave ratio greater than 1 in leads V1 or V2.
- ST elevation in the posterior leads of a posterior ECG (leads V7-V9). Suspicion for a posterior MI must remain high, especially if inferior ST segment elevation is also present.
- ST segment elevation in the inferior leads (II, III and aVF) if an inferior MI is also presents.



Type .4. Inferior-posterior STEMI example 1

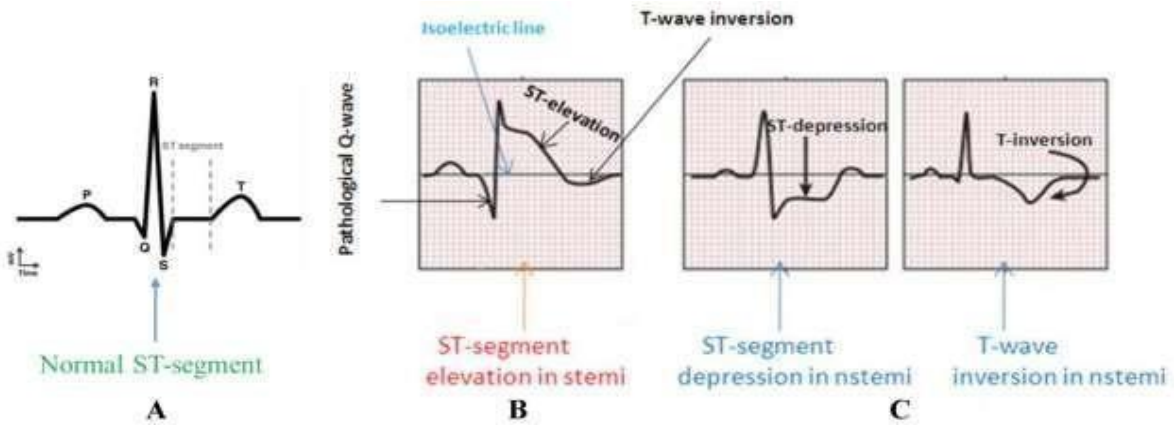


Inferior-posterior STEMI example 2



NSTEMI (Non-ST-Elevation Myocardial Infarction)

This condition occurs when there is a partial blockage or temporary disruption of blood flow in a coronary artery. It results in ischemia and myocardial damage. Difference between STEMI and NSTEMI may not present with significant ST-segment elevation on the ECG. Diagnosis often involves detecting cardiac biomarkers, such as troponin, which indicate myocardial injury.



Treatment of NSTEMI may include medications. They help to ease signs, stabilize the patient, and prevent further complications. Cardiac catheterization and revascularization, depending on the severity of the condition, are helpful.

Etiology

As stated above, myocardial infarction is closely associated with coronary artery disease. INTERHEART is an international multi-center case-control study which delineated the following modifiable risk factors for coronary artery disease.

1. Smoking
2. Abnormal lipid profile/blood apolipoprotein (raised ApoB/ApoA1)
3. Hypertension
4. Diabetes mellitus
5. Abdominal obesity (waist/hip ratio) (greater than 0.90 for males and greater than 0.85 for females)
6. Psychosocial factors such as depression, loss of the locus of control, global stress, financial stress, and life events including marital separation, job loss, and family conflicts
7. Lack of daily consumption of fruits or vegetables
8. Lack of physical activity
9. Alcohol consumption (weaker association, protective)

The INTERHEART study showed that all the above risk factors were significantly associated with acute myocardial infarction except for alcohol consumption, which showed a weaker association. Smoking and abnormal apolipoprotein ratio showed the strongest association with acute myocardial infarction. The increased risk associated with diabetes and hypertension was found to be higher in women, and the protective effect of exercise and alcohol was also found to be higher in women.

Other risk factors include a moderately high level of plasma homocysteine, which is an independent risk factor of MI. Elevated plasma homocysteine is potentially modifiable and can be treated with folic acid, vitamin B6, and vitamin B12.

Some non-modifiable risk factors for myocardial infarction include advanced age, male gender (males tend to have myocardial infarction earlier in life), genetics (there is an increased risk of MI if a first-degree relative has a history of cardiovascular events before the age of 50). The role of genetic loci that increase the risk for MI is under active investigation.

Epidemiology

Prevalence

More than 3 million individuals develop STEMI each year, while over 4 million experience NSTEMI.

STEMI tends to be more common in younger patients, whereas NSTEMI is more frequently seen in older individuals with multiple comorbidities.

Geographical Trends

While myocardial infarction (MI) is predominantly detected in developed countries, it is also increasingly common in developing nations.

In recent years, a decreasing trend in STEMI incidence has been observed in European countries and the United States.

Mortality & Outcomes

The mortality risk within and beyond 6 months after MI is not significantly different between STEMI and NSTEMI patients after adjusting for confounders However, deaths due to post-resuscitation status and heart failure are more frequent in NSTEMI within the first 6 months after MI

Diagnosis

- Regular physical examination.
- **Electrocardiogram:** This first test done to diagnose a heart attack records the electrical activity of your heart via electrodes attached to your skin.
- Because injured heart muscle doesn't conduct electrical impulses normally, the ECG may show that a heart attack has occurred or is in progress.
- **Blood tests:** Certain heart proteins slowly leak into your blood after heart damage from a heart attack. Emergency room doctors will take samples of your blood to test for the presence of these enzymes.

ADDITIONAL TESTS

- **Chest x-ray.** An x-ray image of your chest allows your doctor to check the size of your heart and its blood vessels and to look for fluid in your lungs.
- **Echocardiogram.** An echocardiogram can help identify whether an area of your heart has

been damaged and isn't pumping normally.

- **Coronary catheterization (angiogram).** A liquid dye is injected into the arteries of your heart through a long, thin tube (catheter) that's fed through an artery, usually in your leg or groin, to the arteries in your heart. The dye makes the arteries visible on x-ray, revealing areas of blockage.
- **Exercise stress test.** In the days or weeks after your heart attack, you might also have a stress test to measure how your heart and blood vessels respond to exertion.
- You might walk on a treadmill or pedal a stationary bike while attached to an ECG machine. Or you might receive a drug intravenously that stimulates your heart similar to the way exercise does.
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- **Cardiac CT or MRI.** An x-ray tube inside the machine rotates around your body and collects images of your heart and chest. In a cardiac MRI, you lie on a table inside a long tube-like machine that produces a magnetic field. The magnetic field aligns atomic particles in some of your cells. When radio waves are broadcast toward these aligned particles, they produce signals that vary according to the type of tissue they are. The signals create images of your heart.
- **Metabolic syndrome.** This occurs when you have obesity, high blood pressure and high blood sugar. Having metabolic syndrome makes you twice as likely to develop heart disease as if you don't have it.
- **Family history of heart attack.** If your siblings, parents or grandparents have had early heart attacks (by age 55 for male relatives and by age 65 for female relatives), you might be at increased risk.
- **Lack of physical activity.** Being inactive contributes to high blood cholesterol levels and obesity. People who exercise regularly have better cardiovascular fitness, including lower high blood pressure.
- **Stress.** You might respond to stress in ways that can increase your risk of a heart attack.
- **Illicit drug use.** Using stimulant drugs, such as cocaine or amphetamines, can trigger a spasm of your coronary arteries that can cause a heart attack.

Complications

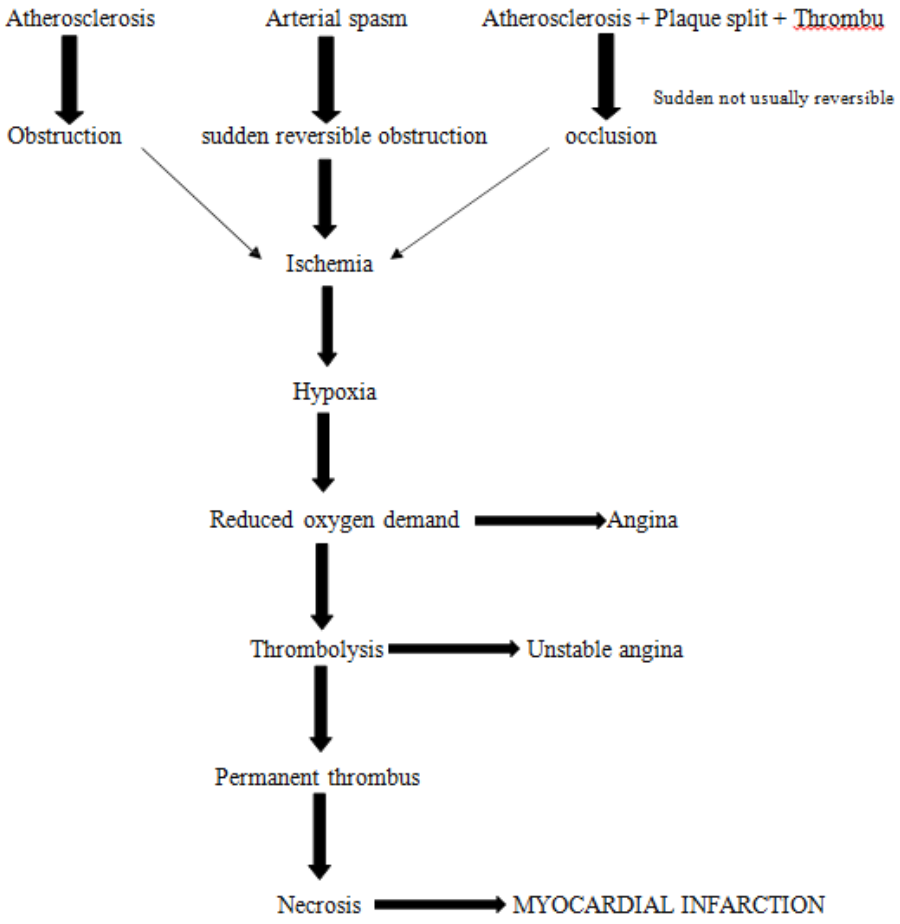
- **Abnormal heart rhythms (arrhythmias).** Electrical "short circuits" can develop, resulting in abnormal heart rhythms, some of which can be serious, even fatal.
- **Heart failure.** An attack might damage so much heart tissue that the remaining heart muscle can't pump enough blood out of your heart. Heart failure can be temporary, or it can be a chronic condition resulting from extensive and permanent damage to your heart.
- **Myocardial rupture.**
- **Arrhythmia.** Since the electrical characteristics of the infarcted tissue change arrhythmias are a frequent complication. The re-entry phenomenon may cause rapid heart rates (ventricular tachycardia and even ventricular fibrillation), and ischemia in the electrical conduction system of the heart may cause a complete heart block (when the impulse from the sinoatrial node, the normal cardiac pacemaker, does not reach the heart chambers).
- **Pericarditis.** As a reaction to the damage of the heart muscle, inflammatory cells are attracted. The inflammation may reach out and affect the heart sac. This is called pericarditis. In Dressler's syndrome, this occurs several weeks after the initial event. If pericarditis were to persist, pericardial effusion may also occur which could in turn lead to cardiac tamponade if not properly treated.
- **Cardiogenic shock.** A complication that may occur in the acute setting soon after a myocardial infarction or in the weeks following is cardiogenic shock. Cardiogenic shock is defined as a hemodynamic state in which the heart cannot produce enough of a cardiac output to supply an adequate amount of oxygenated blood to the tissues of the body

Symptoms

- Anxiety, commonly described as a sense of impending doom
- Pain or discomfort in areas of the body, including the arms, left shoulder, back, neck, jaw, or stomach
- Lightheadedness, with or without syncope
- Cough
- Nausea, with or without vomiting
- Profuse sweating
- Shortness of breath

- Wheezing
- Rapid or irregular heart rate
- Fullness, indigestion, or choking feeling

Pathophysiology



Risk factors

- **Age.** Men age 45 or older and women age 55 or older are more likely to have a heart attack than are younger men and women.
- **Tobacco.** This includes smoking and long-term exposure to second hand smoke.
- **High blood pressure**. Over time, high blood pressure can damage arteries that feed your heart. High blood pressure that occurs with other conditions, such as obesity, high cholesterol or diabetes, increases your risk even more.
- **High blood cholesterol or triglyceride levels.** A high level of low-density lipoprotein (LDL) cholesterol (the "bad" cholesterol) is most likely to narrow arteries. A high level of triglycerides, a type of blood fat related to your diet, also ups your risk of heart attack. However, a high level of high-density lipoprotein (HDL) cholesterol (the "good"

cholesterol) lowers your risk of heart attack.

- **Obesity.** Obesity is associated with high blood cholesterol levels, high triglyceride levels, high blood pressure and diabetes. Losing just 10 percent of your body weight can lower this risk, however.
- **Diabetes.** Not producing enough of a hormone secreted by your pancreas (insulin) or not responding to insulin properly causes your body's blood sugar levels to rise, increasing your risk of heart attack.
- **Metabolic syndrome.** This occurs when you have obesity, high blood pressure and high blood sugar. Having metabolic syndrome makes you twice as likely to develop heart disease as if you don't have it.
- **Family history of heart attack.** If your siblings, parents or grandparents have had early heart attacks (by age 55 for male relatives and by age 65 for female relatives), you might be at increased risk.
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- **Stress.** You might respond to stress in ways that can increase your risk of a heart attack.
- **Illicit drug use.** Using stimulant drugs, such as cocaine or amphetamines, can trigger a spasm of your coronary arteries that can cause a heart attack7
- **Inflammation. There** are accumulating data that the inflammation is considered a major risk factor for atherosclerosis development perhaps exceeding the role of LDL (Libby et al, 2002; Smit et al, 2019). Increased levels of highly sensitive C-reactive protein (CRP) could be an indicator of CHD risk in healthy individuals irrespective of LDL values. Moreover, it is believed that the statins' role in protection against CAD is attributed to its role in reducing the inflammation.

Treatment

Non-Pharmacological therapy

- **Compliance** - give careful advice about disease, treatment, and self-help strategies
- **Diet** - ensure adequate general nutrition and, in obese patients, weight reduction
- **Salt** - advise patients to avoid high salt content foods and not to add salt (particularly in severe cases of congestive heart failure)
- **Fluid** - urge overloaded patients and those with severe congestive heart failure to

restrict their fluid intake

- **Alcohol** - advise moderate alcohol consumption (abstinence in alcohol related cardiomyopathy)
- **Smoking** - avoid smoking (adverse effects on coronary disease, adverse hemodynamic effects)
- **Exercise** - regular exercise should be encouraged
- **Vaccination** - patients should consider influenza and pneumococcal vaccinations

Pharmacological Therapy

The goals of pharmacotherapy for myocardial infarction are to reduce morbidity and to prevent complications. The main goals of emergency department medical therapy are rapid intravenous thrombolysis and/or rapid referral for percutaneous coronary intervention (PCI), optimization of oxygenation, reduction of cardiac workload, and pain control.

Medical Management

- In Medical Management to improve the patient quality of life with the help of this drugs such as
- Clopidogrel, Atorvastatin, Aspirin, Beta Blockers.
- In some cases of LVD dysfunction Lasix and Fruselac are prescribed in order to Improve patient QOL.
- **Aspirin.** Aspirin reduces blood clotting, thus helping maintain blood flow through a narrowed artery.
- **Thrombolytic.** These drugs, also called clot-busters, help dissolve a blood clot that's blocking blood flow to your heart.
- **Anti Platelet agents.** Emergency room doctors may give you other drugs known as platelet aggregation inhibitors to help prevent new clots and keep existing clots from getting larger.

Other blood-thinning medications

- You'll likely be given other medications, such as heparin, to make your blood less "sticky" and less likely to form clots. Heparin is given intravenously or by an injection under your skin. Pain relievers. You might be given a pain reliever, such as morphine.
- **Nitro-glycerine**. This medication, used to treat chest pain (angina), can help improve blood flow to the heart by widening (dilating) the blood vessels.

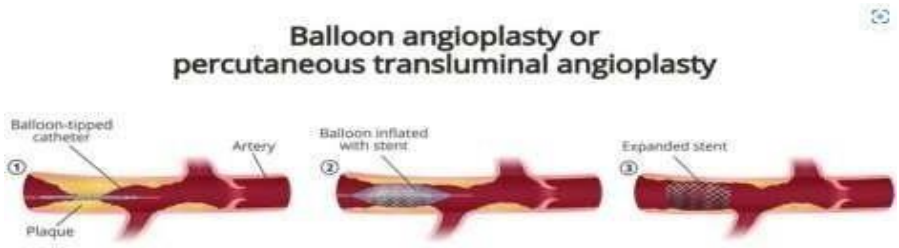
- **Beta blockers.** These medications help relax your heart muscle, slow your heartbeat and decrease blood pressure, making your heart's job easier. Beta blockers can limit the amount of heart muscle damage and prevent future heart attacks.
- **ACE inhibitors.** These drugs lower blood pressure and reduce stress on the heart.
- **Statins.** These drugs help control your blood cholesterol

Surgical management

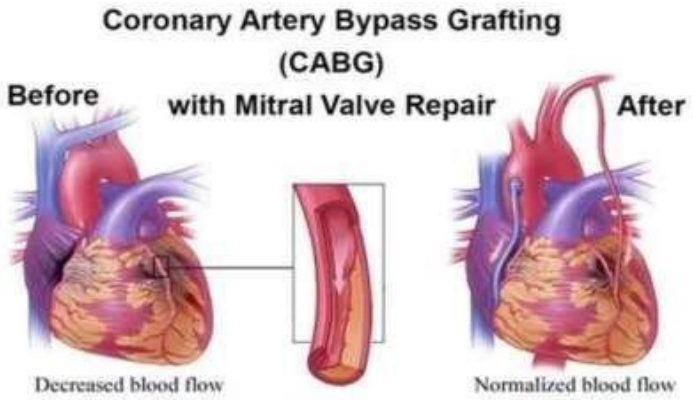
Angioplasty: PTCA (Percutaneous transluminal coronary angioplasty) a procedure to restore blood flow through artery. The doctor threads a thin tube through a blood vessel in the arm or groin up to the involved site in the artery and insert a thin tube into the artery into arm or leg and gently guide towards the problem area in your heart once the tube is in place a small balloon is briefly inflated in order to widen the narrowed artery a short length of mesh tubing called a stent is then inserted into the newly widened artery.

Types of angioplasty

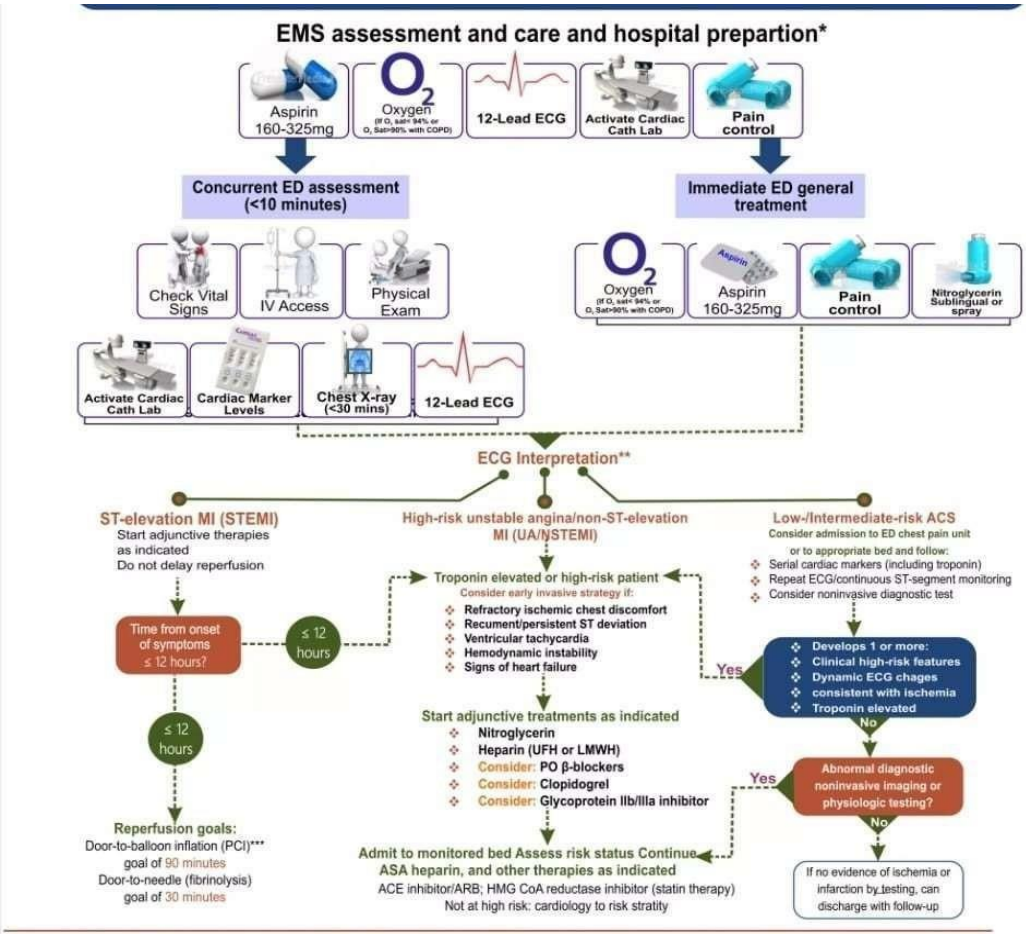
- Balloon angioplasty
- Carotid artery angioplasty (groin to carotid arteries)
- Cerebral angioplasty
- Coronary artery stent
- Laser angioplasty, PTCA OF Femoral artery



CABG (Coronary artery Bypass graft): a surgical procedure performed to redirect blood flow from one area to another by reconnecting blood vessels.



Standard treatment/Algorithm



METHODOLOGY

MATERIALS

- Patient documentation form
- Plan on primary consultant
- Medication chart
- Discharge summary

- Quality of life questionnaire

METHODS

Study design and study period

It is a prospective observational study conducted over a period of 6 months

Study Site

The study was conducted in Gemcare kamineni hospital, Kurnool in the department of Cardiology.

Sample Size

Total numbers of 79 patients were recruited from the departments of Cardiology, in Gemcare kamineni hospitals Kurnool.

STUDY CRITERIA

INCLUSION CRITERIA

- All individuals eligible for participation are men and women with age groups of 20-85 with Myocardial infarction diagnosed with STEMI or NSTEMI based on clinical presentation, ECG & cardiac Biomarkers

EXCLUSION CRITERIA

- Patients with inability to provide informed consent
- Patients with pregnancy or breastfeeding

RESULTS

Table 7.1: DISTRIBUTION BASED ON GENDER.

A total of 79 patients were included in our study. Out of them 56(70%) were males and 23(30%) were females.

S. No	Gender	No. of patients	Percentage
1	Male	56	70%
2	Female	23	30%
Total		**79**	**100%**

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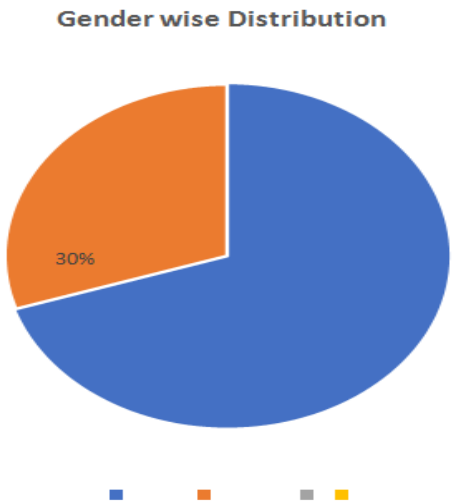


Fig 7.1: REPRESENTING THE DISTRIBUTION BASED ON GENDER.

Table 7.2: DISTRIBUTION BASED ON AGE.

According to age wise categorization, about patients were in the age of (38%) patients were in the age group 31 – 40, (28%) patients were in the group 41-50, (21%) patients were in the age group 51-60, (6%) patients were in the group 61-70, (%) patients were in the age of 71- 80.

S. No	Age Group	No. of Patients	Percentage
1	20 - 30	0	0%
2	31 - 40	30	38%
3	41 - 50	22	28%
4	51 - 60	16	21%
5	61 - 70	05	6%
6	71 - 80	06	7%
Total		**79**	**100%**

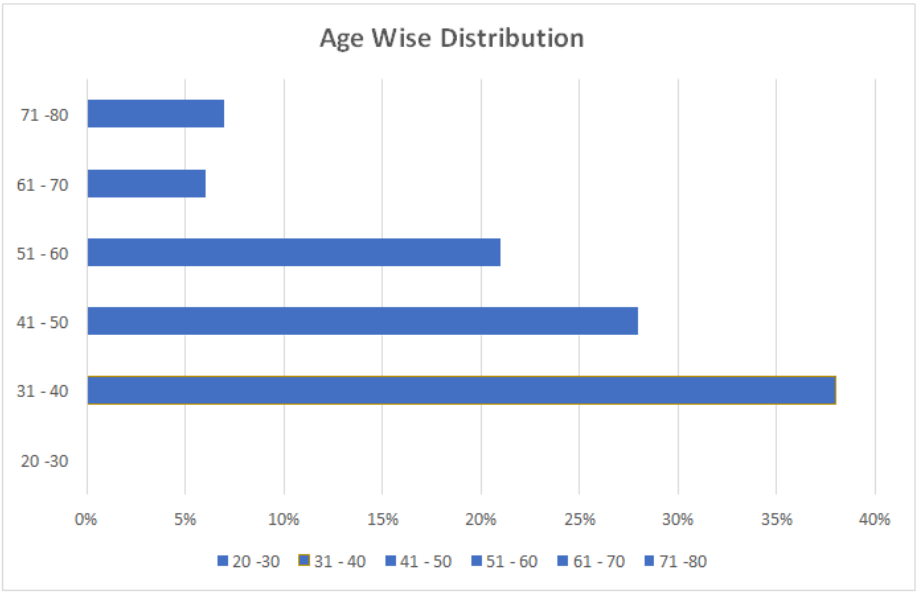


Fig 7.2: REPRESENTING THE DISTRIBUTION BASED ON AGE.

Table 7.3: DISTRIBUTION BASED ON Gender wise distribution in STEMI and NSTEMI.

S. No	GENDER	STEMI	NSTEMI
1	FEMALE	05	18
2	MALE	20	36
TOTAL		**25**	**54**

According to gender wise categorization, in STEMI patients were in (05) females, and were in (20) males. Then NSTEMI patients were in (18) females & (36) in males.

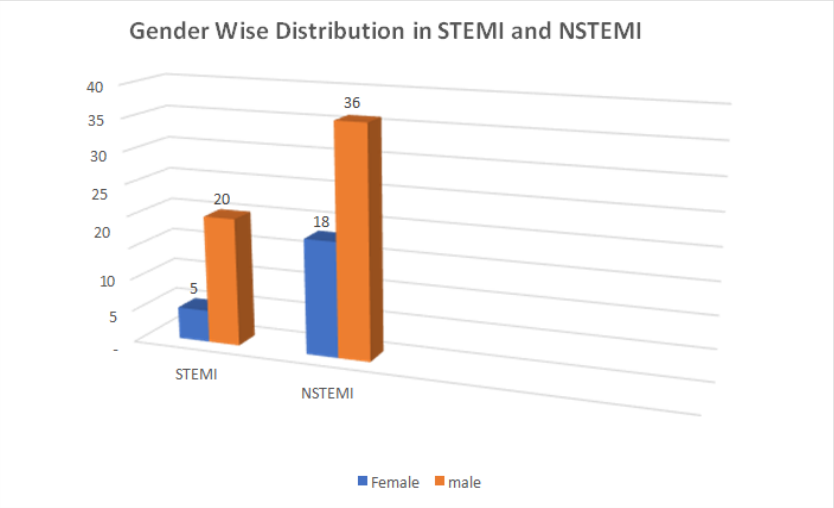


Fig 7.3: Representing Gender wise distribution in STEMI and NSTEMI.

Table 7.4: DISTRIBUTION OF PATIENTS BASED ON CO-MORBIDITIES

Out of 79 patients 21(28%) were without any co-morbidities and 24(30%) of HTN, 08(10%) of TYPE II Diabetes, 20(25%) of Hypothyroidism, were presented with co-morbidities along with MI.

S. No	C0-Morbidities	No. Of patients	Percentage
1	Hypertension	24	30%
2	Type II Diabetes	08	10%
3	Hypothyroidism	20	25%
4	DM + HTN	01	1%
5	COPD	04	5%
6	CKD	01	1%
7	NONE	21	28%
Total		**79**	**100%**

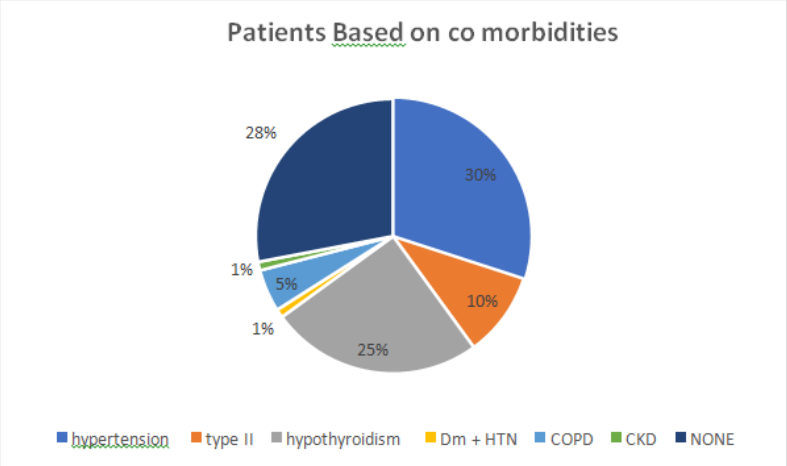


Fig 7.4: REPRESENTATION OF DISTRIBUTION OF PATIENTS BASED ON CO-MORBIDITIES.

Table 7.5: DISTRIBUTION the co-morbidities related drugs.

S. No	C0-Morbidities	TREATMENT
1	Hypertension	Metoprolol, ISMO
2	Type II Diabetes	Glimepride
3	Diabetes mellitus + HTN	Metformin +Telmisartan

Table 7.6: DISTRIBUTION Myocardial Infarction patients with social habits.

Out of 79 patients 10(7.9%) where they are smokers and 08(6.3%) of alcoholic 18(14.2%) of Both 43(73%) don't have any social habits.

S. No	SMOKER	ALCOHOLIC	BOTH	NONE
1	10	08	18	43

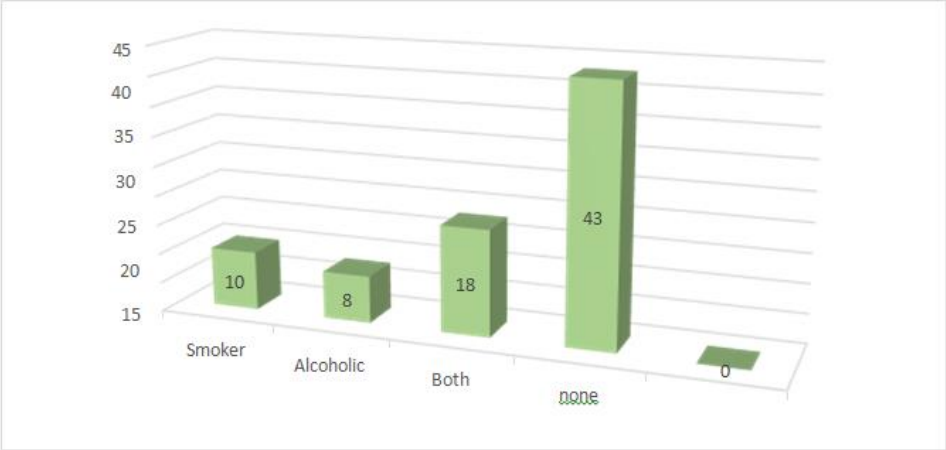


Fig. 7.5: representing DISTRIBUTION Myocardial Infarction patients with social habits.

Table 7.7: REPRESENTING OCCURANCE OF DISEASE IN SINGLE & MULTI VESSEL DISESE IN MI

Based on disease they were differentiated into SVD, DVD, TVD. In SVD, STEMI were 12 patients & NSTEMI were 21 patients. In DVD, STEMI were 9 patients & NSTEMI were 18 patients. In TVD, STEMI were 3 patients & NSTEMI were 16 patients.

DISEASE	SVD	DVD	TVD
STEMI	12	09	03
NSTEMI	21	18	16

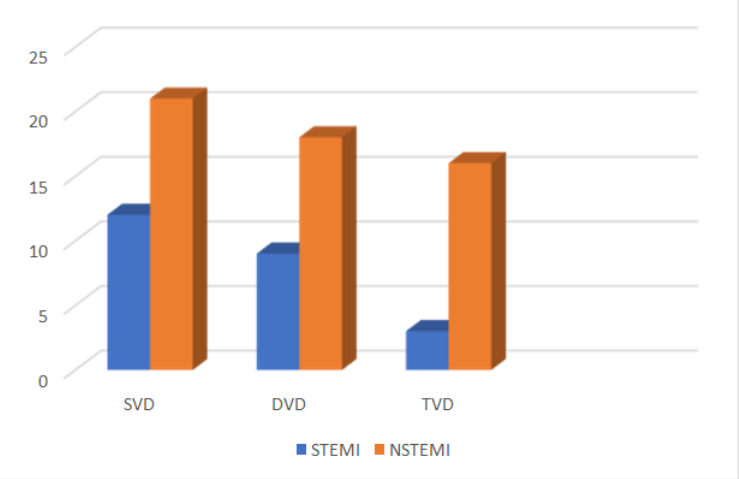


Fig 7.6: REPRESENTING OCCURANCE OF DISEASE IN SINGLE & MULTI VESSEL DISESE IN MI.

Table 7.8: REPRESENTING PERCENTAGE OF REVASCULARIZATION IN BOTH MALE & FEMALE.

Based on percentage of revascularization on PTCA and Medical Management. In PTCA males were 32 patients & females were 13 patients. In medical management males were 24 patients & females were 10 patients.

S. No	GENDER	PTCA	MEDICAL MANAGEMENT
1	MALE	32	24
2	FEMALE	13	10

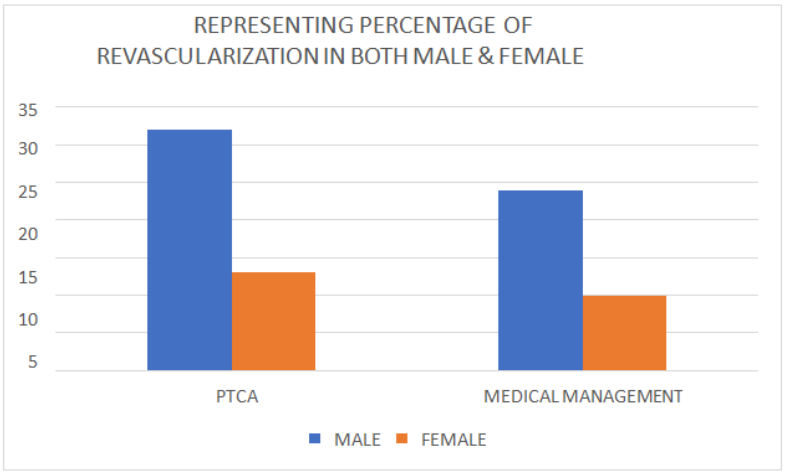
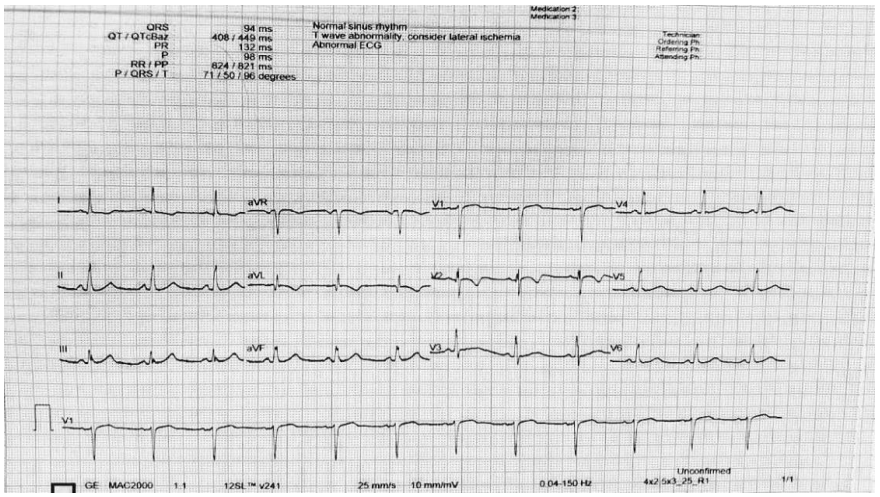


Fig 7.7: REPRESENTING PERCENTAGE OF REVASCULARIZATION IN BOTH MALE & FEMALE.



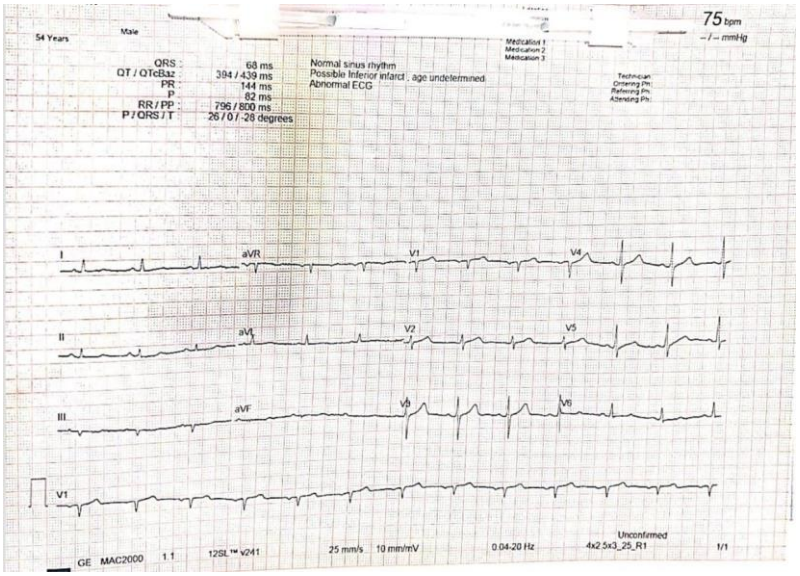


Fig 7.8: Representing the clinical scenario on STEMI & NSTEMI patients (ECG) STEMI.

(CARDIC BIOMARKERS)

SNO	PARAMETERS	NSTEMI	STEMI
1	TROPONIN -I	POSITIVE	POSITIVE

Table 7.9: Representing the prevalence of STEMI and NSTEMI patients.

SNO	GENDER	NSTEMI	STEMI	RECOVERD	MORTALITY
1	MALE	36	20	56	0
2	FEMALE	18	05	23	0
	TOTAL	**54**	**25**	**79**	**0**

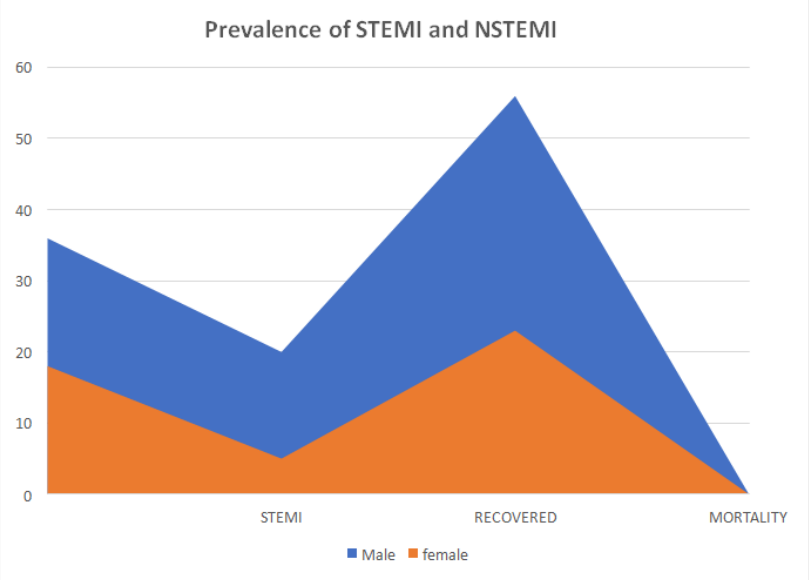


Fig 7.9: Representing the prevalence of STEMI and NSTEMI patients.

Based on disease patients where in to NSTEMI & STEMI.in STEMI males were 36 patients & females are 18 INSTEMI, males for 20 patients &females for 5 patients. Recovered in male are 56 & in female were 23 patients.

QUALITY O U T COMES IN STEMI BEFORE PTCA

Table 7.10: Distribution of patients based on staging before PTCA in STEMI.

Stages	Before PTCA	
	Male	Female
Mild	3	0
Moderate	10	2
Severe	7	3
Total	**20**	**5**

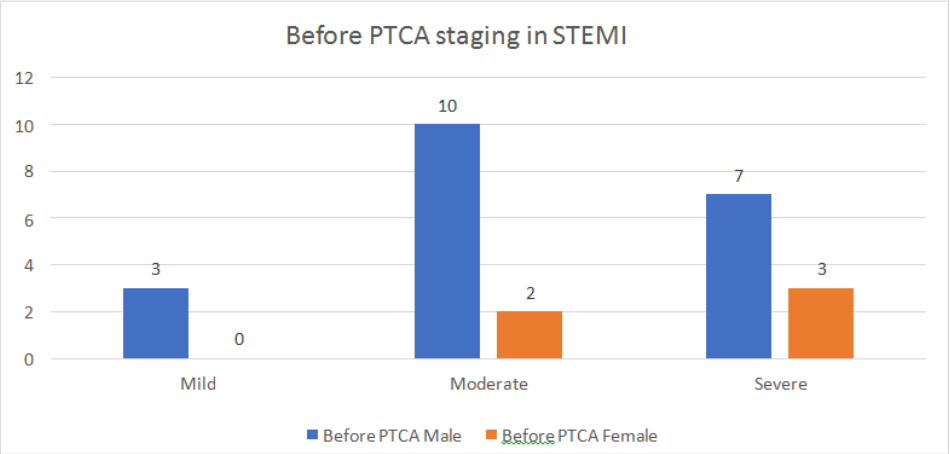


Fig. 7.10: Representing Distribution of patients based on staging before PTCA in STEMI.

The bar graph represents the distribution of STEMI patients according to the severity of disease staging (Mild, Moderate, Severe) before undergoing Percutaneous Transluminal Coronary Angioplasty (PTCA), categorized by gender.

- Improvement in severe cases for both genders after PTCA.
- Increase in mild and moderate cases post-treatment, indicating clinical improvement or shift in classification.
- Males had higher representation in all categories both before and after PTCA**.**

Table 7.11: Distribution of patients based on staging After PTCA in STEMI.

Stages	**After PTCA**	
	Male	**Female**
Mild	6	1
Moderate	12	3
Severe	2	1
Total	**20**	**5**

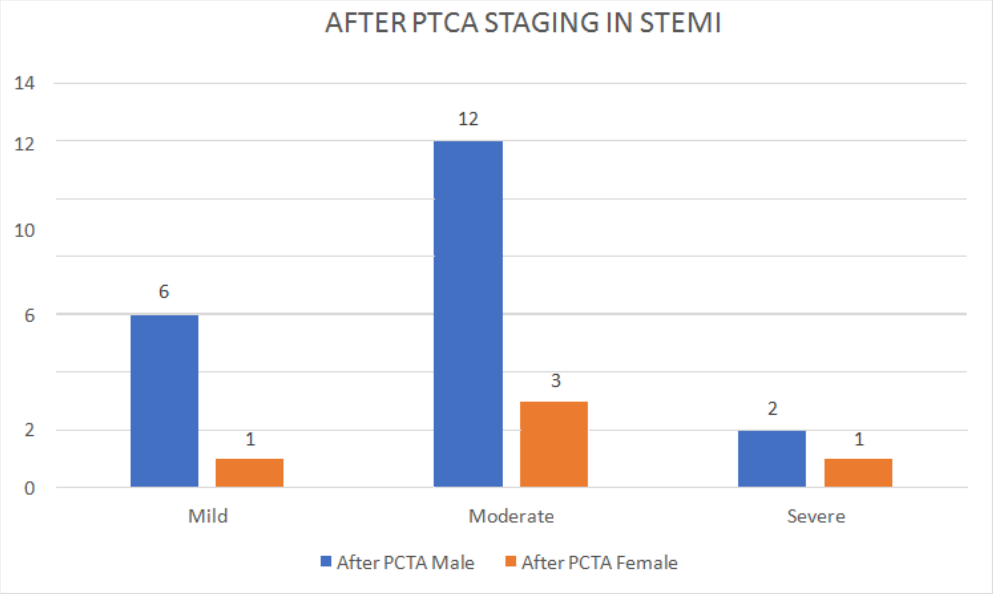


Fig 7.11 Representing Distribution of patients based on staging After PTCA in STEMI.

Here in this graph represents the Distribution of patients based on staging After PCTA in NSTEMI in this

- Males had higher numbers in all categories.
- Most males were in the "Moderate" category post-PCTA.
- Females showed fewer cases overall, with the highest number also in the "Moderate" category.

Table 7.12: Distribution of patients based on staging before PTCA in NSTEMI.

Stages	**Before PTCA**	
	Male	**Female**
Mild	10	2
Moderate	13	10
Severe	13	6
Total	**36**	**18**

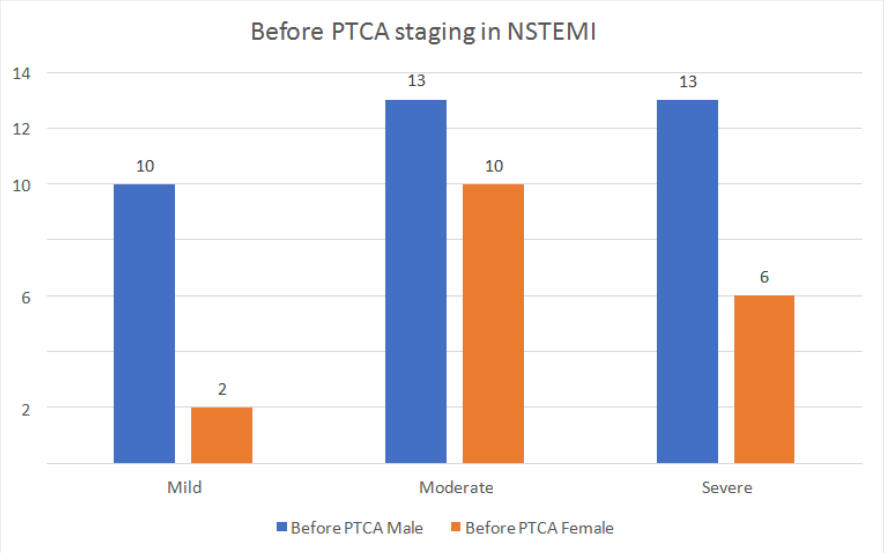


Fig 7.12: Distribution of patients based on staging before PTCA in NSTEMI.

The bar graph represents the distribution of NSTEMI patients according to the severity of disease staging (Mild, Moderate, Severe) before undergoing Percutaneous Transluminal Coronary Angioplasty (PTCA), categorized by gender.

In the Mild stage, a higher number of male patients were observed compared to females. In the Moderate stage, both genders showed a notable presence, with males slightly higher than females. However, in the Severe stage, males again dominated, while the number of female patients reduced to 6.

Table 7.13: Distribution of patients based on staging After PTCA in NSTEMI.

Stages	**After PTCA**	
	Male	**Female**
Mild	14	4
Moderate	16	11
Severe	6	3
Total	**36**	**18**

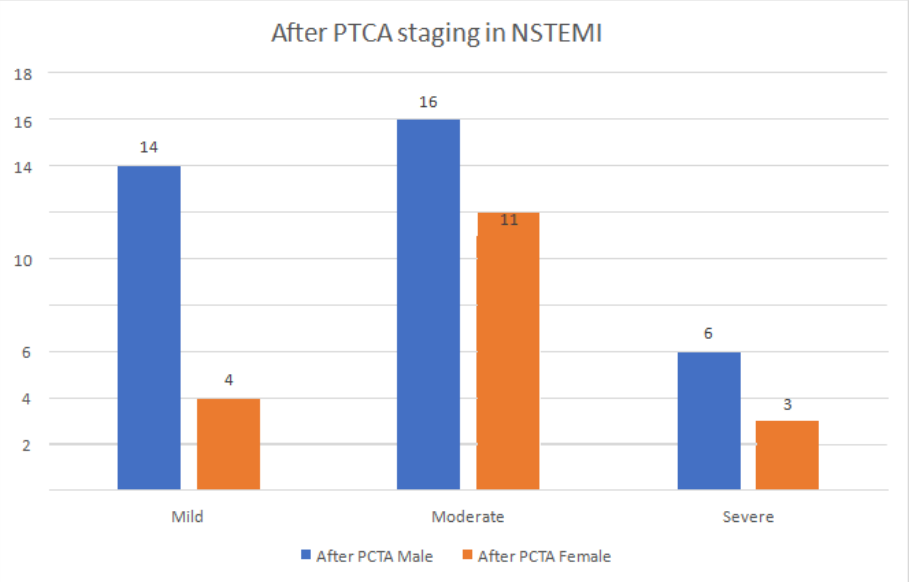


Fig 7.13: Distribution of patients based on staging After PTCA in NSTEMI.

Here in this graph represents the Distribution of patients based on staging After PCTA in NSTEMI in this

- Males have higher counts than females in all three stages.
- The Moderate stage has the highest number of patients overall for both genders.
- Females show lower counts, especially in the Mild and Severe stages.

Table 7.14: Representing the prescribing patterns of drug in STEMI & NSTEMI.

DRUGS	STEMI	NSTEMI
ECOSPIRIN	**20**	**52**
CLOPIDOGREL	**15**	**20**
METOPROLOL	**14**	**22**
ATORVASTATIN	**25**	**50**
HEPARIN	**18**	**40**
TICAGRELOR	**08**	**30**
FUROSEMIDE	**12**	**25**
ALPRAZOLAM	**18**	**30**

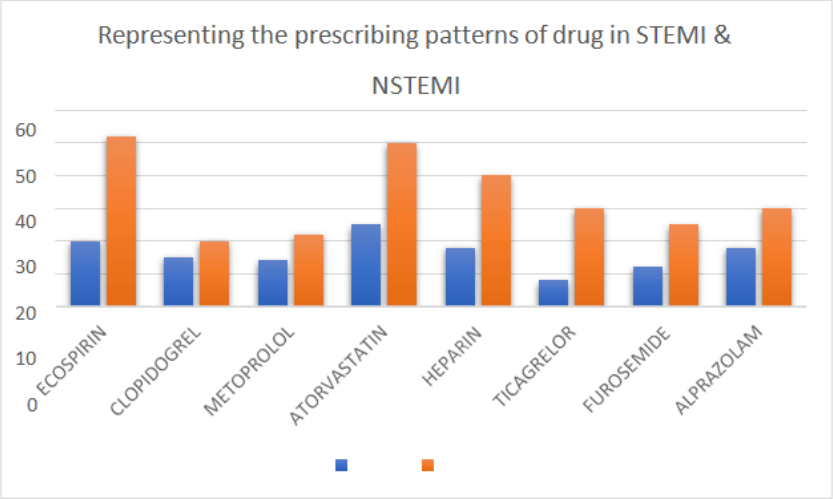


Fig 7.14: Representing the prescribing patterns of drug in STEMI & NSTEMI.

Treatment of Myocardial Infarction involves various categories of drugs namely Anti platelet drugs, Anticoagulants, Anti Anginal, Anti hypertensives, and Anti hyperlipidemic, Beta Blockers, Anti-Anxiety agents.

DISCUSSION

Myocardial Infarction (MI) occurs when the blood flow to a part of the heart muscle is blocked**,** usually by a blood clot in a coronary artery**.** Without oxygen-rich blood, the heart muscle tissue begins to die, which can cause serious damage or be fatal.

The mainly triggered by many risk factors such as blood plessor, high cholesterol, obesity, diabetes, which can be control by healthy diet and regular exercise and by avoiding smoking & alcohol.

This study state that hypertension and diabetes were the most common co-morbid conditions associated with myocardial infarction and Our findings revealed that STEMI was more prevalent in younger males**,** often presenting with sudden-onset, severe chest pain and clear ECG changes. In contrast, NSTEMI patients were typically older with a higher burden of comorbidities such as diabetes, hypertension, and chronic kidney disease. In this present study myocardial infarction was seen in males (70%) and (30%) females and mostly seen NSTMI cases when compare to STMI patients.

In terms of treatment patterns**,** STEMI patients predominantly received reperfusion therapy (thrombolysis or primary PCI) in the acute phase, while NSTEMI patients were more likely to undergo risk stratification followed by conservative or delayed interventional strategies. These

treatment differences were guided by standard protocols and influenced by ECG changes, troponin levels, and TIMI risk scores. The use of guideline recommended pharmacotherapy was quite high in our study including dual antiplatelet therapy (DAPT) that was administered to nearly all patients. this usage is much higher than in many Indian studies and comparable with studies from the western world.

In our study the drugs prescribing in medical management and PTIC clopidogrel, aspirin, atorvastatin, betablockers, in some cases LV dysfunction Furosemide are prescribed.

In our current study we observed expose of MI in age group 31-40yrs (38%) &41-50 yr (28%) this groups are mostly expose to the MI.

In this study mostly effect to NSTEMI (65%) in both male &females exposed when compared to STEMI (35%) and in this study we are observed clinical scenario of STEMI patients typically presented with sudden, intense, and prolonged chest pain, often radiating to the left arm or jaw, associated with autonomic symptoms such as diaphoresis and nausea. Electrocardiogram (ECG) showed persistent ST-segment elevation**,** and elevated cardiac biomarkers confirmed the diagnosis.

And in NSTEMI patients, however, had more atypical presentations**,** including epigastric discomfort, fatigue, dyspnea**,** or even silent infarction, especially in diabetic or elderly populations. ECG often revealed ST-segment depression, T-wave inversions**,** these patients were more likely to be hemodynamically stable at admission but faced higher in-hospital complications due to comorbidities**.**

In current study was observed the patient quality of before and after in best on the WHOQOL scale and we observed based on score and we find that STMI patients generally had better QoL outcomes, particularly in the physical and psychological domains**,** likely due to more rapid intervention and recovery. In NSTEMI patients reported lower QoL scores across all four domains (physical, psychological, social, and environmental)**,** emphasizing the impact of comorbidities.

We are calculated the scale based on the score before and after with mild is (>60) and moderate (>40) and severe (<30) and based on this score we can identify the patient quality of life.

In STMI subgroup anterior wall MI was common that inferior MI Similar to the study by jose

And Gupta. The results however differed from study by Singh.

In our study quality of life in STEMI and NSTEMI was related to the study by prihati Pujowaskito and Dinda Dwi Lazuardi emha. These results however differed from our study.

CONCLUSION

STEMI and NSTEMI are both serious acute coronary syndromes but differ significantly in patient demographics, treatment approaches, and post-recovery outcomes. STEMI patients, typically younger, receive rapid intervention through PCI, leading to faster recovery and better quality of life. In contrast, NSTEMI patients, often older with more comorbidities, require a more cautious treatment approach and tend to report lower physical and emotional health outcomes post- discharge.

The study highlights the importance of timely diagnosis, personalized treatment plans, and enhanced post-discharge care especially for NSTEMI patients. Greater adherence to guideline- directed therapy and tailored management strategies are crucial for improving long-term survival and overall patient well-being.

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