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A META-ANALYSIS OF PREVALENCE, RISK FACTORS, COMORBIDITIES AND TREATMENT PATTERNS IN MYOCARDIAL INFARCTION

A.Gunalatha *, T.Mngilal,Shaista Tabassum, K.Nagalaxmi, B.Anusha, J.Anand Kumar, Ali Abdul Basith

Smt.Sarojini Ramulamma College of Pharmacy, Mahabubnagar-509001, Telangana, India.

Abstract:

Myocardial Infarction (MI) remains one of the leading causes of morbidity and mortality worldwide and represents a major public health challenge. This systematic review aimed to evaluate the prevalence, demographic characteristics, associated comorbidities, risk factors, and treatment approaches among patients with myocardial infarction. Data from 18 published studies comprising more than 85,725 patients were analyzed. The findings demonstrated a marked male predominance, with 72.4% males and 27.6% females among the study population. The findings demonstrated a higher prevalence of myocardial infarction among males, with most patients belonging to the middle-aged and elderly population. Hypertension, diabetes mellitus, dyslipidemia, chronic kidney disease, and obesity were the most commonly reported comorbidities. Major risk factors included smoking, advancing age, sedentary lifestyle, alcohol consumption, genetic predisposition, and psychosocial stress. Treatment modalities varied across studies and included antiplatelet agents, statins, beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, thrombolytic therapy, and percutaneous coronary intervention (PCI), which remain the cornerstone of contemporary myocardial infarction management. The reviewed studies consistently emphasized the importance of early diagnosis, prompt treatment, and effective risk-factor modification in improving clinical outcomes. This review highlights the substantial burden of myocardial infarction and underscores the need for comprehensive preventive strategies, multidisciplinary management, and adherence to evidence-based therapies to reduce mortality and improve patients' quality of life.

Keywords: *Myocardial Infarction; Acute Coronary Syndrome; Cardiovascular Risk Factors; Hypertension; Diabetes Mellitus; Dyslipidemia; Smoking; Comorbidities; Treatment Outcomes; Percutaneous Coronary Intervention; Mortality; Systematic Review.*

Corresponding author:

A.Gunalatha,
Department of Pharmacology,
Smt.Sarojini Ramulamma College of Pharmacy,
Mahabubnagar-509001, Telangana, India.
Email-asreddy115@gmail.com,Mobile-+919346763662.

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INTRODUCTION:

A heart attack occurs when the flow of blood to the heart is severely reduced or blocked. The blockage is usually due to a buildup of fat, cholesterol and other substances in the heart (coronary) arteries. The fatty, cholesterol-containing deposits are called plaques. The process of plaque buildup is called atherosclerosis. Sometimes, a plaque can rupture and form a clot that blocks blood flow. A lack of blood flow can damage or destroy part of the heart muscle. A heart attack is also called a myocardial infarction. (1)

Types of Myocardial Infarction

Type 1 MI: Plaque rupture causing coronary thrombosis.
Type 2 MI: Oxygen supply-demand imbalance without thrombosis.
Type 3 MI: Sudden cardiac death due to suspected myocardial ischemia.
Type 4a MI: MI associated with PCI.
Type 4b MI: MI caused by stent thrombosis.
Type 5 MI: MI associated with CABG surgery.

Etiology

Smoking
High Blood Pressure
Being Overweight
Cholesterol
Inactivity
Diet
Diabetes
Family History

Heart attack Symptoms

Chest pain or discomfort, which may spread to your jaw, neck, shoulder, arm or back
Shortness of breath or trouble breathing
Feeling very tired or weak for no clear reason
Upset stomach, nausea or vomiting
Anxiety or feeling like something bad is going to happen
Sweating a lot
Feeling dizzy or lightheaded
Passing out

It's possible to have a heart attack without feeling chest pain. Some people only have shortness of breath, nausea or sweating. You might hear this called a "silent heart attack." That's because it doesn't have the traditional symptoms. But it's still a heart attack. And you still need care right away.

Pathophysiology

Myocardial infarction (MI) results from a severe and prolonged imbalance between the myocardial O2 supply and its requirements. In most cases, this occurs as a result of occlusive coronary atherosclerosis superimposed with luminal thrombus (Christia and Frangogiannis, 2013). The atherosclerosis is a dynamic, progressive process. Its effects are attributed to a combination of inflammation and endothelial dysfunction (Szmitko et al, 2003). (2)In about 10% of cases, myocardial infarction may occur without detection of coronary artery obstruction. The prognosis and management in such cases are different from coronary artery occlusion (Niccoli and Camici, 2020). (3) The exact mechanism in non-obstructive cases has not yet been clearly established. However, some potential causes such as coronary artery spasm, plaque disruption, spontaneous coronary artery dissection, in-situ thrombosis and microvascular dysfunction have been suggested (Tamis-Holland et al, 2019; Hegazy et al, 2022). (4) In these cases, it is important to rule out other causes of elevated troponin such as myocarditis, Takotsubo cardiomyopathy and pulmonary embolism (Li et al, 2020).

The central mechanism of the pathophysiology is the same regardless of the etiological factors that may only modify the outcome. For example, hypertension is a main risk factor involved in strokes while smoking increases the incidence of MI (Bentzon et al, 2014). (5)

The pathophysiology process can be summarized in the following steps:

Endothelial dysfunction

Coronary endothelial dysfunction precedes the lesion formation and increases with progression of CAD. It results from endothelial injury that occurs in response to oxidized LDL, increased levels of blood glucose, hypertension and increased free radicals derived from oxygen. There is usually a decrease in release of endothelial nitric oxide (NO) that affects vasomotion; thus, an increase in platelet and monocyte adhesions occurs with increased smooth muscle cell proliferation (Lüscher et al, 2009). The NO inhibits atherogenesis by promoting vasodilation and reducing the platelets' adhesions to the vascular endothelium (de Graaf et al, 1992). (6)

Plaque and necrotic core formation

Atherosclerosis is the main cause of plaque formation by causing internal inflammation, fibrosis, necrosis and calcification. The direct mechanism is through accumulation of LDLs in the arterial endothelium with an intrinsic adaptive thickening (Bentzon et al, 2014). Then, LDLs become modified by oxidation as well as aggregation. The modified LDLs produce chronic stimulation of innate and adaptive immunity resulting in differentiation of monocytes to phagocytes . These phagocytes engulf lipid particles forming so-called foam cells that are the hallmark of atherosclerotic plaques (Libby et al, 2011). The endothelium is then becoming leaky (Bentzon et al, 2014). (7)

Matrix metallopeptidase 9 (MMP-9), also known as gelatinase B, 92-kDa type IV collagenase, has been produced by SMCs and macrophages in response to oxLDL, reactive oxygen specious (ROS) and tumour necrosis factor (TNF)- α (Galis and Khatri, 2002). The level of MMP-9 is increased in cases of unstable angina and MI. Overexpression of MMP-9

enhances the process of atherosclerosis and fibrous cap degradation of plaques and intravascular thrombosis (Belaaouaj et al, 2000; Morishige et al, 2003). (8)

Inflammation contributes to foam cell death and muscle fibers' migration. This could result in formation of necrotic core within the plaque. Therefore, a mature atheromatous plaque is composed of a lipid-rich necrotic core surrounded by collagen rich capsule (Smit et al, 2019). (8)

Plaque angiogenesis and intervening haemorrhage

New vessels originate from the vasa vasorum within the vessel adventitia and give another route for monocytes and immune cells to reach the base of atheromatous plaque (Kumamoto et al, 1995). These vessels are fragile resulting in bleeding and extravasation leakage to erythrocytes and plasma proteins. Such bleeding expands the plaque and promotes occurrence of more inflammation. Moreover, rupture of fibrous cap of plaque is another cause for bleeding (Bentzon et al, 2014). (9)

Fibrous cap rupture and thrombosis

Plaque rupture occurs through the thin fibrous cap. Rupture of vulnerable plaque could be preceded by severe exertion. The increase in amount of free cholesterol and phagocytes' infiltration in the plaque cap as well as the decrease in its thickness are factors involved in rupture mechanism (Burke et al, 1999). Plaque rupture might occur spontaneously; however, in other cases it could be triggered by psychological, physical or sexual stress, infections or substance abuse (Mittleman and Mostofsky, 2011). The triggered pathways' mechanism might be hastened through the increase in the blood pressure and heart rate leading to plaque rupture or via increasing the coagulability and thrombosis of platelets superimposed on already vulnerable plaque (Martin et al, 2012; Bentzon et al, 2014). (10)

Plaque rupture leads to exposure of blood with the necrotic core that is rich with erythrocytes and highly thrombogenic material leading to clot formation. Plaque rupture represents the most common cause for occurrence of thrombosis (Bentzon et al, 2014). This might be associated with acute coronary syndromes and myocardial infarction. The occlusive and sustained thrombosis often accompanies cases of ESTMI; however, in non- ESTMI, the thrombus is often dynamic and does not clog the vessel or is even absent (Davies, 2000). (11)

Treatment of Myocardial Infarction

Each minute after a heart attack, more heart tissue is damaged or dies. Urgent treatment is needed to fix blood flow and restore oxygen levels. Oxygen is given immediately. Specific heart attack treatment depends on whether there's a partial or complete blockage of blood flow.

Medications (12-20)

Medications to treat a heart attack might include

Aspirin

Aspirin reduces blood clotting. It helps keep blood moving through a narrowed artery. If you called 911 or your local emergency number, you may be told to chew aspirin. Emergency medical providers may give you aspirin immediately.

Clot busters (thrombolytics or fibrinolytics)

These drugs help break up any blood clots that are blocking blood flow to the heart. The earlier a thrombolytic drug is given after a heart attack, the less the heart is damaged and the greater the chance of survival.

Other blood-thinning medicines

A medicine called heparin may be given by an intravenous (IV) injection. Heparin makes the blood less sticky and less likely to form clots.

Nitroglycerin

This medication widens the blood vessels. It helps improve blood flow to the heart. Nitroglycerin is used to treat sudden chest pain (angina). It's given as a shot, a pill that goes under the tongue, as a pill to swallow or as a skin patch.

Morphine

This medicine is given to relieve chest pain that doesn't go away with nitroglycerin.

Beta blockers

These medications slow the heartbeat and decrease blood pressure. Beta blockers can limit the amount of heart muscle damage and prevent future heart attacks. They are given to most people who are having a heart attack.

Blood pressure medicines called angiotensin-converting enzyme (ACE) inhibitors

These drugs lower blood pressure and reduce stress on the heart.

Statins

These drugs help lower unhealthy cholesterol levels. Too much bad (low-density lipoprotein, or LDL) cholesterol can clog arteries.

Surgical and other procedures

If you've had a heart attack, a surgery or procedure may be done to open a blocked artery. Surgeries and procedures to treat a heart attack include:

Coronary angioplasty and stenting

This procedure is done to open clogged heart arteries. It may also be called percutaneous coronary intervention (PCI). If you've had a heart attack, this procedure is often done during a procedure to find blockages (cardiac catheterization).

During angioplasty, a heart doctor (cardiologist) guides a thin, flexible tube (catheter) to the narrowed part of the heart artery. A tiny balloon is inflated to help widen the blocked artery and improve blood flow.

A small wire mesh tube (stent) may be placed in the artery during angioplasty. The stent helps keep the artery open. It lowers the risk of the artery narrowing again. Some stents are coated with a medication that helps keep the arteries open.

Coronary artery bypass grafting (CABG)

This is open-heart surgery. A surgeon takes a healthy blood vessel from another part of the body to create a new path for blood in the heart. The blood then goes around the blocked or narrowed coronary artery. It may be done as an emergency surgery at the time of a heart attack. Sometimes it's done a few days later, after the heart has recovered a bit.

Materials and Methods

Study Design

The study was Meta-Analysis, observation study.

Source of data

Science basic studies

Editorials/letters

Articles

Study criteria

Inclusion criteria

All the articles showing prevalence of data in India.

Published between 2015-2025.

All articles showing Detailed information about myocardial infarction treatment safety and efficacy data.

Human studies.

Studies reporting: Age and Gender distribution, risk factors, co-morbidities, treatment details.

Exclusion criteria

Animal model studies.

Duplicate data.

Insufficient data.

Studies with complete data.

Method of data collection (21-23)

A systematic and comprehensive literature search was conducted across six major electronic database-PubMed, MEDICINE, Scopus, Embase, WEB OF Science and clinical trials. Gov-covering the period from January 1,2015, to dec 31, 2025. This t imeframe was selected to capture the critical advancements in myocardial infarction. Boolean operators (AND/OR), truncation symbols and database-specific filters (e.g., human studies, clinical trials, and English language) were applied to refine search results.

To enhance completeness, citation chaining, reference list checking and hand-searching of key journals and conference proceeding were conducted. Additionally, clinical trials registries and grey literature sources (i.e., non-peer-reviewed materials such as conference abstracts, dissertations, government reports, and preprints) were explored to capture unpublished or ongoing studies.

Statistical analysis (24-25)

It was done by using MS EXCEL.

Results

The systematic review and meta-analysis were done.

This study included the following factors or parameters:

1.Prevalence of myocardial infarction in different age groups.

2.Prevalence of myocardial infarction based on gender.

3.Prevalence of Risk factors in myocardial infarction.

4.Comorbidities in myocardial infarction patients.

5.General prescribed drugs in myocardial infarction.

Table age group Distribution in MI

AGE	PERCENTAGE (%)
30-45	1.2%
40-60	35.6%
50-70	3.3%
60-80	59.9%

According to the analysed data, myocardial infarction was most prevalent in the 60–80 years age group, indicating a higher risk in older populations. A significant number of cases were also observed in the 40–60 years age group, suggesting increasing incidence in middle-aged individuals. The least number of cases were reported in younger age groups (30–45 years).

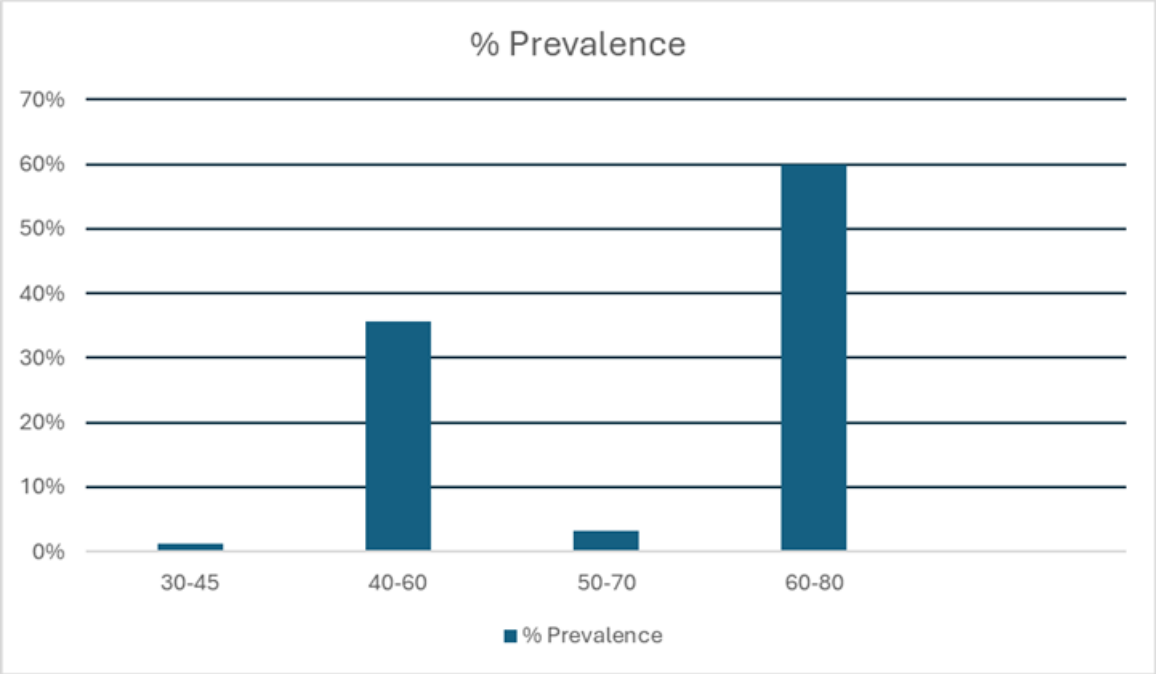

Table gender wise Distributions in MI

GENDER	PERCENTAGE
MALE	68%
FEMALE	32%

The gender-wise distribution of patients shows that males constitute approximately 68.43%, while females account for 31.57% of the total population. This indicates that myocardial infarction is significantly more prevalent in males compared to females.

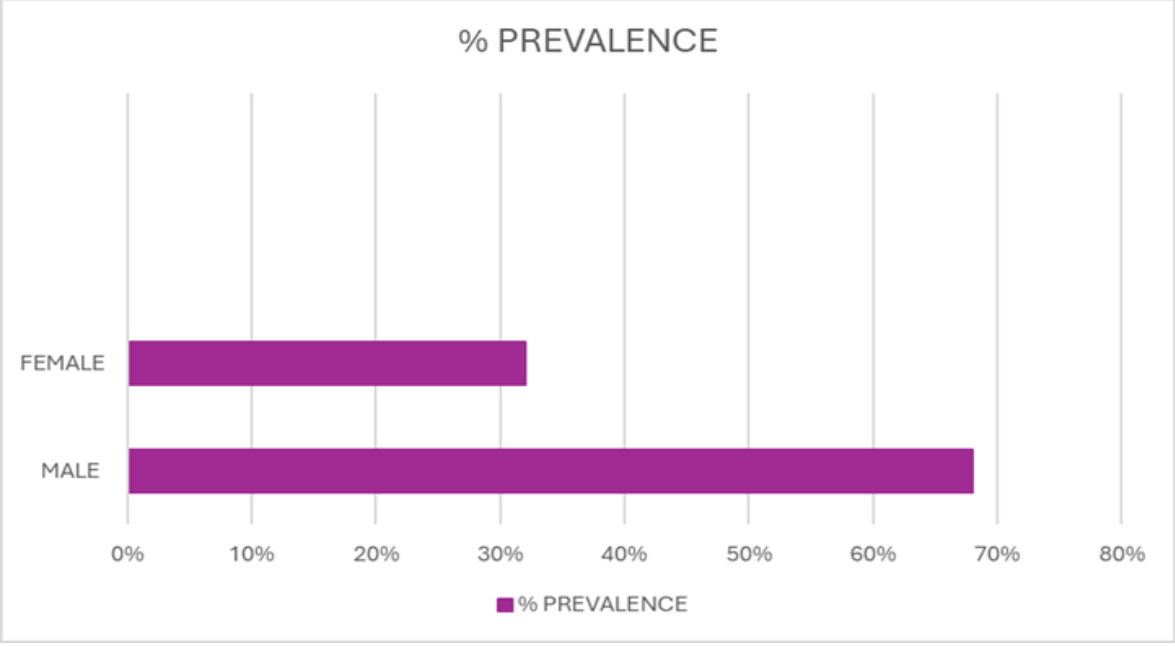

Figure Bar Graph showing prevalence of MI based on gender

The bar graph shows that males have higher prevalence at around 65-70%, compared to females have a lower prevalence at about 30-35%.

Risk factors in Myocardial Infarction

Table prevalence of risk factors in MI

RISK FACTORS	% PREVALENCE
OBESITY	**15%**
SMOKERS	**30%**
ALCOHOL	**20%**
STRESS	**25%**
DIET	**10%**

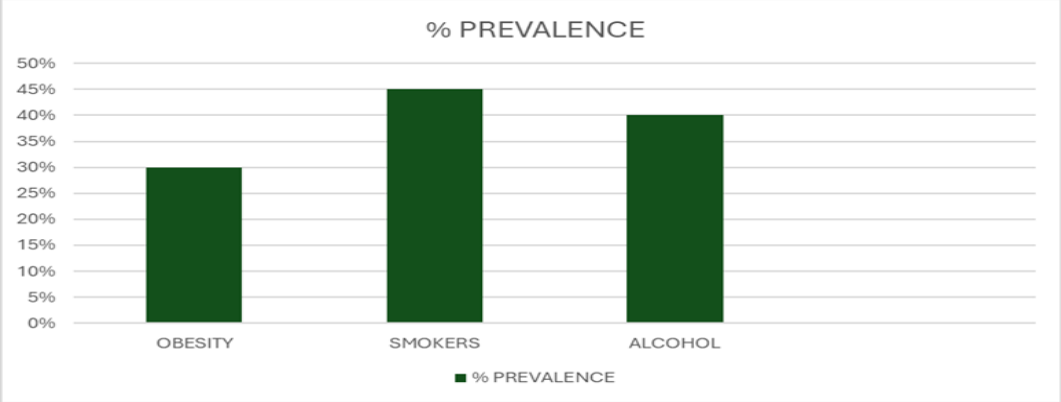

Figure bar graph showing prevalence of risk factors in MI

The bar graph shows the prevalence of risk factors in MI patients, with smoking being the most common, followed by obesity and alcohol used being slightly lower.

Prevalence Comorbidities in MI Patients

Table Comorbidities in MI Patients

COMORBIDITIES	% OF PREVALENCE
Hypertension	40%
DM	25%
CKD+HTN	20%
Dyslipidemia	15%

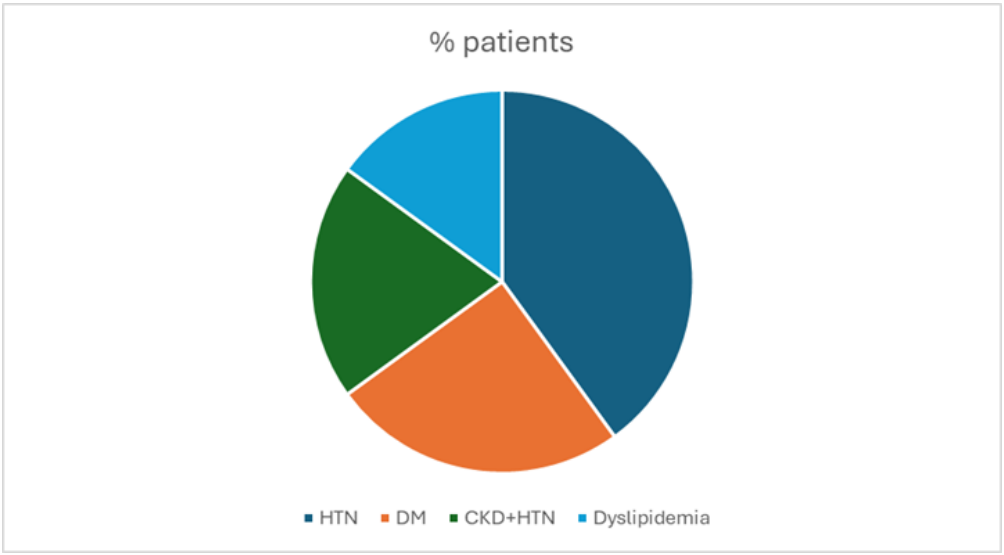

Figure PIE chart showing prevalence of Comorbidities in MI Patients

The pie chart shows the prevalence of comorbidities in MI patients with hypertension being the most common, followed by diabetes (DM) and CKD with HTN while the dyslipidaemia is the least common.

Prevalence of General prescribed drugs in Myocardial Infarction

Table general prescribed drugs in MI

TREATMENT	% OF DRUG PRESCRIBED
Antiplatlets	15%
Beta blockers	15%
ACE Inhibitors	30%
Diuretics	15%
Statins	25%

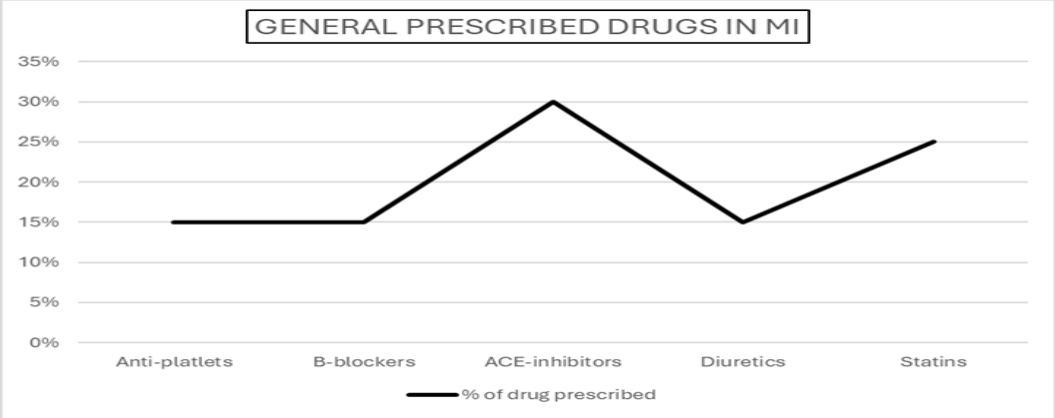

Figure line chart showing % 0f Drugs prescribed in MI Patients

The graph shows the percentage of different drugs prescribed to MI patients, with ACE inhibitors being the most prescribed and diuretics the least, while other drugs are used at moderate levels.

DISCUSSION:

The present meta-analysis was conducted to evaluate the prevalence, demographic characteristics, risk factors, comorbidities, and treatment patterns associated with myocardial infarction. The findings indicate that myocardial infarction predominantly affects middle-aged and elderly individuals, with the highest prevalence observed among patients aged 60–80 years. A considerable number of cases were also reported in the 40–60 years age group, suggesting that the burden of the disease extends beyond the elderly population. The increasing occurrence of myocardial infarction among younger adults may be associated with lifestyle-related factors such as smoking, physical inactivity, stress, obesity, and unhealthy dietary habits.

Gender-wise analysis demonstrated a clear male predominance, with males accounting for 68.43% of cases and females representing 31.57%. This higher prevalence among males may be attributed to greater exposure to cardiovascular risk factors, including smoking, alcohol consumption, and occupational stress. However, the risk among females tends to increase after menopause due to the decline in the cardioprotective effects of estrogen.

The analysis also highlighted the significant role of comorbidities in the development and progression of myocardial infarction. Hypertension, diabetes mellitus, dyslipidemia, and chronic kidney disease were the most frequently reported associated conditions. These comorbidities contribute to endothelial dysfunction, atherosclerotic plaque formation, and impaired cardiovascular health, thereby increasing the risk of myocardial infarction and its complications.

Treatment patterns across the reviewed studies revealed that antiplatelet agents, statins, beta-blockers, and ACE inhibitors remain the cornerstone of pharmacological management. In addition, interventional procedures such as percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG) were commonly employed in patients requiring advanced treatment. Early diagnosis and timely intervention were consistently associated with improved outcomes and reduced mortality.

Overall, the findings of this meta-analysis reinforce the substantial global burden of myocardial infarction and emphasize the importance of risk-factor modification, early detection, and evidence-based treatment strategies. Public health initiatives focusing on lifestyle modification, regular

screening, and effective management of comorbidities may contribute significantly to reducing the incidence and complications of myocardial infarction.

CONCLUSION:

This meta-analysis highlights that myocardial infarction remains a major global health burden, predominantly affecting middle-aged and elderly individuals, with a significantly higher prevalence among males. Hypertension, diabetes mellitus, dyslipidemia, chronic kidney disease, and smoking were identified as the most common risk factors and comorbidities associated with the disease. The findings emphasize the critical role of early diagnosis, effective management of cardiovascular risk factors, and timely therapeutic interventions in improving patient outcomes. Pharmacological therapies, including antiplatelet agents, statins, beta-blockers, and ACE inhibitors, along with revascularization procedures such as PCI and CABG, remain essential components of treatment. Overall, increasing public awareness, promoting healthy lifestyle practices, and implementing evidence-based preventive and management strategies are crucial for reducing the incidence, complications, and mortality associated with myocardial infarction.

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