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Risk Factors of Myocardial Infarction among Patients Admitted to Al-Wahdah Teaching Hospital, Yemen

Mohammed Haider Aldholae*, Mohammed Ali Al-Huthi, Dhiafullah Jayed Abdulsalam Mohammed Almkdad

Faculty of Medicine, Thamar University, Dhamar, Yemen
Email: *mhaldholae@gmail.com

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Abstract

Background: Coronary artery diseases (CAD) are one of the most important health problems in the world. The necessity of examining effective factors and exploring risk factors on CAD must be one of the most important health priorities in many countries like Yemen. **Aim:** This study aimed 1) To explore the risk factors of myocardial infarction among patients who admitted to Alwahdah Teaching Hospital, Ma'bar City, Thamar governorate, Yemen. 2) To detect the most important risk factors of MI among these patients in our study field. **Methods:** Our retrospective study started in the beginnings of year 2019. The research methods include collecting data of patients from their personal profiles in males & females ward from the beginning of January 2015 to June 2018. Collected data were written in standard tables & analyzed using SPSS program. **Results:** Our study showed that the commonest myocardial infarction patients males 70% in 60 years and above. The commonest risk factor was hypertension 49% followed by stressful jobs, diabetes history, smoking chewing Q at 37%, 28%, 26%, 23% respectively, other risk factors 9% dyslipidemia, 7% with family history of MI, 5% with history of vascular disease, and 4% with family history of chronic disease. The majority of people who referred had MI (93%) have STEMI. **Conclusion:** The commonest risk factor of MI was Hypertension and STEMI was the commonest type of MI in our study.

Subject Areas

Cardiology

Keywords

Acute Myocardial Infarction, Risk Factors, Yemen

1. Introduction

Myocardial infarction (MI) (*i.e.* heart attack) is the irreversible death (necrosis) of heart muscle secondary to prolonged lack of oxygen supply (ischemia). Atherosclerosis is the disease primarily responsible for most acute coronary syndrome (ACS) cases. Approximately 90% of myocardial infarctions (MIs) result from an acute thrombus that obstructs an atherosclerotic coronary artery. Plaque rupture and erosion are considered to be the major triggers for coronary thrombosis. Following plaque erosion or rupture, platelet activation and aggregation, coagulation pathway activation, and endothelial vasoconstriction occur, leading to coronary thrombosis and occlusion.

Coronary artery disease (CAD) is the leading cause of death in the United States; approximately 500,000 - 700,000 deaths related to CAD occur each year, making it the cause of death in an estimated one third of all deaths in the population for those older than 35 years. Approximately 1.5 million cases of myocardial infarction (MI) occur annually in the United States; the yearly incidence rate is approximately 600 cases per 100,000 people. The proportion of patients diagnosed with non-ST-elevation MI (NSTEMI) compared with ST-elevation MI (STEMI) has progressively increased. Despite an impressive decline in age-adjusted death rates attributable to acute MI since the mid-1970s, the total number of MI-related deaths in the United States has not declined [1].

Ischemic heart disease is the 1st leading cause of death worldwide according to WHO published in 24 May 2018 in its official website [2].

Coronary heart disease (CHD) is the leading cause of death among women in developed and developing countries [3]. The incidence of CHD is markedly lower among women than men prior to the age of 50 years after which time CHD increases and approaches that seen among men by the eighth decade [4] [5]. CAD is also the number one cause of death in European countries. In the European Union, death rates related to CAD dropped by almost 30% between the mid 1960s to the mid and late 1990s; however, within Eastern European countries, there was an increase in death rates related to acute MI in the early 1990s, followed by a subsequent decline. In the Russian Federation, cardiovascular mortality remained the same [6].

Cardiovascular disease in other developed countries and in developing nations: An analysis of death certificates from the World Health Organization (WHO) database demonstrated that CAD mortality in Japan was significantly lower than in the United States and Europe, and it was further reduced by about 30% by the mid 1990s. In China, there has been a significant increase in mortality related to CAD, this is most likely attributed to the increase in cardiovascular disease risk factors, predominantly smoking and dyslipidemia [7].

The incidence of CAD and related mortality expected to rise dramatically in other developing countries including India, Latin America, the Middle East and Sub-Saharan Africa, with an estimated 80% increase, from approximately 9 mil-

lion in 1990 to a projected 20 million by 2020 [8] [9].

Objectives

This study aims:

1) To explore the risk factors of myocardial infarction among patients who admitted to Al-Wahdah Teaching Hospital, Ma'bar City, Thamar governorate, Yemen.

2) To detect the most important risk factors of MI among these patients in our study field.

2. Literature Review

2.1. Background

The heart is a muscular organ enclosed in protective fibrous sac (the pericardium), a fibrous layer is also closely affixed to the heart called the epicardium.

The wall of the heart is composed primarily of cardiac muscle cells, the myocardium.

The inner surface of the cardiac chambers as well as the inner wall of all blood vessels is lined by a thin layer of cells known as endothelium.

Cardiac muscle:

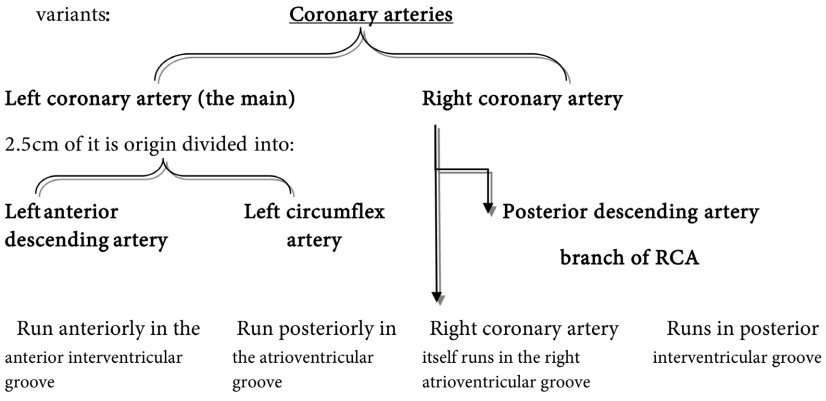
It is specialized muscle cells with amazing resiliency and stamina arranged in layers that are tightly bound together and completely encircle the blood filled function when the walls of the walls of a chamber contract they come together like squeezing fist and exert pressure on the blood they enclose.

Every heart cell contracts with every beat of the heart. The human heart has a limited ability to replace it is muscle cells. Recent experiments suggest that only about 1% of heart muscle cells are replaced per year.

The blood being pumped through the heart chambers does not exchange nutrients metabolic end products with the myocardial cells. They like the cells of all other organs receive their blood supply via arteries that branch from the aorta called coronary arteries [1].

Coronary circulation:

Coronary anatomy varies greatly from person to person There are many normal



Gives branches to supply:

1. The anterior part of the septum.

2.the anterior, lateral and apicawalls of the left ventricle.

Gives marginal branches to supply:lateral posterior and inferior segment of the left ventricle.

Giving branches that supply:1.right atrium.

2.right ventricle.

3.inferoposterior aspects of the left ventricle.

RCA supplies SAN in about60%

And AVN in about 90%

Supplies the inferior part of the interventricular septum

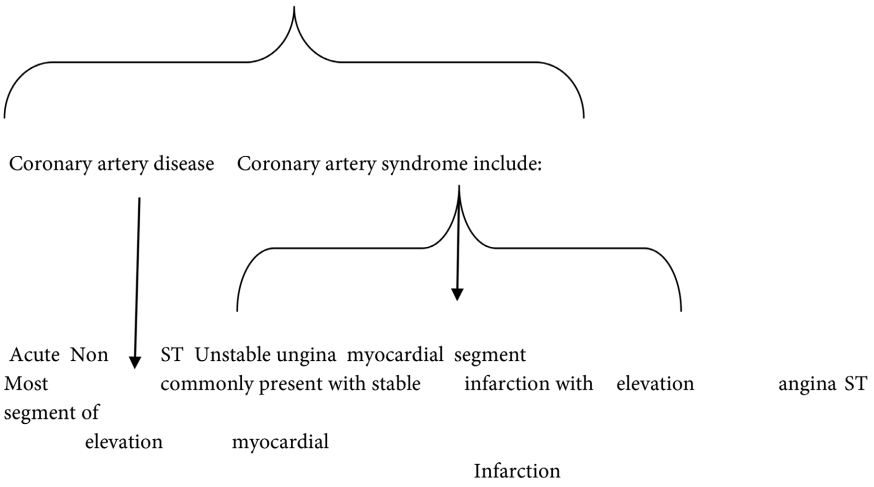
Occlusion of left main coronary artery is usually fatal.

Proximal occlusion in RCA result in:

*Sinus bradycardia

*abrupt occlusion in RCA due to thrombus result in: infarction of inferior part of the left ventricle and often right ventricle.

***Patients with ischemic heart disease fall into two large groups** [10]



2.2. Definition of MI

Myocardial infarction or Heart attack: Under group acute coronary syndrome defined as acute myocardial ischemia with evidence of myocardial necrosis of an area of myocardium occurring as a result of critical imbalance between coronary blood supply and myocardial demand.

The Third Universal Definition of myocardial infarction (MI) expert consensus document was published in October 2012 by the global Myocardial Infarction Task Force so:

An myocardial infarction is defined by a typical rise and or fall of cardiac troponin (cTn), With at least one value above the assays upper reference limit (URL) accompanied by at least one of the other feature of ischemia e.g. typical

symptoms or ECG [1] [2].

2.3. The Third Universal Classification of Myocardial Infarction

Type 1: Spontaneous MI

Spontaneous MI due to atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis. The patient may have underlying severe CAD, non-obstructive coronary disease or no CAD.

Type 2: MI secondary to an ischemic imbalance

Myocardial injury with necrosis occurs due to conditions other than CAD that contribute to an imbalance between myocardial oxygen supply and/or demand such as coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachycardia-bradycardia arrhythmias, anemia, respiratory failure, hypotension, and hypertension.

Type 3: MI resulting in death when biomarker values are unavailable

Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic ECG changes or new LBBB, but death occurs before blood samples can be obtained, before cardiac troponins biomarkers rise, or when cardiac biomarkers were not collected.

Type 4A: MI related to percutaneous coronary intervention

MI associated with PCI is defined by elevation of cTn values greater than five times the 99th percentile upper normal reference limit (URL) in patients with normal baseline values (<99th percentile URL) or a rise of cTn values by >20% if the baseline troponins are elevated and are stable or falling. In addition one of the following criterion are required: 1) symptoms suggestive of myocardial ischemia; 2) new ischemic ECG changes or new LBBB; 3) angiographic loss of patency of a major coronary artery or a side branch or persistent slow- or no coronary flow or coronary embolization; or 4) demonstration with imaging of a new loss of viable myocardium or new regional wall motion abnormality.

Type 4B: MI related to stent thrombosis

MI associated with stent thrombosis detected by coronary angiography or autopsy in the presence of myocardial ischemia with a rise and/or fall of troponin biomarkers. One troponin measurement should be above the 99th percentile UR.

Type 4C: MI related to restenosis

MI associated with restenosis defined as ≥50% stenosis or a complex lesion demonstrated at coronary angiography after initial successful stent.

Deployment or dilatation of a coronary artery stenos is with balloon angioplasty. These coronary angiographic changes should be associated with an increase and/or decrease of cTn values > 99th percentile URL and no other significant obstructive CAD.

Type 5: MI related to coronary artery bypass grafting

MI associated with CABG is defined by elevation of cardiac troponins greater than ten times the 99th percentile URL in patients with normal baseline cTn values (<99th percentile URL). In addition, one of the following should be present: new pathological Q waves or new LBBB; or angiographic documented new graft or new native coronary artery occlusion or new loss of viable myocardium or new regional wall motion abnormality as shown by an imaging modality [10].

2.4. Pathology of Myocardial Infarction

Myocardial infarction is defined by pathology as myocardial cell death due to prolonged ischemia. Cell death categorized pathologically as coagulation and/or contraction band necrosis, which usually evolves through oncosis but can result to a lesser degree from apoptosis. Careful analysis of histological sections by an after the onset of myocardial ischemia, cell death is not immediate but takes a finite period to develop (as little as 20 min or less in some animal models). It takes several hours before myocardial necrosis can be identified by macroscopic or microscopic post-mortem examination. Complete necrosis of all myocardial cells at risk requires at least 2 - 4 h or longer depending on the presence of collateral circulation to the ischemic zone persistent or intermittent coronary arterial occlusion the sensitivity of the myocytes to ischemia, pre-conditioning, and/or finally individual demand for myocardial oxygen and nutrients. Myocardial infarctions are usually classified by size: microscopic (focal necrosis), small (10% of the left ventricular [LV] myocardium), moderate (10% - 30% of the LV myocardium) and large (30% of the LV myocardium), and by location. The pathological identification of myocardial necrosis is made without reference to morphological changes in the coronary arterial tree or to the clinical history [3]. The vast majority of MIs are caused by acute coronary artery thrombosis. In most instances disruption of preexisting atherosclerotic plaque serves, as the nidus for thrombus generation, vascular occlusion, and subsequent transmural infarction of the downstream myocardium. In 10% of MIs, however transmural infarction occurs in the absence of occlusive atherosclerotic vascular disease such infarcts are mostly attributable to coronary artery vasospasm or to embolization from mural thrombi [4].

Myocardial Response to Ischemia: Loss of the myocardial blood supply leads to profound functional, biochemical and morphologic consequences. Within seconds of vascular obstruction, aerobic glycolysis ceases, leading to a drop in adenosine triphosphate (ATP) and accumulation of potentially noxious metabolites (e.g., lactic acid) in the cardiac myocytes. The **functional** consequence is a rapid loss of contractility, which occurs within a minute or so of the onset of ischemia. These early changes are potentially **reversible.** Only severe ischemia lasting at least 20 to 40 minutes causes **irreversible** damage and myocyte death leading to coagulation necrosis [5].

The microscopic appearance also undergoes a characteristic sequence of

changes and typical features of coagulative necrosis become detectable within 4 to 12 hours of infarction. "Wavyfibers" also can be present at the edges of an infarct; these reflect the stretching and buckling of noncontractile dead fibers. Sublethal ischemia can also induce intracellular **myocyte vacuolization;** such myocytes are viable but frequently are poorly contractile (see Table 1 and Figure 1).

Irreversible Injury

central yellow-tan softening necrosis; pyknosis of nuclei; myocytehypereosinophilia; marginal contraction band necrosis; beginning neutrophilic infiltrate Coagulation necrosis, with loss of nuclei and striations; interstitial infiltrate of neutrophils Beginning disintegration of dead myofibers, with dying neutrophils; early phagocytosis of dead cells by macrophages at infarct border 01. In a typical MI, the following sequence of events transpires: There is a sudden disruption of an atheromatous plaque-for example, intraplaque hemorrhage, erosion or ulceration, or rupture or fissuring-exposing subendothelial collagen and necrotic plaque contents.

Platelets adhere, aggregate, become activated, and release potent secondary aggregators including thromboxane A_2, adenosine diphosphate, and serotonin.

Vasospasm is stimulated by platelet aggregation and mediator release.

Other mediators activate the extrinsic pathway of coagulation, adding to the bulk of the thrombus. Within minutes the thrombus can evolve to completely occlude the coronary lumen of the coronary vessel.

Table 1. Timing of histopathological changes of MI.

Time	Gross Features	Light Microscopic Findings	Electron Microscopic Findings
Reversible Injury		central yellow-tan softening necrosis	
0 - ½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
½ - 4 hr	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
4 - 12 hr	Occasionally dark mottling	Beginning coagulation necrosis; edema; hemorrhage	
12 - 24 hr	Dark mottling	Ongoing coagulation	
1 - 3 days	Mottling with yellowtan infarct center	central yellow-tan softening necrosis; pyknosis of nuclei; myocytehypereosinophilia; marginal contraction band necrosis; beginning neutrophilic infiltrate Coagulation necrosis, with loss of nuclei and striations; interstitial	
3 - 7 days	Hyperemic border	infiltrate of neutrophils Beginning disintegration of dead myofibers, with dying neutrophils; early phagocytosis of dead cells by macrophages at infarct border	

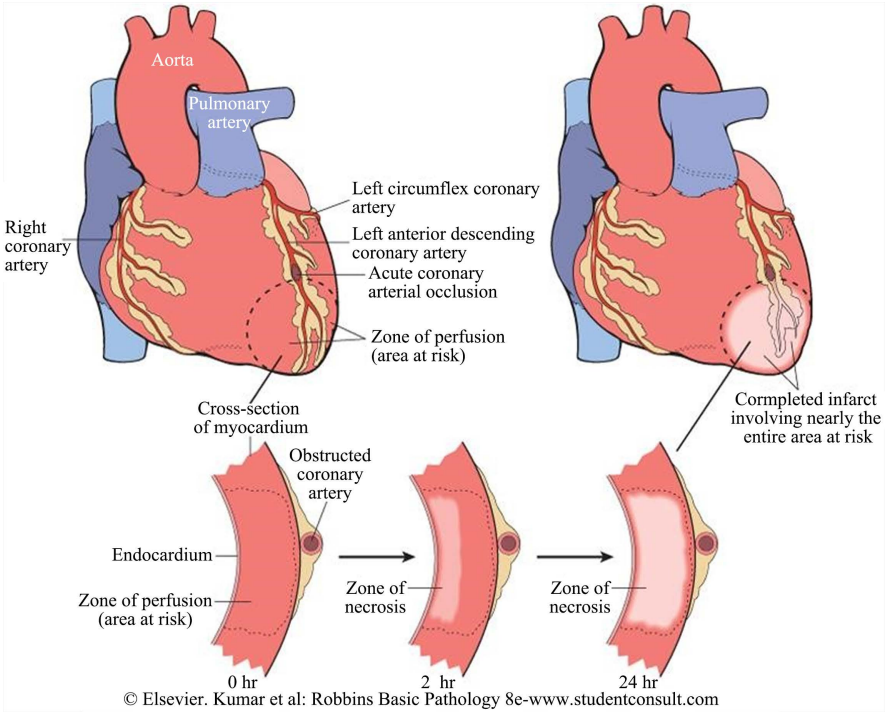


Figure 1. Progression of myocardial necrosis after coronary artery occlusion. Necrosis begins in a small zone of the myocardium beneath the endocardial surface in the center of the ischemic zone. This entire region of myocardium depends on the occluded vessel for perfusion and is the area at risk, Note that a very narrow zone of myocardium immediately beneath the endocardium is spared from necrosis because it can be oxygenated by diffusion from the ventricle. The end result of obstruction to blood flow is necrosis of the muscle that was dependent on perfusion from the coronary artery obstructed, Nearly the entire area at risk loses viability.

2.5. Clinical Manifestations of Myocardial Infarction

While the classic symptoms of a heart attack are chest pain and shortness of breath, the symptoms can be quite varied. The most common symptoms of a heart attack include:

pressure or tightness in the chest, pain in the chest back, jaw and other area of the upper body that lasts more than few minutes or that goes away and come back shortness of breath, sweating, nausea, vomiting, anxiety, cough, dizziness & tachycardia. It is important to note that not all people who have heart attacks experience the same symptoms or the same severity of symptoms. Chest pain is the most commonly reported symptom among both women and men. However, women are more likely than men to have shortness of breath, jaw pain, upper back pain, lightheadedness, nausea & vomiting. In fact some women who have had a heart attack report that their symptoms felt like the symptoms of the flu [6].

2.6. Risk Factors of MI

2.6.1. Non-Modifiable Risk Factors for Atherosclerosis Include the Following

1-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.

2-Sex: 3-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.

4-Male-pattern baldness: some researches provides support for the hypothesis that vertex pattern baldness is a marker for increased risk of CHD events.

2.6.2. Modifiable Risk Factors for Atherosclerosis Include the Following

1) Smoking or other types of tobacco use,

2) Hypercholesterolemia and hypertriglyceridemia, including inherited lipoprotein disorders, dyslipidemia: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.

3) Diabetes mellitus: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.

4) 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. 5-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.

6) Psychosocial stress

7) 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.

8) Reduced consumption of fruits and vegetables, poor oral hygiene

9) Type A personality, elevated homocysteine levels, presence of peripheral vascular disease [7].

2.7. Causes of Myocardial Infarction

A) Atherosclerosis is the disease primarily responsible for most acute coronary syndrome (ACS) cases. Approximately 90% of myocardial infarctions (MIs) result from an acute thrombus that obstructs an atherosclerotic coronary artery. Plaque rupture and erosion are considered to be the major triggers for coronary thrombosis. Following plaque erosion or rupture, platelet activation and aggregation, coagulation pathway activation, and endothelial vasoconstriction occur, leading to coronary thrombosis and occlusion. Within the coronary vasculature, flow dynamics and endothelial shear stress are implicated in the pathogenesis of vulnerable plaque formation [7]. A large body of evidence indicates that in numerous cases, culprit lesions are stenoses of less than 70% and are located proximally within the coronary tree [8] [9]. Coronary atherosclerosis is especial-

ly prominent near branching points of vessels. Culprit lesions that are particularly prone to rupture are atheromascontaining abundant macrophages, a large lipid-rich core surrounded by a thinned fibrous cap [11] (See **Table 2**).

B) MI can also occur for causes other than atherosclerosis (Nonatherosclerotic) causes of MI include the following:

1) Coronary occlusion secondary to vasculitis; new researches shows that inflammation also plays a role in the evolution of heart attacks. It appears that the coronary artery walls become inflamed over time, further increasing the buildup of fatty plaques.

2) Ventricular hypertrophy (e.g. left ventricular hypertrophy, hypertrophic cardiomyopathy).

3) Coronary artery emboli, secondary to cholesterol, air, or the products of sepsis.

4) Coronary trauma.

5) Primary coronary vasospasm (variant angina); heart attacks may also be caused by coronary artery spasm, where a heart artery is temporarily constricted, using tobacco and illicit drugs, such as cocaine, can cause a life-threatening spasm.

6) Drug use (e.g. cocaine, amphetamines, ephedrine).

7) Arteritis.

8) Coronary anomalies, including aneurysms of coronary arteries.

9) Factors that increase oxygen requirement, such as heavy exertion, fever, or hyperthyroidism.

10) Factors that decrease oxygen delivery, such as hypoxemia of severe anemia.

11) Aortic dissection, with retrograde involvement of the coronary arteries.

12) Respiratory infections, particularly influenza [12] [13].

C) In addition, MI can result from hypoxia due to carbon monoxide poisoning or acute pulmonary disorders.

D) Although rare, pediatric coronary artery disease may be seen with Marfan syndrome, Kawasaki disease, Takayasu arteritis, progeria, and cystic medial necrosis. Acute MI is rare in childhood and adolescence. Although adults acquire coronary artery disease from lifelong deposition of atheroma and plaque, which causes coronary artery spasm and thrombosis, children with acute MI usually have either an acute inflammatory condition of the coronary arteries or an anomalous origin of the left coronary artery.

Intrauterine MI also does occur, often in association with coronary artery stenosis [14].

2.8. Diagnosis of MI

In 1971, WHO proposed that the diagnosis of myocardial infarction required the presence of at least 2 of the following:

1) Typical symptoms.

2) Typical ECG pattern involving the development of Q waves.

3) An initial rise and subsequent fall in serum or plasma Biomarkers of myocardial necrosis [15].

1-History and physical Examination: Note that up to 30% of MI are recognized or silent due to Atypical symptoms more common in women, DM, elderly, post Heart transplant because of denervation [16]. *The first step is to determine if the pain is truly cardiac in nature [17]. The assessment of acute chest pain depends on analysis of character of the pain or collapse and it is associated features. If the history of chest pain is strongly consistent with ischemia, it is more important than EKG alone (See **Table 3**).

2-ECG: ECG is critical if the patient was admitted for chest pain. It is central to confirming the diagnosis but may be difficult to interpret if there is BBB or previous MI. the initial ECG may be normal or normal diagnostic in one third of cases. Repeated or serial ECG are important especially if the diagnosis is uncertain or the or if the patient has recurrent or persistent symptoms.

3-plasma cardiac biomarkers: Cardiac markers or cardiac enzymes are proteins that leak out of injured myocardial cells through their damaged cell membranes into blood stream. the third universal definition of myocardialinfarction recommends that patients who are suspected on clinical grounds of having ACS should undergo serial sampling for cardiac troponin (cTn). Now, the markers most widely used in detection of myocardial infarction are CK-MB and cTn T and I, as they are more specific for myocardial injury. Troponin T and I have nearly complete tissue specificity myocardial damage [18].

Other serum cardiac markers:

Until 1980, enzymes AST or SGOT and LDH were used to assess cardiac injury. May be from damaged red blood cells, kidney, liver and lung, usually not performed now except in small centers [3].

#Other blood test: CBC (polymorphoneuclear leukocytosis), ESR: raised chest X ray: may demonstrate pulmonary edema. Heart size is usually normal. Enlarged cardiac shadow indicates preexisting myocardial damage [2] [3].

Echocardiography:

May be performed in equivocal cases. Useful for assessing ventricular function and for detecting important complication [2].

Coronary angiogram:

Allows visualization of narrowing or obstruction on heart vessels.

It is performed in difficult cases or in situation where the intervention to restore blood flow is appropriate .Catheter is inserted into an artery (usually the femoral artery) and pushed to the vessels supplying the heart radio opaque dye is administered through the catheter and a sequence of X ray (fluoroscopy is performed.

Nuclear medicine:

Can be used in stable patient whose symptoms have resolved by the time of evaluation as technetium 99 mtcsestamibi, thallium 201 chloride or rubidium 82 chloride to visualize areas of reduced blood flow in conjunction with physiologic or pharmacologic stress [8] [9].

Table 2. Algorithm for risk stratification [11].

Clinical	STEMI	NSTEMI angina	Unstable	Stable angina	Non cardiac chest pain
ECG	Agonizing pain	Rest pain, DM, post MI, prior ASA		Exertional pain	Atypical pain
Cardiac markers	ST elevation	ST, T wave changes		Negative	Negative
Risk assessment	Positive	Positive High risk 11		Negative	Negative
	STEMI			Low risk	Low probability

Table 3. Summary for cardiac biochemical markers and its significance [17].

Test	Details
Troponin	Begins to rise at 3 - 4 hours Maximum sensitivity at 12 - 18 hours Stays positive 1 - 2 weeks after event
CK-MB	Begins to rise at 3 - 4 hours Maximum sensitivity at 12 - 18 hours CK-MB lasts 1 - 2 days
Myoglobin	Rises at 1 - 4 hours
Catheterizati on	Clear history and abnormal EKG needs evaluation for angiography
BNP	Use when etiology of dyspnea not clear
Stress test	Use when history and EKG are not clear
Echocardiogr am	Looks at wall and valve motion Ejection fraction
Telemetry	Continuous EKG monitor

Significance

- Negative first testexcludes nothing
- Positive test suggests Ml
- False positive with CHF and renal failure
- Cannot detect re infarction in last week
- Negative first test excludes nothing
- Positive test suggests Ml
- Best test for detecting re infarction
- Lacks specificity
- Negative test at 4 hours excludes Ml
- Positive test is useless
- Continued pain with maximal medical therapy needs angiography
- Normal BNP excludes CHF
- Abnormal test is Nonspecific
- Reversible ischemia is the main thing you are looking for
- Catheterize abnormal
- Normal wall motion excludes Ml
- High troponin with normal wall motion is a false-positive troponin
- All ACS patients need telemetry

2.9. Treatment of MI

Pre-hospital care: oxygen, aspirin, nitrates, and triage to an appropriate medical center (see Figure 2).

Drug treatment: Anti-platelet drugs, anti-anginal drugs, anticoagulants, and in some cases other drugs.

Reperfusion therapy: Fibrinolytics or angiography with percutaneous coronary intervention or coronary artery bypass surgery.

Postdischarge rehabilitation and chronic medical management of coronary artery disease. Choice of drug therapy and choice of reperfusion strategy are discussed elsewhere.

Pre-hospital care: Oxygen, aspirin, nitrates & triage to appropriate medical center. A reliable IV route must be established, oxygen given (typically 2 L by nasal cannula), and continuous single-lead ECG monitoring started. Prehospital interventions by emergency medical personnel including ECG.

Hospital admission: Risk-stratify patient and choose reperfusion strategy. Drug therapy with anti-platelet drugs, anticoagulants and other drugs based on reperfusion strategy. On arrival to the emergency room, the patient's diagnosis is confirmed. Drug therapy and timing of revascularization depend on the clinical picture and diagnosis. For STEMI, reperfusion strategy can include fibrinolytic therapy or immediate PCI. For patients with NSTEMI, angiography may be done within 24 to 48 h of admission if the patient is clinically stable. If the patient is unstable (e.g. ongoing symptoms, hypotension or sustained arrhythmias), then angiography must be done immediately [19].

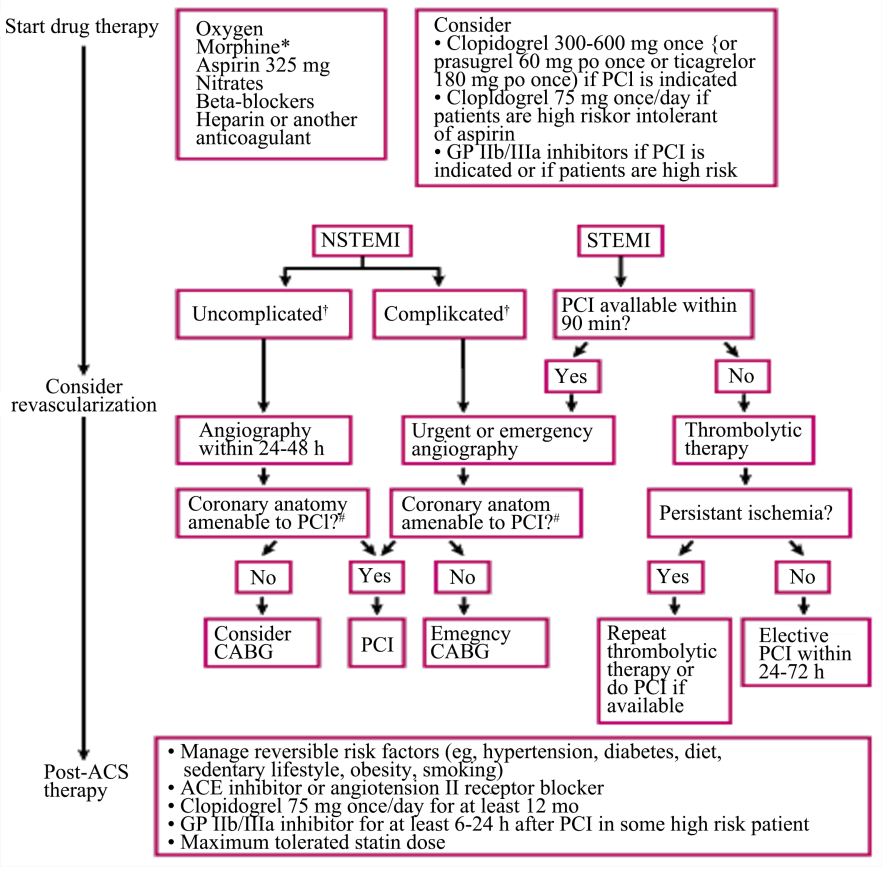


Figure 2. Approach to myocardial infarction.

Morphine should be used judiciously (e.g. if nitroglycerin is contraindicated or if the patient has symptoms despite nitroglycerin therapy).

Complicated means that the hospital course was complicated by recurrent angina or infarction, heart failure, or sustained recurrent ventricular arrhythmias. Absence of any of these events is termed uncomplicated.

Drug treatment of acute myocardial infarction

All patients should be given anti platelet drugs, anticoagulants, and if chest pain is present, anti-anginal drugs. The specific drugs used depend on the reperfusion strategy and other factors; their selection and use is discussed in drugs for Acute Coronary Syndrome. Other drugs, such as beta-blockers, ACE inhibitors, and statins, should be initiated during admission.

Patients with acute myocardial infarction should be given the following (unless contraindicated):

- Anti-platelet drugs: Aspirin, clopidogrel, or both (prasugrel or ticagrelor are alternatives to clopidogrel).
- Anticoagulants: A heparin (unfractionated or low molecular weight heparin) or bivalirudin.
- Glycoprotein IIb/IIIa inhibitor for some high risk patients.
- Antianginal therapy usually nitroglycerin.
- Beta-blockers.
- ACE inhibitor.
- Statin.

All patients are given aspirin 160 to 325 mg (not enteric-coated), if not contraindicated, at presentation and 81 mg once/day indefinitely thereafter. Chewing the first dose before swallowing quickens absorption. Aspirin reduces short-term and long-term mortality risk. In patients undergoing PCI, a loading dose of clopidogrel (300 to 600 mg po once), prasugrel (60 mg po once), or ticagrelor (180 mg po once) improves outcomes, particularly when administered 24 h in advance. For urgent PCI, prasugrel and ticagrelor are more rapid in onset and may be preferred. Either a low molecular weight heparin (LMWH), unfractionated heparin, or bivalirudin is given routinely to patients unless contraindicated (e.g. by active bleeding).

Unfractionated heparin is more complicated to use because it requires frequent (q 6 h) dosing adjustments to achieve target activated PTT (aPTT). The LMWHs have better bioavailability, are given by simple weight-based dose without monitoring aPTT and dose titration, and have lower risk of heparin-induced thrombocytopenia. Bivalirudin is recommended for patients with a known or suspected history of heparin-induced thrombocytopenia. Anticoagulants are continued for:

- Duration of PCI in patients undergoing this procedure.
- Duration of hospital stay (in patients on LMWH) or 48 h (in patients on unfractionated heparin) in all other cases.

Consider a glycoprotein IIb/IIIa inhibitor for high-risk patients (patients with recurrent ischemia, dynamic ECG changes, or hemodynamic instability). Ab-

ciximab, tirofiban, and eptifibatide appear to have equivalent efficacy, and the choice of drug should depend on other factors (e.g., cost, availability, familiarity). This agent is continued for 6 to 24 h.

Chest pain can be treated with nitroglycerin or sometimes morphine. Nitroglycerin is preferable to morphine, which should be used judiciously (e.g., if a patient has a contraindication to nitroglycerin or is in pain despite nitroglycerin therapy). Nitroglycerin is initially given sublingually, followed by continuous IV drip if needed. Morphine 2 to 4 mg IV, repeated q 15 min as needed, is highly effective but can depress respiration, can reduce myocardial contractility, and is a potent venous vasodilator. Evidence also suggests that morphine use interferes with some P2Y12 receptor inhibitors. A large retrospective trial showed that morphine may increase mortality in patients with acute myocardial infarction.

Hypotension and bradycardia secondary to morphine can usually be overcome by prompt elevation of the lower extremities.

Standard therapy for all patients with unstable angina includes beta-blockers, ACE inhibitors, and statins. Beta-blockers are recommended unless contraindicated (e.g. by bradycardia, heart block, hypotension, or asthma), especially for highrisk patients. Beta-blockers reduce heart rate, arterial pressure, and contractility, thereby reducing cardiac workload and oxygen demand. ACE inhibitors may provide long-term cardioprotection by improving endothelial function. If an ACE inhibitor is not tolerated because of cough or rash (but not angioedema or renal dysfunction), an angiotensin II receptor blocker may be substituted. Statins are also standard therapy regardless of lipid levels and should be continued indefinitely.

Reperfusion therapy in acute myocardial infarction

For patients with STEMI: Immediate percutaneous coronary intervention or fibrinolytics. For patients with NSTEMI: Immediate percutaneous coronary intervention for unstable patients or within 24 to 48 h for stable patients. For STEMI patients, emergency PCI is the preferred treatment of ST-segment elevation myocardial infarction when available in a timely fashion (door to balloon-inflation time < 90 min) by an experienced operator. If there is likely to be a significant delay in availability of PCI, thrombolysis should be done for STEMI patients meeting criteria. Reperfusion using fibrinolytics is most effective if given in the first few minutes to hours after onset of myocardial infarction. The earlier a fibrinolytic is begun, the better. The goal is a door-toneedle time of 30 to 60 min. Greatest benefit occurs within 3 h, but the drugs may be effective up to 12 h. Characteristics and selection of fibrinolytic drugs are discussed elsewhere.

Unstable NSTEMI patients (*i.e.* those with ongoing symptoms, hypotension, or sustained arrhythmias) should proceed directly to the cardiac catheterization laboratory to identify coronary lesions requiring PCI or CABG.

For **uncomplicated NSTEMI patients**, immediate reperfusion is not as urgent because a completely occluded infarct-related artery at presentation is uncommon. Such patients typically undergo angiography within the first 24 to 48 h of hospitalization to identify coronary lesions requiring PCI or CABG. Fibrino-

lytics are not indicated for any NSTEMI patients. Risk outweighs potential benefit. Choice of reperfusion strategy is further discussed in Revascularization for Acute Coronary Syndromes.

Rehabilitation and post-discharge treatment

1-Functional evaluation.

2-Changes in lifestyle: Regular exercise, diet modification, weight loss, smoking cessation.

3-Drugs: Continuation of antiplatelet drugs, beta-blockers, ACE inhibitors, and statins.

Patients who did not have coronary angiography during admission, have no high-risk features (e.g., heart failure, recurrent angina, ventricular tachycardia or ventricular fibrillation after 24 h, mechanical complications such as new murmurs, shock), and have an ejection fraction > 40% whether or not they received fibrinolytics usually should have stress testing of some sort before or shortly after discharge.

The acute illness and treatment of myocardial infarction should be used to strongly motivate the patient to modify risk factors. Evaluating the patient's physical and emotional status and discussing them with the patient, advising about lifestyle (e.g. smoking, diet, work and play habits, exercise), and aggressively managing risk factors may improve prognosis.

On discharge, all patients should be on appropriate anti-platelet drugs, statins, anti-anginals, and other drugs based on comorbidities [20]. Chewed aspirin [325 mg], and pain management with nitrates can reduce risk of mortality and complications. Early diagnostic data and response to treatment can help determine the need for and timing of revascularization.

2.10. Prevention of MI

Urgency for health care policy and research encompasses this contemporary concept:

1) Cardiac rehabilitation

2) Secondary prevention services are comprehensive, long term programs involving: medical evaluation, exercise, cardiac risk modification, education, counseling, this program are designed to limit:

1-The physiological and psychological effect of cardiac illness.

2-Reduce the risk factor for sudden death or re infraction.

3-Control cardiac symptoms.

4-stabilize or reverse the atherosclerotic process.

5-enhance the psych-social and vocational status of selected patient [15]. Although lack the power to change such as family history, sex, age, there are some key heart diseases prevention steps:

1-Smoking cessation significantly reduces the risk of dying from tobacco related disease (26) within 1 years coronary artery disease is cute in half [2].

2-Exercises for about 30 minutes on most day of the week physical activity can reduce the risk of cardiovascular disease by control weight, reduces chances of

developing other condition that put strain on the heart such as high BP, high cholesterol land diabetes [21] [22].

3-Heart-healthy diet.

Two examples of heart healthy-food planes including:

1-dietary approaches to stop HTN (DASH) eating plane.

2-Mediterranean diet: diet rich in fruits, vegetables, whole grain, limited or avoid saturated fat and tans fat.

3-Manage of stress some people cope with stress in un healthy ways such as over eating, drinking or smoking so alternative ways to managed stress such as physical activity, relaxation, exercise [21] [22].

Secondary prevention drug therapy:

1-aspirin and clopidogrel:

- For most low risk patients low to medium dose aspirin 75 - 325 mg.
- reduce risk of future infarction and other vascular events by approximately 25%. if patient are intolerant of long term aspirin clopidogrel is suitable alternative [2] [23].

2-several trials have shown that long term treatment with ACEI can counteract:

3) Ventricular remodeling, reduce recurrent MI, Prevent the onset of heart failure, reduce re-hospitalization and improve survival so they should therefore be considered in all patients with ACS [2].

3-lipid lowering drugs such as statin:

Lowering cholesterol in people of high risk of ischemic heart disease [23].

4-coronary revascularization, for all patient at moderate or high risk.

It is associated & short & long term benefits including reduction in myocardial infarction &death.

5-implantable cardiac defibrillators: prevents sudden cardiac death in patients who have severe left ventricular impairment [2].

3. Subjects and Methods

3.1. Subjective

Scanning patients of both gender who were diagnosed and treated as a case of myocardial infarction were incorporated into this study.

3.2. Study Area

This study was conducted in Thamar governorate, Ma'bar City, Yemen, of AL-wahdah teaching hospital, Thamar University, Internal medicine Department.

3.3. Study Design

This study was retrospective conducted from January 2018 to June 2021.

3.4. Study Population

This study included 120 patients diagnosed as a case of MI and Subjected to

treatment in AL-wahdah teaching hospital.

20 cases were excluded due to incomplete data or empty file.

3.5. Ethics and Consents

Approval for this study was obtained from Thamar University Medical Ethics committee (TUMEC), in addition the approval was made by our supervisors.

3.6. Data Collection

This study was obtained from patients files who were diagnosed and treated as a cases of MI.

The related data was taken from each file and recorded in special Sheet designed for this purpose.

3.7. Statistical Analysis

Data were entered and analyzed by using statistical package for social sciences (SPSS).

4. Results

Our study includes 100 patients admitted to Alwahadah teaching hospital who were diagnosed and treated as a cases of myocardial infarction. Among 100 patients, 70% was males, The age distributed incidence rate of myocardial infarction among the study patients was 29%, 27%, 36%, 8% in the ages 40 - 49 years, 50 - 59 years, 60 - 79 years, 80 - 100 years, respectively (See Tables 4-12 and Figures 3-5).

Around (23%)cases had khat chewing and (13%) of cases tobacco smoking, 37 (37%) cases in stressful jobs, 49 (49%) of cases with hypertension, 28 (28%) cases with diabetes mellitus, 9 (9%) cases with dyslipidemia, 5 (5%) cases with vascular diseases and 7 (7%) cases had family history of myocardial infarction, also 4 (4%) cases with family history of chronic diseases, p. values were significant in DM equal 0.000, occupations equal 0.000 and chronic disease equal 0.013.

Table 4. Male to female ratio.

Sex	Number of patient	Percentage
Male	70	70%
Female	30	30%

Table 5. The frequencies and percentage of MI occurrence according to age.

Age of patient	**Number of patient**	**Percentage**
40 - 49	29	29%
50 - 59	27	27%
60 - 79	36	27%
80 - 100	8	8%

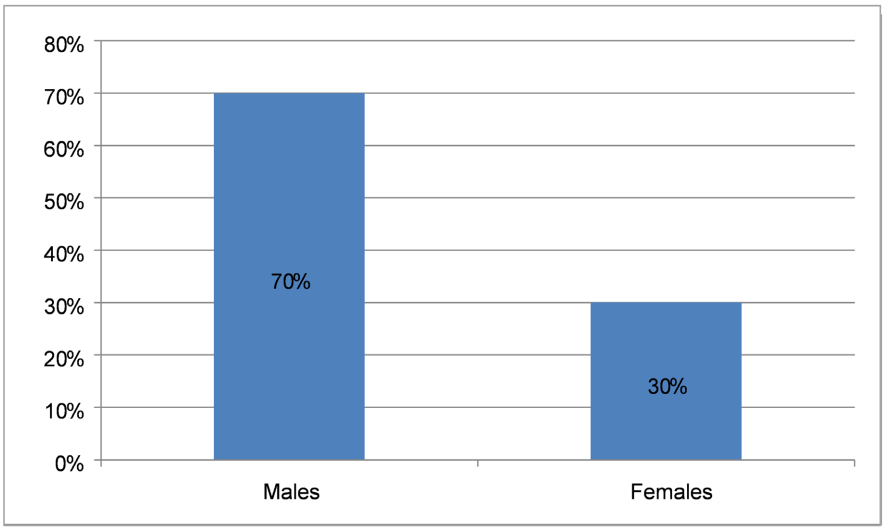


Figure 3. Ratio of MI incidence/Gender.

Table 6. The frequencies and percentages of occurrence according to risk factors of MI.

Risk factor	Number of patient	Percentage
History of HTN	49	49%
History of qat chewing	23	23%
Stressful jobs	37	37%
History of DM	28	28%
History of smoking	26	26%
Dyslipidemia	9	9&
History of MI	7	7%
Vascular disease	5	5%
Family history of chronic diseases	4	4%

Table 7. Frequencies and percentage for samples according to type of MI.

Type of MI	Number of patient	Percentage
STEMI	93	93%
NSTEMI	7	7%

Among 100 patients with myocardial infarction 93% were ST-segment elevation (STEMI) and 7% were non ST-segment elevation (non-STEMI).

Table 8. Incidence of MI/Years.

Date of admission	Number of patient	Percentage
2015	4	4%
2016	25	25%
2017	27	27%
2018	44	44%

Table 9. Explains the risk factors in males and females and percentages and their p. values for each risk factor.

	SEX					
	M		F		X^2	P.V
	N	%	N	%		
Qat showing	20	28.6	11	36.7	0.643	0.422
Tobacco Smoking	19	27.1	4	13.3	2.261	0.133
Water pipe	5	7,1	8	26.7	7.078	0.008
Marital status						
Married	70	100	30	100		
Occupation					70.868	0.000
Farmer	26	37.1	8	29.7		
Solider	3	4.3	0	0		
Unknown	41	58.6	0	0		
Dyslipidemia	4	5.7	5	16.7	3.076	0.076
Vascular disease	5	7.1	0	0	2.256	0.133
Family history of MI	6	8.6	1	3.3	0.885	0.347
Family history of chronic disease	2	2.9	2	6.7	0.794	0.373
ECG					6.152	0.013
STE	68	97.1	25	83.3		
NSTE	2	2.9	5	16.7		
Date					5.553	0135
2015	3	4.3	1	3.3		
2016	18	25.7	7	23.3		
2017	23	32.9	4	13.3		
2018	26	37.1	18	60		

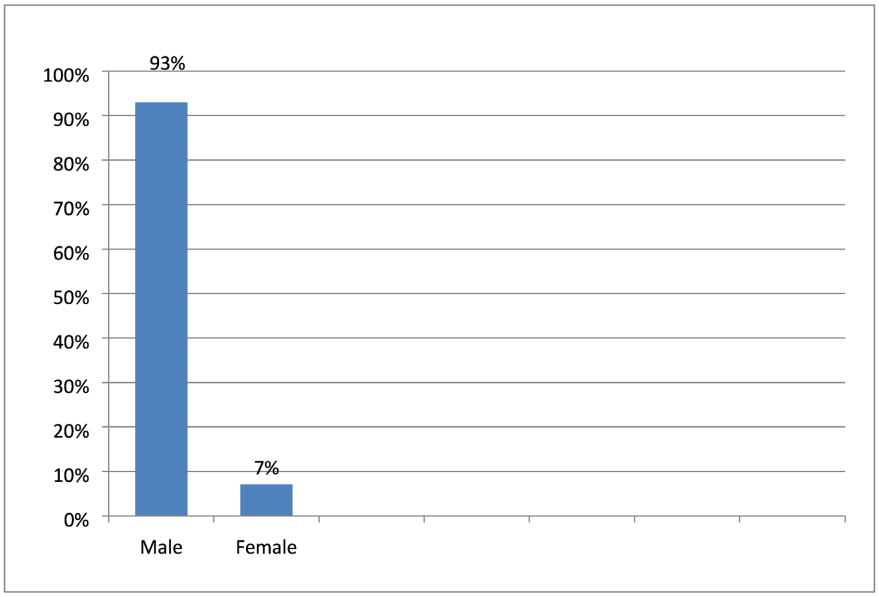


Figure 4. STEMI & Non-STEMI values.

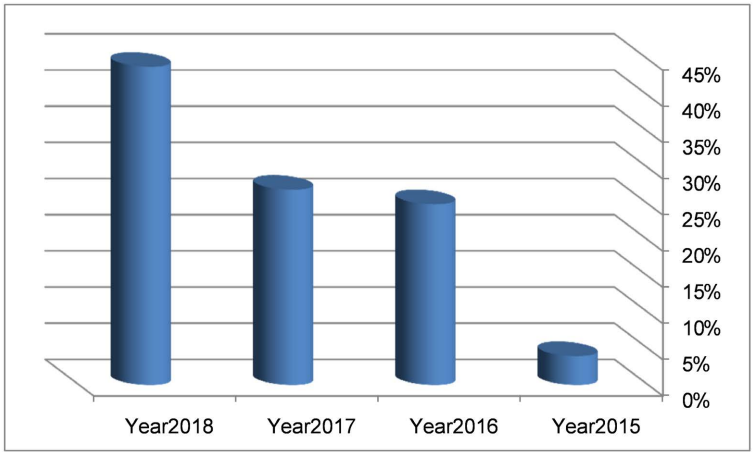


Figure 5. Incidence of MI/Year.

Table 10. Involving the HTN and DM in principles to male and female and their p. value for each one.

	SEX				X^2	P.V
	M		F			
	N	%	N	%		
Hypertension	31	44.3	18	60	2.075	0.150
D.M	12	17.1	16	53.3	13.643	0.000

Table 11. Explain the risk factors in principle to age and their p. value for each one of these risk factors.

	AGE								X^2	P.V
	40 - 49 Y		50 - 59 Y		60 - 79 Y		80 - 100 Y			
	N	%	N	%	N	%	N	%		
Qat showing	10	34.5	8	29.6	12	33.3	1	12.5	1.560	0.669
Tobacco Smoking	6	20.7	4	14.8	13	36.1	0	0	6.993	0.72
Water pipe	5	17.2	4	14.8	4	11.1	0	0	1.849	0.604
Marital status										
Married	29	100	27	100	36	100	8	100		
Occupation									9.533	0.390
Farmer	10	34.5	13	48.1	9	25	2	25		
House wife	6	20.7	8	29.6	6	16.7	2	25		
Solider	1	3.4	1	3.7	1	2.8	0	0		
Unknown	12	41.4	5	18.5	20	55.6	4	50		
Dyslipidemia	4	13.8	4	14.8	0	0	1	12.5	5.608	0.132
Vascular disease	3	10.3	1	3.7	1	2.8	0	0	2.635	0.451
Family history of MI	3	10.3	4	14.8	0	0	0	0	6.343	0.096
Family history of chronic disease	2	6.9	1	3.7	1	2.8	0	0	1.113	0.774
ECG									3.224	0.358
STE	25	86.2	26	96.3	34	94.4	8	100		
NSTE	4	13.8	1	3.7	2	6.5	0	0		

Table 12. Explains the HTN and DM in certain age and their p. value.

	AGE								X^2	P.V
	40 - 49 Y		50 - 59 Y		60 - 79 Y		80 - 100 Y			
	N	%	N	%	N	%	N	%		
Hypertension	13	44.8	17	63	18	50	1	12.5	6.588	0.086
D.M	4	13.8	10	37	11	30.6	3	37.5	4.472	0.215

5. Discussion

Our study was performed on 100 patients who were diagnosed and treated as a case of myocardial infarction in Thamar University AL-wahda teaching hospital Thamar governorate ma'ber city (TUWTH).

Their age range was 40 - 100 years, in this study we focused on the risk factors of myocardial infarction which include:

HTN, gender, age, dyslipidemia, vascular disease and habits as khat chewing and smoking.

The present study showed that: The commonest Myocardial infarction patients was males 70% with mean age 60.13 years, The male to female ratio was (2.5:1), The highest risk factor was HTN 49%, followed by stressful jobs, DM, chewing khat, 37%, 28%, 23% respectively, p. value was significant in DM equal 0.000, occupation equal 0.000 and chronic disease 0.013.

This findings was compared with European study: In which the commonest age of myocardial infarction occurrence was 65 years, the highest R.F was smoking 61.4% followed by overweight 60.9% and the p. value was less than 0.01 for both risk factors [10] [24] [25].

Our study compared also to studies done in Sudia Arabia.

Where in Sudia Arabia, the commonest age was 25 - 75 years. The highest risk factors was dyslipidemia 68.6% followed by abdominal obesity, for both risk factors p. value was significant less than 0.001 [26].

6. Conclusion and Recommendations

6.1. Conclusions

Acute MI above 40 years is becoming raising problem in Thamar city which is more common in men. The commonest risk factor of MI was Hypertension and the chest pain which is most common clinical presentations.

STEMI was the commonest type of MI in our study.

6.2. Recommendations

This study recommends that:

1) The Ministry of Health to perform many of its duties, carry out awareness campaigns to raise the level of the community culture about the tobacco related diseases and the necessity to smoking cessation.

2) Medical contributions from specialists in writing and circulating publica-

tions among members of the community about the importance of exercises and free fatty diet as a food rich in fibers on the health of the heart and body.

3) Routine screening which would help in preventing MI in risky patients.

Conflicts of Interest

The authors declare no conflicts of interest.

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List of Abbreviations

ACEI	Angiotensin Converting Enzyme Inhibitor
ACS	Acute coronary syndrome
APTT	Activated Partial Thromboplastin Time
AST	Aspartate Transaminase
ATP	Adenosine Triphosphate
AVN	Atrioventricular Node
BMI	Body Mass Index
BNP	Brain natriuretic peptide
CABG	Coronary Artery Bypass Graft
CAD	Coronary Artery Diseases
CBC	Complete Blood Count
CHD	Coronary Heart Diseases
CHF	Congestive Heart Failure
CK	Creatine kinas-MB
CTn	Cardiac Troponin
DASH	Dietary Approaches to Stop HTN
DM	Diabetes Miletus
DR	Doctor
ECG (EKG)	Electrocardiogram
F	Female
FH	Family History
ESR	Erythrocyte Sedimentation Rate
HDL	High Density Lipoprotein
HTN	Hypertension
IV	Intra venous
LMWH	Low Molecular Weight Heparin
LBBB	Left Bundle Branch Bloke
LDL	Low Density Lipoprotein
LV	Left Ventricular
M	Male
MI	Myocardial Infarction
NSTEMI	None ST segment Elevation
PCI	Percutaneous Coronary Intervention
PT	Prothrombin Time
P2Y12	
P. value	probability value
RCA	Right Coronary Artery
RF	Risk factor
RV	Right ventricular
SAN	Sino Atrial Node
SGOT	Serums Glutamic Oxalic acetic Transaminase
STEMI	ST Segment Elevation Myocardial Infarction
SPSS	Statistical package for social Sciences
TUMEC	Tamar University Medical Ethics Committee
UR	Upper Reference
URL	Upper Reference Limit
WHO	World Health Organization