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Evaluation of Medication Use among Discharged IHD Patients at Private and Public Hospitals in Sana'a city, Yemen.

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الآية

قال تعالى في كتابه الكريم : ﴿... قُلْ هَلْ يَسْتَوِي الَّذِينَ يَعْلَمُونَ وَالَّذِينَ لَا يَعْلَمُونَ إِنَّمَا يَتَذَكَّرُ أُولُو الْأَلْبَابِ ﴾ صدق الله العظيم

«سورة الزمر : الآية ٩»

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الإهداء

نهدي هذا البحث إلى كل طالب علم يسعى لكسب المعرفة وتزويد رصيده العلمي والثقافي .
إلى مصدر افتخارنا واعتزازنا الى من نفتخر ان نرفع اسامهم عالياً إلى من قدموا كل ما يملكون في سبيل
وصولنا إلى تحقيق كل طموحتنا وذللو الصعاب في طريقنا وساعدونا لإكمال مسيرتنا **اباءنا الكرام** .
إلى منبع عزنا وفخرنا من لهن جل حبنا واحترامنا من سهرن من اجل راحتنا وعانين من اجل رفعتنا فصارين
عنواناً لنجاحنا الى سعادتنا الدائمة وقلوبنا المضيئة الى من جعل الله الجنة تحت اقدامهن **امهاتنا الحبيبات** .
الى الروح الذي سكنت ارواحنا الى اسنادنا وقواتنا وزهور حياتنا **إخواننا واخواتنا الأعزاء** .
اخيراً الى كل من شجعنا ووثق بقدراتنا ولهم أثر وذكرى جميلة في حياتنا .
ولا ينبغي ان ننسى **دكاترتنا** ممن كان لهم الدور الأكبر في مساندتنا ومدنا بالمعلومات القيمة نهدي لكم
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LIST OF ABBREVIATIONS

Abbreviation	Meaning
ACC	American College of Cardiology
ACE	Angiotensin-converting enzyme
ACS	Acute coronary syndromes
AHA	American Heart Association
ARB	Angiotensin receptor blocker
ASCVD	Atherosclerotic cardiovascular disease
AV	Atrioventricular
BMS	Bare metal stent
BP	Blood pressure
CABG	Coronary artery bypass graft
CAD	Coronary artery disease
CCB	Calcium channel blocker
cGMP	Cyclic guanosine monophosphate
CHD	Coronary heart disease
COX-2	Cyclooxygenase-2
CT	Computed tomography
CYP	Cytochrome P450
DES	Drug-eluting stent
DAPT	Dual antiplatelet therapy
EBCT	Electron beam computed tomography
ECG	Electrocardiogram
FDA	Food and Drug Administration
HDL	High-density lipoprotein
HF	Heart failure
HFrEF	Heart failure with reduced ejection fraction
HMG-CoA	3-hydroxy-3-methylglutaryl coenzyme A
HRT	Hormone replacement therapy

Abbreviation	Meaning
HTN	Hypertension
IHD	Ischemic heart disease
IV	Intravenous
LDL	Low-density lipoprotein
MACE	Major adverse cardiac events
MI	Myocardial infarction
MR	Magnetic resonance
MRA	Mineralocorticoid receptor antagonist
Ms	Milliseconds
MVO ₂	Myocardial oxygen consumption
NSTEMI	Non–ST-segment elevation myocardial infarction
NSAID	Nonsteroidal anti-inflammatory drug
OCT2	Organic cation transporter 2
PCI	Percutaneous coronary intervention
P-gp	P-glycoprotein
PTCA	Percutaneous transluminal coronary angioplasty
SIHD	Stable ischemic heart disease
STEMI	ST-segment elevation myocardial infarction
TXA ₂	Thromboxane A ₂
UA	Unstable angina

Kuijh

ABSTRACT

Background: Ischemic heart diseases (IHDs) are a leading cause of death worldwide. Although prescribing according to guidelines improves health outcomes. We determined whether interventions targeted at healthcare professionals are effective to enhance prescribing and health outcomes in patients with IHDs.

Aims of the study: The study was undertaken to evaluate the medication use in discharged patients with IHD among hospitalized patients in a private hospital and public hospital in Sana'a, Yemen, according to IHD Guideline 2021. As well as the major drug-drug interactions.

Materials and Methods: A retrospective cross-sectional study was conducted on patient medication discharge. Patients of age 24 years and above of both sexes were included in the study. The relevant data was collected from Files and computerized hospital. The medications use in discharged with IHD were evaluated and reviewed based Guideline. Then the obtained data was analyzed using descriptive statistics SPSS version 21.

Results: A total of 131 patient prescriptions were enrolled. 108 (83%) were male patients and 23 (17%) were female. The data showed that the major morbidity pattern was HTN 23 (34.4%). And D.M 17 (26.4%). Based on Guideline, among prescribed drugs, the results demonstrated in private hospital 34 (48.6%) of patients were yes followed to 4 classes. while in Public hospital 50 (82%) of patients were yes followed to 4 classes. The results also revealed that 7 (10%) of patients had major drug-drug interactions in private hospital. While 13 (21.3%) of patients had major drug – drug interaction in public hospital.

Chapter-I

1.1 Introduction.

1.1.1 Definition Ischemic heart disease:

Ischemic heart disease (IHD) is also called coronary heart disease (CHD) or coronary artery disease (CAD). The term *ischemic* refers to a decreased supply of oxygenated blood to the heart muscle. IHD is caused by stenosis, or narrowing, in one or more of the major coronary arteries that supply blood to the heart, most commonly by atherosclerotic plaques. Atherosclerotic plaques may impede coronary blood flow to the extent that cardiac tissue distal to the coronary artery narrowing is deprived of sufficient oxygen to meet oxygen demand. IHD results from an imbalance between myocardial oxygen supply and oxygen demand (Figure 1) ⁽⁴⁶⁾. Common clinical manifestations of IHD include chronic stable angina and the ACS of unstable angina (UA), non–ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI).

Angina pectoris, or simply angina, is the most common symptom of IHD. Angina is discomfort in the chest that occurs when the blood supply to the myocardium is compromised. Chronic stable angina is a chronic occurrence of chest discomfort due to transient myocardial ischemia with physical exertion or other conditions that increase oxygen demand. The primary focus of this chapter is the management of stable IHD (SIHD), the most common manifestation of which is chronic stable angina. However, some information is also provided related to acute coronary syndrome (ACS), given the overlap between the two disease states. The American College of Cardiology (ACC), the American Heart Association (AHA), and several other organizations have jointly published practice guidelines for the management of SIHD. Refer to these and related guidelines for further information ⁽¹⁻³⁾.

1.1.2 Epidemiology and Etiology:

IHD affects over 16 million Americans and is the leading cause of death for both men and women in the United States ⁽⁴⁾. The incidence of IHD is higher in middle-aged men compared with women. However, the rate of IHD increases twofold to threefold in women after menopause. Chronic stable angina is the initial manifestation of IHD in about 50% of patients, whereas ACS is the first sign of IHD in other patients. Patients with SIHD experience considerable morbidity and frequently require hospitalization for chest pain and evaluation for potential ACS. In addition, SIHD negatively impacts health-related quality of life. Thus, in patients with SIHD, it is important to optimize pharmacotherapy to reduce symptoms, improve quality of life, slow disease progression, and prevent ACS.

1.1.3 Conditions Associated with Angina:

(Figure 2) ⁽⁴⁵⁾ shows the anatomy of the coronary arteries. The major epicardial coronary arteries are the left main, left anterior descending, left circumflex, and right coronary arteries. Atherosclerosis leading to obstructive lesions in one or more of the major coronary arteries or their principal branches is the major cause of angina. Vasospasm at the site of an atherosclerotic plaque may further constrict blood flow and contribute to angina. Less commonly, vasospasm in coronary arteries with no or minimal atherosclerotic disease can produce angina and even precipitate ACS. This uncommon form of angina is referred to as variant or Prinzmetal angina. Other nonatherosclerotic conditions that can cause angina-like symptoms are listed in Table 1 ⁽¹⁾. It is important to differentiate the etiology of chest discomfort because treatment varies depending on the underlying disease process.

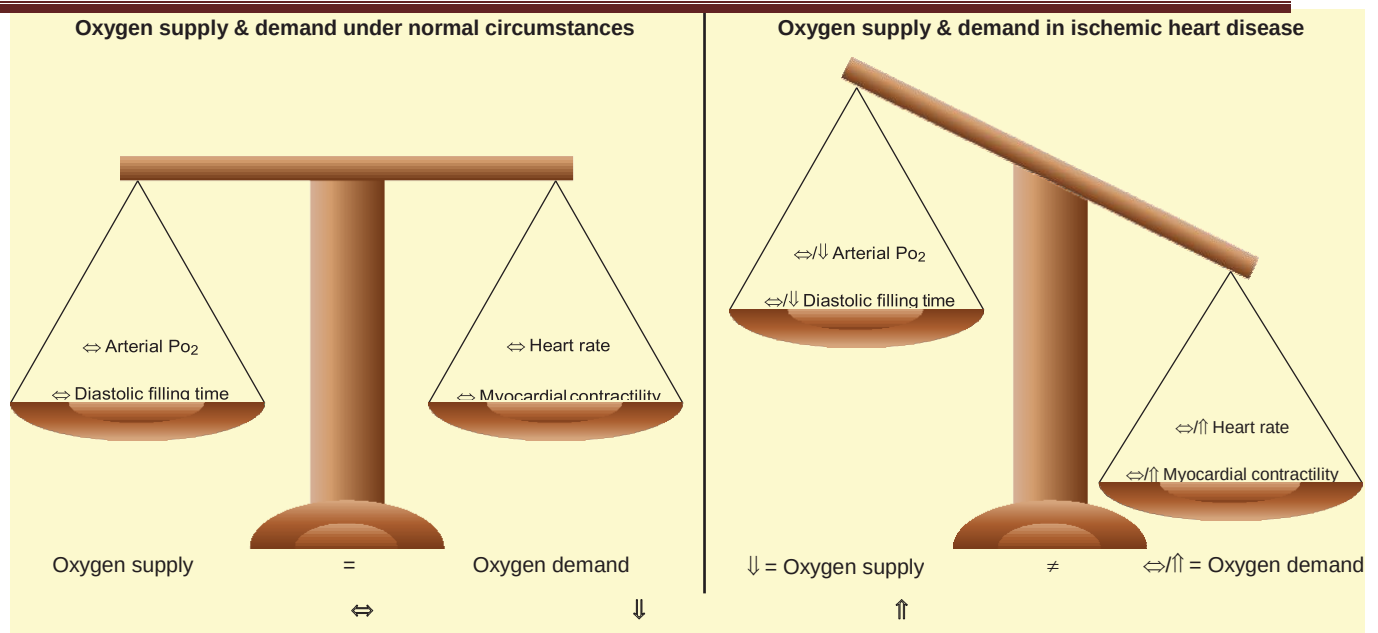


FIGURE 1 ⁽⁴⁶⁾ .This illustration depicts the balance between myocardial oxygen supply and demand and various factors that affect each. It should be noted that diastolic filling time is not an independent predictor of myocardial oxygen supply per se, but rather a determinant of coronary blood flow. On the left is myocardial oxygen supply and demand under normal circumstances. On the right is the mismatch between oxygen supply and demand in patients with IHD. In patients without IHD, coronary blood flow increases in response to increases in myocardial oxygen demand. However, in patients with IHD, coronary blood flow cannot sufficiently increase (and may decrease) in response to increased oxygen demand, resulting in angina. (IHD, ischemic heart disease; P_{O_2} , partial pressure of oxygen) .

1.2 Risk Factors:

Factors that predispose an individual to IHD are listed in Table 2 ⁽¹⁾ . Optimization of modifiable risk factors can significantly reduce the risk of myocardial infarction (MI) ⁽¹⁾ .Hypertension, diabetes, dyslipidemia, and cigarette smoking are associated with endothelial damage and dysfunction and contribute to the development of atherosclerosis. Physical inactivity and obesity independently increase the risk for IHD, in addition to predisposing individuals to hypertension, dyslipidemia, and diabetes. IHD, in addition to predisposing individuals to hypertension, dyslipidemia, and diabetes.

Patients with multiple risk factors, particularly those with diabetes, are at the greatest risk for IHD, experiencing fivefold to sevenfold higher risk compared to individuals without risk factors ⁽⁴⁾ .

Although alternative definitions exist, metabolic syndrome is generally considered a constellation of

common cardiovascular risk factors including hypertension, abdominal obesity, dyslipidemia, and insulin resistance. Metabolic syndrome increases the risk of developing IHD and related complications by twofold ⁽⁵⁾. According to a joint statement from the International Diabetes Federation, National Heart, Lung, and Blood Institute, AHA, World Heart Federation, International Atherosclerosis Society, and International Association for the Study of Obesity, patients must meet at least three of the following criteria for the diagnosis of metabolic syndrome (5):

- Increased waist circumference (40 in [102 cm] or greater in men and 35 in [89 cm] or greater in women).
 - Triglycerides of 150 mg/dL (1.70 mmol/L) or greater or active treatment to lower triglycerides.
 - Low high-density lipoprotein (HDL) cholesterol (< 40 mg/dL [1.03 mmol/L] in men and < 50 mg/dL [1.29 mmol/L] in women) or active treatment to raise HDL cholesterol.
 - Systolic blood pressure (BP) of 130 mm Hg or greater, diastolic BP of 85 mm Hg or greater, or active treatment with antihypertensive therapy.
 - Fasting blood glucose of 100 mg/dL (5.6 mmol/L) or greater or active treatment for diabetes.
- Early detection and aggressive modification of risk factors are among the primary strategies for delaying IHD progression and preventing IHD-related events including death.

1.3 Pathophysiology:

The determinants of myocardial oxygen supply and demand are shown in Figure 1⁽⁴⁶⁾.

Increases in heart rate, cardiac contractility, and left ventricular wall tension increase the rate of myocardial oxygen consumption (MVO_2). Ventricular wall tension is a function of BP, systemic vascular resistance, left ventricular end-diastolic volume, and ventricular wall thickness. Physical exertion increases MVO_2 and commonly precipitates symptoms of angina in patients with significant coronary atherosclerosis.

Reductions in coronary blood flow (secondary to atherosclerotic plaques, vasospasm, or thrombus formation) and arterial oxygen content (secondary to hypoxia) decrease myocardial oxygen supply. Because the coronary arteries fill during diastole, decreases in diastolic filling time (eg, tachycardia) can also reduce coronary perfusion and myocardial oxygen supply. Anemia, carbon monoxide poisoning, and cyanotic congenital heart disease are examples of

conditions that can lead to diminished myocardial oxygen delivery and precipitate angina in the face of adequate coronary perfusion.

1.3.1 Coronary Atherosclerosis:

The normal arterial wall is illustrated in panel A of (Figure 3)⁽⁷⁾. The intima consists of a layer of endothelial cells that line the lumen of the artery and form a selective barrier between the vessel wall and blood contents. Vascular smooth muscle cells are found in the media. The vascular adventitia comprises the artery's outer layer. Atherosclerotic lesions form in the subendothelial space in the intimal layer.

Endothelial damage and dysfunction, commonly caused by hypertension, diabetes, and smoking, allow low-density lipoprotein (LDL) cholesterol and inflammatory cells (eg, monocytes and T lymphocytes) to migrate from the plasma to the subendothelial space.

Macrophages also secrete growth factors that promote smooth muscle cell migration from the media to the intima. The result is the development of early atherosclerosis in the form of a fatty streak consisting of lipid-laden macrophages and smooth muscle cells.

The fatty streak enlarges as foam cells, smooth muscle cells, and necrotic debris accumulate in the subendothelial space. A collagen matrix forms a fibrous cap that covers the lipid core of the lesion to establish an atherosclerotic plaque. The atherosclerotic plaque may progress until it protrudes into the artery lumen and impedes blood flow. When the plaque occludes 70% or more of a major coronary artery or 50% or more of the left main coronary artery, the patient may experience angina during activities that increase myocardial oxygen demand.

Compared with men, women with angina are more likely to present with microvascular disease. Microvascular angina, also called cardiac syndrome X, refers to disease of the smaller coronary vessels causing typical angina in the absence of obstructive CAD of the epicardial arteries. Endothelial dysfunction and reduced smooth muscle relaxation, resulting in reduced vasodilation and enhanced vasoconstriction, are proposed to contribute to microvascular disease⁽⁶⁾.

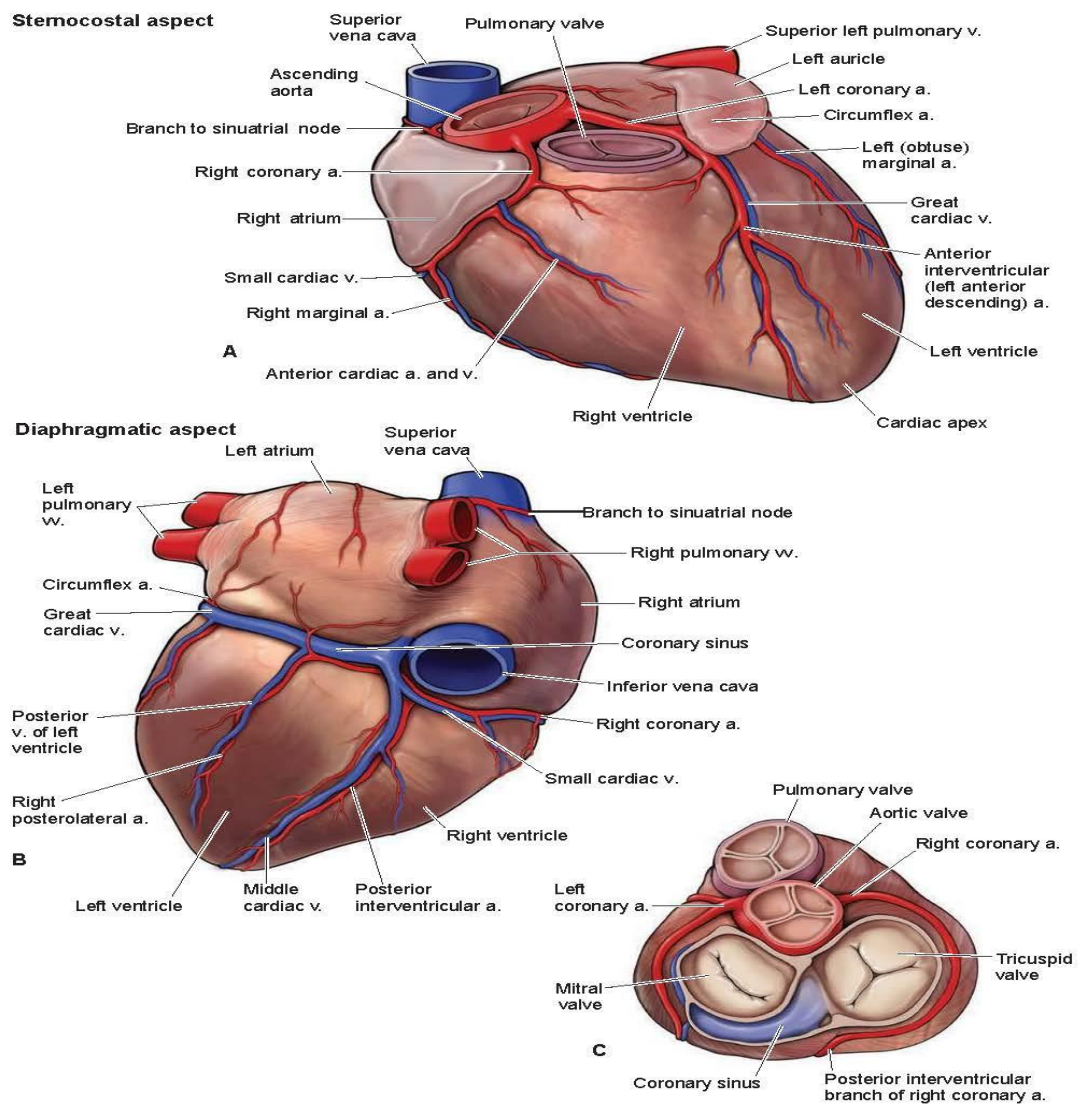


FIGURE 2 ⁽⁴⁵⁾. Coronary artery anatomy with sternocostal and diaphragmatic views. (a., artery; v., vein.) . Heart. In: Morton DA, Foreman K, Albertine KH, eds. The Big Picture:Gross Anatomy. New York, NY: McGraw-Hill; 2011. <http://accesspharmacy.mhmedical.com/content.aspx?bookid=381&sectionid=40140010>, with permission.

(Table 1)	
Non atherosclerotic Conditions That Can Cause Angina-Like Symptoms ⁽¹⁾.	
Organ System	Condition
Cardiac	Aortic dissection, aortic stenosis, coronary artery vasospasm, pericarditis, valvular heart disease, severe uncontrolled Hypertension.
Noncardiac	Anemia, anxiety disorders, carbon monoxide poisoning, chest wall trauma, cocaine use, esophageal reflux or spasm, peptic ulcer disease, pleuritis, pneumonia, pneumothorax, pulmonary embolus, pulmonary hypertension, thyrotoxicosis.

(Table 2)	
Major Risk Factors for Ischemic Heart Disease ⁽¹⁾.	
Modifiable	No modifiable
Cigarette smoking Dyslipidemia • Elevated LDL or total cholesterol • Reduced HDL cholesterol Diabetes mellitus Hypertension Physical inactivity Obesity (body mass index ≥ 30 kg/m ²) Low daily fruit and vegetable consumption Alcohol overconsumption.	Age ≥ 45 years for men, age ≥ 55 years for women Gender (men and postmenopausal women) Family history of premature cardiovascular disease, defined as cardiovascular disease in a male first-degree relative (ie, father or brother) < 55 years or a female first-degree relative (ie, mother or sister) < 65 years.

1.3.2 Stable versus Unstable Atherosclerotic Plaques:

The hallmark feature in the pathophysiology of SIHD is an established atherosclerotic plaque in one or more of the major coronary arteries that impedes coronary blood flow such that myocardial oxygen supply can no longer meet myocardial oxygen demand. In contrast, the hallmark feature in the pathophysiology of ACS is atherosclerotic plaque rupture with subsequent thrombus formation. Plaque rupture refers to fissuring of the fibrous cap and exposure of the plaque contents to elements in the blood. Plaque composition, rather than the degree of coronary stenosis, determines the stability of the plaque and the likelihood of rupture and ACS. As depicted in Figure 3B,⁽⁷⁾ a stable lesion characteristic of SIHD consists of a small lipid core surrounded by a thick fibrous cap that protects the lesion from the shear stress of blood flow. In contrast, an unstable plaque consists of a thin, weak cap covering a large lipid-rich core that renders the plaque vulnerable to rupture Figure 3C.⁽⁷⁾ The transformation of a stable plaque into an unstable plaque involves the degradation of the fibrous cap by substances released from macrophages and other inflammatory cells. Following plaque rupture, platelets adhere to the site of rupture (Figure 3D),⁽⁷⁾ aggregate Figure 3E,⁽⁷⁾ and generate thrombin leading to the development of a fibrin clot Figure 3F⁽⁷⁾.

Coronary thrombi extend into the vessel lumen, where they partially or completely occlude blood flow, potentially resulting in UA or MI.

Unstable plaque often produces minimal occlusion of the coronary vessel, and the patient remains asymptomatic until the plaque ruptures. In fact, many ACS arise from vulnerable plaques that occlude less than 50% of the coronary lumen⁽⁷⁾.

1.3.3 Coronary Artery Vasospasm:

Prinzmetal or variant angina results from spasm (or vasoconstriction) of a coronary artery in the absence of significant atherosclerosis. Variant angina usually occurs at rest, especially in the early morning hours. Although vasospasm is generally transient, it may persist long enough to cause myocardial ischemia and subsequent infarction. Patients with variant angina are typically younger than those with chronic stable angina and often do not possess the classic risk factors for IHD. The cause of variant angina is unclear but appears to involve vagal withdrawal, endothelial dysfunction, and paradoxical response to agents that normally cause vasodilation. Precipitants of variant angina include cigarette smoking, cocaine or amphetamine use, hyperventilation, and exposure to cold temperatures. The management of variant angina differs from that of classic angina, and thus it is important to distinguish between the two.

1.4 Clinical presentation and diagnosis history:

The evaluation of a patient with suspected IHD begins with a detailed history of symptoms. The classic presentation of angina is described in the Clinical Presentation and Diagnosis text box. Chronic stable angina should be distinguished from UA because the latter is associated with a greater risk for MI and death and requires hospitalization for more aggressive treatment. Because the pathophysiology of SIHD is due primarily to increases in oxygen demand rather than acute changes in oxygen supply, symptoms are typically reproducible when provoked by exertion, exercise, or stress. The exception may be a patient with coronary artery vasospasm, in whom symptoms may be more variable and unpredictable. In contrast to SIHD, ACS generally occurs in response to an acute decrease in coronary blood flow leading to inadequate oxygen supply. Consequently, ACS is marked by prolonged symptoms, often occurring at rest, or an escalation in the frequency or severity of angina over a short period of time. The presentation of UA is described in Table 3 ⁽⁸⁾.

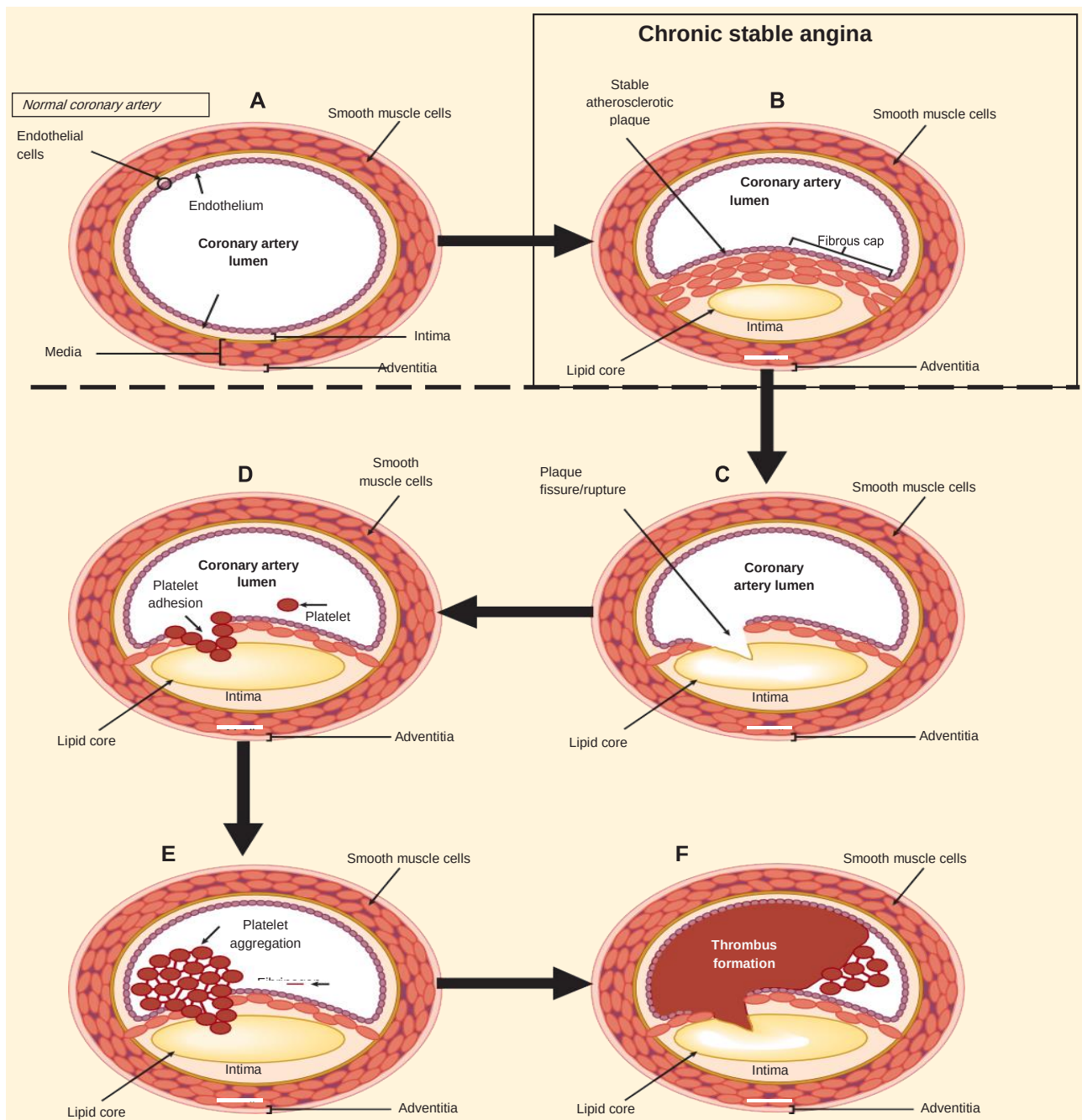


FIGURE 3 ⁽⁷⁾. Pathophysiology of chronic stable angina versus acute coronary syndromes. Panel A depicts the cross-section of a normal coronary artery. Panel B depicts the cross-section of a coronary artery with a stable atherosclerotic plaque. Note that the lipid core is relatively small in size and the fibrous cap is made up of several layers of smooth muscle cells. Panel C depicts an unstable atherosclerotic plaque with a larger lipid core, and a thin fibrous cap composed of a single layer of smooth muscle cells with a fissure or rupture. Panel D depicts platelet adhesion in response to the fissured plaque. Platelet activation may ensue, leading to platelet aggregation as fibrinogen binds platelets to one another to form a mesh-like occlusion in the coronary lumen (Panel E). At this stage, patients may experience symptoms of acute coronary syndrome. If endogenous anticoagulant proteins fail to halt this process, platelet aggregation continues and fibrinogen is converted to fibrin, resulting in an occlusive thrombus (Panel F).

(Table 3)
Presentations of Acute Coronary Syndromes ⁽⁸⁾
<ul style="list-style-type: none"> • Angina at rest that is prolonged in duration, usually lasting over 20 minutes. • Angina of recent onset (within 2 months) that markedly limits usual activity. • Angina that increases in severity (ie, by Canadian Cardiovascular Society Classification System Class of one level or greater [Table 7–4]), frequency, or duration, or that occurs with less provocation over a short time period (ie, within 2 months).

1.4.1 Canadian Cardiovascular Society Classification System:

The Canadian Cardiovascular Society Classification System (Table 4) ⁽⁹⁾ is commonly used to assess the degree of disability resulting from IHD ⁽⁹⁾ .Patients are categorized into one of four classes depending on the extent of activity that produces angina. Grouping patients according to this or a similar method is commonly used to assess changes in IHD severity over time and the effectiveness of pharmacologic therapy.

(Table 4)	
Canadian Cardiovascular Society Classification System of Angina ⁽⁹⁾	
Class	Description
I	Able to perform ordinary physical activity (eg, walking, climbing stairs) without symptoms. Strenuous, rapid, or prolonged exertion causes symptoms.
II	Symptoms slightly limit ordinary physical activity. Walking rapidly or for more than two blocks, climbing stairs rapidly, or climbing more than one flight of stairs causes symptoms.
III	Symptoms markedly limit ordinary physical activity. Walking less than two blocks or climbing one flight of stairs causes symptoms.
IV	Angina may occur at rest. Any physical activity causes symptoms.

1.4.2 Physical Findings and Laboratory Analysis:

A thorough medical history, physical examination, and laboratory analysis are necessary to ascertain cardiovascular risk factors and to exclude nonischemic and noncardiac conditions that could mimic angina-like symptoms. Laboratory analyses should assess for glycemic control (ie, fasting glucose, glycated hemoglobin), fasting lipids, hemoglobin, and organ function (ie, blood urea nitrogen, creatinine, liver function tests, thyroid function tests). For patients with ACS, serial measurements of cardiac troponin (usually 2–3 measurements over a period of 3–12 hours) are performed to exclude the diagnosis of an acute MI. Cardiac findings on the physical examination are often normal in patients with SIHD. However, findings such as carotid bruits, abdominal and/or renal bruits, or abnormal peripheral pulses would indicate atherosclerosis in other vessel systems and raise the suspicion for IHD.

1.4.3 Diagnostic Tests:

A resting 12-lead ECG is indicated in all patients with new or worsening symptoms of ischemia. Patients with characteristic chest discomfort, accompanied by ST-segment elevation in two or more contiguous leads, or a new left bundle branch block are often diagnosed with MI (STEMI) and are at the highest risk of death. Consequently, STEMI is treated as a medical emergency, often requiring expeditious percutaneous coronary intervention (PCI) or fibrinolytic therapy to restore blood flow in the occluded artery and reperfuse the myocardium. For patients with ACS, cardiac troponin is measured serially to distinguish between MI (elevated cardiac troponin) and either UA or noncardiac causes of chest discomfort (normal cardiac troponin).

“Stress” testing with either exercise or pharmacologic agents increases myocardial oxygen demand and is commonly used to evaluate the patient with suspected IHD. Approximately 50% of patients with IHD who have a normal ECG at rest will develop ECG changes with exercise on a treadmill (most commonly) or bicycle ergometer.

Dobutamine is a common pharmacologic agent administered as an IV infusion to patients who are unable to exercise. Dobutamine increases oxygen demand by stimulating the B1_receptor, increasing heart rate and contractility. Echocardiography is commonly performed along with exercise or dobutamine (eg, treadmill or dobutamine stress echocardiography) to identify stress-induced wall motion abnormalities of the left ventricle indicative of IHD.

Exercise or pharmacologic agents are also commonly combined with radionuclide myocardial perfusion imaging (nuclear imaging studies) or cardiac MR to detect IHD.

An intravenous (IV) radioactive tracer is administered and accumulates in normal myocytes in proportion to coronary blood flow. Adenosine, dipyridamole, and regadenoson are coronary vasodilators that increase coronary blood flow in healthy arteries but not in atherosclerotic vessels. Radionuclide myocardial perfusion imaging is performed at rest and following exercise or pharmacologic stressor to detect perfusion defects indicative of IHD. Positive emission tomography, which utilizes a radioactive tracer that emits γ rays, is an alternative to conventional myocardial perfusion imaging that may be preferred in obese patients. Similar to other myocardial perfusion imaging, stress cardiac MR can detect wall motion abnormalities as well as perfusion defects, the latter often performed using late gadolinium enhancement, to determine the extent and severity of myocardial scarring that may be present.

Coronary artery calcium scoring via computed tomography (CT), also known as electron beam CT (EBCT) or “ultra-fast CT,” may be performed as a noninvasive means to assess for CAD. Calcium deposits within the atherosclerotic coronary arteries are detected on CT, a calcium score is calculated, and the risk for IHD-related events is estimated.

Coronary angiography (also referred to as a cardiac catheterization or “cardiac cath”) is indicated when stress testing results are either abnormal or inconclusive, symptoms of angina are poorly controlled, or in patients for whom the clinical suspicion of IHD remains high despite a normal

stress test result. Angiography involves catheter insertion into either the femoral or radial artery with advancement of the catheter into the ascending aorta near the coronary ostia. Contrast medium is injected through the catheter into the coronary arteries and fluoroscopy is performed to visualize the coronary anatomy and detect IHD. Contrast medium must be used cautiously in patients with preexisting renal disease (especially in those with diabetes) to avoid contrast-induced nephropathy and often warrants prophylactic hydration periprocedurally. The decision to establish the diagnosis of IHD via stress testing, coronary angiography, or both is often individualized for each patient based on several factors including baseline assessment of IHD risk (eg, pretest probability), sensitivity and specificity of the diagnostic test, results of previous diagnostic tests, and risk for complications (eg, contrast nephropathy).

1.5 Treatment:

Desired Outcomes:

Once a patient is diagnosed with IHD, the clinician should provide counseling on lifestyle modifications, institute appropriate pharmacologic therapy, and evaluate the need for revascularization. Optimal medical therapy is essential for managing patients with SIHD. The major goals for the treatment of SIHD are to:

- Prevent ACS and death.
- Alleviate acute symptoms of angina.
- Prevent recurrent symptoms of angina.
- Prevent progression of the disease
- Reduce complications of IHD.
- Avoid or minimize adverse treatment effects.

The treatment approach to address these goals is illustrated in Figure 4 ⁽⁴⁶⁾.

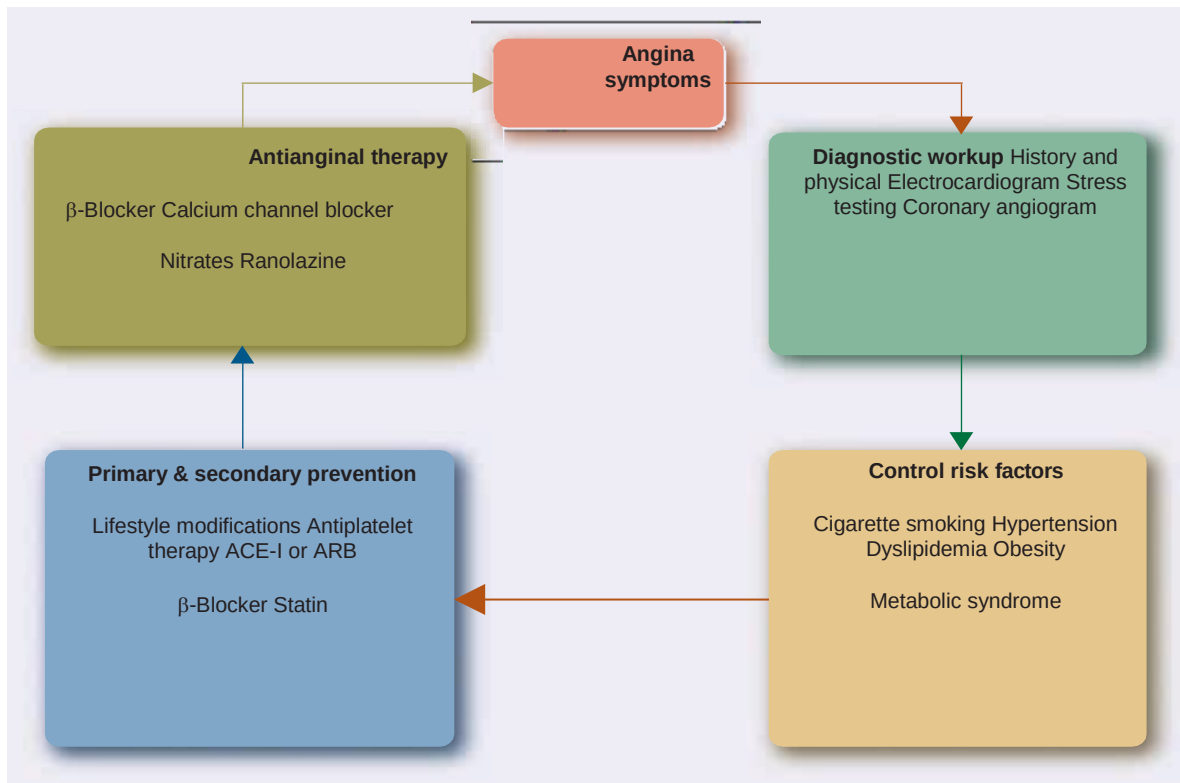


FIGURE 4 ⁽⁴⁶⁾ . General treatment strategies for angina follow in clockwise fashion from the top center. (ACE-I, angiotensin- converting enzyme inhibitor; ARB, angiotensin receptor blocker.).

1.5.1 General Approach to Treatment:

The primary strategies for preventing ACS and death (eg, primary or secondary prevention) are to:

- Aggressively modify cardiovascular risk factors.
- Slow the progression of coronary atherosclerosis.
- Stabilize existing atherosclerotic plaques.

The treatment algorithm in Figure 5 ⁽⁴⁶⁾ summarizes the appropriate management of IHD.

Risk factor modification is accomplished through lifestyle changes and pharmacologic therapy. Both 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (HMG-CoA reductase inhibitors or statins) and angiotensin-converting enzyme (ACE) inhibitors possess vasculoprotective effects (eg, anti-inflammatory effects, antiplatelet effects, improvement in endothelial function, and/or improvement in arterial compliance and tone). Together with aspirin, these drugs reduce the risk of acute coronary events and death in patients with SIHD. In select patients with IHD (following hospitalization for ACS $\pm$ PCI and/or following intracoronary stent placement), dual antiplatelet therapy (DAPT) with aspirin and a P2Y₁₂ antagonist has also been shown to reduce ischemic events. Angiotensin receptor blockers (ARBs) may be used in patients who cannot tolerate ACE inhibitors because of side effects (eg, chronic cough). B-blockers decrease morbidity and improve survival in patients who have suffered an MI, particularly in patients with concomitant heart failure with reduced ejection fraction (HFrEF).

The goal of therapies that alleviate and prevent angina is improving the balance between myocardial oxygen demand and supply. Drug treatment is primarily used to reduce oxygen demand, whereas revascularization by PCI and coronary artery bypass graft (CABG) surgery effectively restore coronary blood flow, improving myocardial oxygen supply.

Adverse treatment effects can be averted by avoiding drug interactions and the use of drugs that may have unfavorable effects on comorbid diseases. Appropriate drug dosing and monitoring reduces the risk for adverse treatment effects. Drugs should be initiated in low doses, with careful up-titration as necessary to control symptoms of angina and cardiovascular risk factors.

1.5.2 Lifestyle Modifications:

Lifestyle modifications (smoking cessation, avoidance of secondhand smoke, dietary modifications, increased physical activity, and weight loss) reduce cardiovascular risk factors, slow the progression of SIHD, and decrease the risk for SIHD-related complications. Cigarette smoking is the single most preventable cause of SIHD and SIHD-related death. Smoking may also attenuate the antianginal effects of drug therapy.

Clinicians should counsel patients and/or family members who smoke on the importance of smoking cessation at each encounter and offer referral to smoking cessation programs. There are several pharmacologic aids for smoking cessation. Transdermal nicotine replacement therapy and bupropion have been studied in patients with SIHD and appear safe and effective ^(10,11). Varenicline, a partial nicotine receptor agonist, is similarly efficacious but has been associated with worsening of preexisting depression and suicidal ideation ^(12,13). Thus, it should be used cautiously, particularly in patients treated for depression, and the risk:benefit ratio should be discussed with patients. Weight loss, through caloric restriction and increased physical activity, should be encouraged in patients who have a body mass index greater than 25 kg/m². Dietary counseling should be provided to all patients with newly diagnosed angina regardless of weight. The AHA recommends a diet that includes a variety of fruits, vegetables, grains, low-fat or nonfat dairy products, fish, legumes, poultry, and lean meats ⁽¹⁴⁾.

The addition of plant stanols/sterols (2 g/day) and/or viscous fiber (over 10 g/day) are effective in lowering LDL cholesterol ⁽¹⁵⁾ . Specific dietary recommendations for patients with IHD should include the following ^(1,14) :

- Limit cholesterol intake to less than 200 mg/day.
- Limit consumption of saturated fat to less than 7% and *trans* fatty acids to less than 1% of total calories.
- Limit daily sodium intake to 2.4 g (6 g of salt) for BP control.
- Limit alcohol consumption to 1 drink/day for women and 1 to 2 drinks/day for men unless otherwise contraindicated.

Exercise facilitates weight loss, BP reduction, and glycemic control. In addition, regular exercise in patients with SIHD improves functional capacity, reduces symptoms of angina, and reduces mortality.^{1,16} Guidelines recommend moderate intensity aerobic activity, such as brisk walking, ideally for 30 to 60 minutes at least 5 and preferably 7 days weekly ⁽¹⁾ . Medically supervised cardiac rehabilitation programs are recommended for high-risk patients.

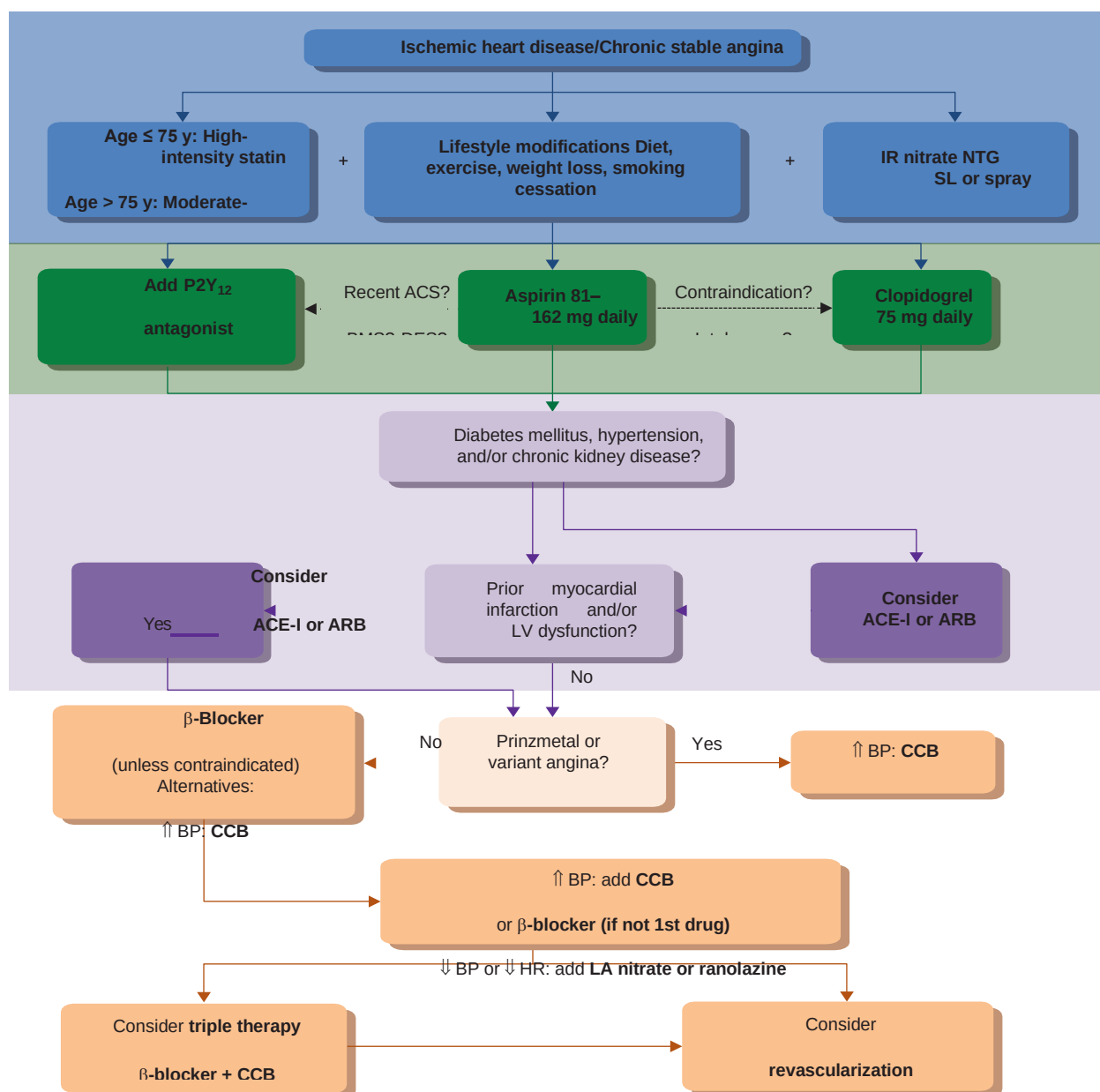


FIGURE 5 (46). Treatment algorithm for ischemic heart disease. It begins at the top (blue section⁴²), which suggests risk factor modifications as the first treatment modality. Moving down to the green section, appropriate antiplatelet therapy is selected. The purple section identifies patients at high risk for major adverse cardiac events (MACE) and suggests appropriate drug therapy to decrease cardiovascular risk. The orange section at the bottom recommends appropriate antianginal therapy. ^aThe minimum duration of P2Y inhibitors in combination with aspirin (DAPT) following intracoronary stent placement is as follows: at least 1 month for BMSs and at least 6 months for DESs for patients with stable ischemic heart disease, at least 12 months in patients with stents placed for ACS regardless of stent type. (ACE-I, angiotensin-converting enzyme inhibitor; ACS, acute coronary syndrome; ARB, angiotensin receptor blocker; BMS, bare metal stent; BP, blood pressure; CABG, coronary artery bypass graft; CCB, calcium channel blocker; DES, drug-eluting stent; HR, heart rate; IR, immediate release; LA, long acting; LDL, low-density lipoprotein; LV, left ventricular; NTG, nitroglycerin; PCI, percutaneous coronary intervention; SL, sublingual.).

1.5.3 Interventional Approaches to Revascularization:

1.5.3.1 Percutaneous Coronary Intervention:

For patients with SIHD, optimal medical therapy is equivalent to PCI in reducing major adverse cardiac events (MACE), which include death, nonfatal MI, and revascularization ⁽¹⁷⁾. Therefore, medical therapy is preferred as the initial treatment strategy.

However, when optimal medical therapy fails, symptoms are unstable, or extensive coronary atherosclerosis is present (eg, > 70% occlusion of coronary lumen), PCI is often performed to restore coronary blood flow and relieve symptoms. Several catheter-based interventions may be used during PCI, including:

- Percutaneous transluminal coronary angioplasty (PTCA)
- Intracoronary bare metal stent (BMS) placement
- Intracoronary drug-eluting stent (DES) placement
- Rotational atherectomy

During PCI, a catheter is advanced into the diseased coronary artery, as described for cardiac catheterization. If PTCA is performed, a balloon at the end of the catheter is inflated inside the artery at the site of the critical stenosis, compressing the atherosclerotic plaque from the coronary lumen and restoring normal myocardial blood flow. Most PCI procedures involve the placement of a stent, a small metal scaffold-like device similar in size and shape to the spring at the tip of a ballpoint pen, at the site of angioplasty. Coronary stents are contained on special balloon catheters that are inflated at the site of stenosis to deploy the stent in the wall of the coronary artery, forming a sort of bridge to maintain a patent artery and improve coronary blood flow. Either a BMS or a DES may be used. DESs are impregnated with low concentrations of an antiproliferative drug (paclitaxel, everolimus, sirolimus, or zotarolimus), which is released locally over a period of weeks to inhibit in-stent restenosis of the coronary artery after PCI.

Compared to BMS, DESs have been associated with a significant reduction in all-cause mortality for patients with IHD ⁽¹⁸⁾ . Stents are thrombogenic, especially until they become endothelialized (covered in endothelial cells like a normal coronary artery). To reduce the risk for in-stent thrombosis, MI, or death, DAPT (discussed later) is required until the stent becomes endothelialized. Lastly, rotational atherectomy may be performed wherein a special catheter is used to essentially cut away the atherosclerotic plaque, restoring coronary blood flow.

1.5.3.2 Coronary Artery Bypass Graft Surgery:

As an alternative to PCI, CABG surgery, or open-heart surgery, may be performed if the patient is found to have extensive coronary atherosclerosis (generally > 70% occlusion of three or more coronary arteries) or is refractory to optimal medical treatment. In the former case, recent evidence suggests that CABG surgery is associated with a reduction in both the need for revascularization and mortality compared with PCI, particularly in patients with diabetes ⁽¹⁹⁾ . During CABG surgery, veins from the leg (ie, saphenous veins) and/or arteries from the chest wall (ie, internal mammary arteries) or less commonly from the arm (ie, radial artery) or stomach (gastroepiploic artery) are harvested and used as conduits to restore coronary blood flow.

A median sternotomy, in which an incision is made the length of the sternum followed by surgical division of the sternum with an oscillating saw, is commonly required to gain access to the thoracic cavity and expose the heart. As the “new” blood vessels are being engrafted, the patient is typically placed on cardiopulmonary bypass (ie, heart-lung machine) to maintain appropriate myocardial and systemic perfusion. Alternative surgical approaches for advanced IHD may be used in some settings including “off-pump” CABG (cardiopulmonary bypass is not required) and minimally invasive CABG (ie, thoracoscopic surgery), although these techniques are uncommon. Because of its extremely invasive nature, CABG surgery is generally reserved for patients with extensive IHD or as a treatment of last resort in patients with symptoms refractory to medical therapy.

1.5.4 Pharmacologic Therapy:

Pharmacotherapy to Prevent Acute Coronary Syndromes and Death:

1.5.4.1 Control of Risk Factors:

A major component of any SIHD treatment plan is control of modifiable risk factors, including dyslipidemia, hypertension, and diabetes. Because lipoprotein metabolism and the pathophysiology of atherosclerosis are closely linked, treatment of dyslipidemias is critical for both primary and secondary prevention of IHD- related cardiac events. In 2013, the ACC/AHA revised guidelines for the management of patients with dyslipidemia ⁽²⁰⁾.

Unlike previous guidelines for dyslipidemia, current guidelines no longer recommend specific targets for LDL and non-HDL cholesterol and de-emphasize the use of non-statin therapies for the treatment of dyslipidemia. Rather, current guidelines focus on the use of statins stratified by presence of or 10-year risk for atherosclerotic cardiovascular disease (ASCVD). Recommendations for initiation of statin therapy for dyslipidemia include ⁽²⁰⁾ :

- High-intensity statin therapy in the presence of clinical ASCVD and for patients without clinical ASCVD in whom LDL cholesterol is 190 mg/dL (4.91 mmol/L) or greater; moderate-intensity therapy for elderly patients (age > 75 years) or those for whom safety concerns exist.
- Moderate to high-intensity statin therapy for patients at least 40 years of age with diabetes.
- Moderate to high-intensity statin therapy for patients with LDL cholesterol less than 190 mg/dL (4.91 mmol/L) who are at least 40 years of age and have a 10-year ASCVD risk of 7.5% or greater in the absence of diabetes; consideration may be given to moderate-intensity statin therapy in patients with a 10-year ASCVD risk between 5% and 7.5%.

Hypertension is another major, modifiable risk factor for the development of IHD and related complications. Aggressive identification and control of hypertension are warranted in patients with SIHD to minimize the risk of MACE. Most recently, the ACC and AHA released updated guidelines for the management of hypertension in adults, with specific recommendations for patients with SIHD ⁽²¹⁾.

Patients with SIHD and a BP of 130/80 mm Hg or higher should be started on antihypertensive therapy to reduce BP to a target of less than 130/80 mm Hg.

Selection of initial agent is guided by the presence of compelling indications (eg, chronic stable angina, HFrEF, previous MI). B₁ blockers (with the exception of atenolol and B₁ blockers with intrinsic sympathomimetic activity), ACE inhibitors, or ARBs are all recommended as first-line therapy in these patients. For patients who remain hypertensive (BP > 130/80 mm Hg) despite treatment with a B₁ blocker, ACE inhibitor, or ARB, additional medications should be added to further reduce BP to target. The choice of agent is dependent on the presence of angina symptoms. For patients with uncontrolled

hypertension with angina, addition of a dihydropyridine calcium channel blocker (CCB) is recommended. For patients with SIHD whose angina symptoms are controlled, the addition of dihydropyridine CCBs, thiazide diuretics, and/or mineralocorticoid receptor antagonists (MRAs) may all be considered.

1.5.4.2 Antiplatelet Agents:

Platelets play a major role in the pathophysiology of ACS. Thromboxane A₂ (TXA₂) is a potent platelet activator. Aspirin inhibits cyclooxygenase, an enzyme responsible for the production of TXA₂, thereby inhibiting platelet activation and aggregation. In patients with IHD. Aspirin has been consistently shown to reduce the risk of MACE, particularly MI ⁽²²⁾.

Antiplatelet therapy with aspirin should be considered for all patients with SIHD, particularly in patients with a history of MI, in the absence of contraindications. Aspirin doses of 75 to 162 mg daily are recommended in patients with or at risk for SIHD ^(1,3,23) .

Daily doses of aspirin above 162 mg offer no additional benefit but increase bleeding risk. If aspirin is contraindicated (eg, aspirin allergy) or is not tolerated by the patient, an alternative antiplatelet agent such as clopidogrel should be considered ⁽¹⁾ .

Binding of adenosine diphosphate to the P2Y₁₂ receptors on platelets activates glycoprotein IIb/IIIa receptors leading to platelet aggregation and thrombus formation. Inhibition of the P2Y₁₂ receptor with either a thienopyridine (clopidogrel, prasugrel) or ticagrelor prevents platelet aggregation and is indicated in combination with aspirin in select patients with IHD. Ticlopidine is a P2Y₁₂ receptor inhibitor used historically in combination with aspirin, but is rarely used in practice because of hematologic toxicity. DAPT with aspirin and a P2Y₁₂ inhibitor is recommended following hospitalization for ACS and/or following PCI with stent placement to prevent ischemic events, although indications for specific drugs differ slightly. Clopidogrel is a prodrug that is converted via a two-step process to its active thiol metabolite. The cytochrome P450 (CYP) 2C19 enzyme is involved in both steps of the biotransformation. Individuals with reduced CYP2C19 activity, either from inherited deficiencies of CYP2C19 or use of CYP2C19 inhibitors (eg, proton pump inhibitors), may produce less of the active thiol metabolite. These individuals are at increased risk for stent thrombosis and MACE during clopidogrel treatment compared to those with “normal” CYP2C19 activity.

The clopidogrel labeling now warns of reduced effectiveness in CYP2C19 poor metabolizers, who carry two dysfunctional CYP2C19 gene alleles. The CYP2C19_2 and CYP2C19_3 alleles are the most common alleles leading to reduced CYP2C19 activity ⁽²⁶⁾ .

Prasugrel is a thienopyridine that also requires biotransformation to its active metabolite. However, unlike clopidogrel, CYP2C19 deficiency does not alter its effectiveness. Ticagrelor is a direct-acting P2Y₁₂ inhibitor that does not require biotransformation to exert its antiplatelet effects and is unaffected by CYP2C19 activity. Thus, prasugrel or ticagrelor may be suitable alternatives to clopidogrel when reduced CYP2C19 activity is suspected.

Antiproliferative drugs in DES reduce the risk of in-stent restenosis but delay endothelialization. Until endothelialization occurs, platelets are exposed to the foreign surface of the stent and can stimulate platelet adhesion, activation, aggregation, and eventual thrombus formation within the implanted stent resulting in acute ischemia.

Thus, a longer period of DAPT is recommended in patients with SIHD treated with DES compared with BMS to prevent in-stent thrombosis. Following BMS implantation, patients with SIHD should be treated with DAPT for a minimum of 1 month compared to a minimum of 6 months following DES implantation. In contrast to patients with SIHD, a longer duration of DAPT (minimum of 12 months) is recommended for patients who have had stent implantation in the setting of ACS, regardless of the type of stent implanted ⁽³⁾.

Select patients with IHD who have not experienced a bleeding complication on DAPT and are not at high risk for bleeding (eg, prior bleeding on DAPT, coagulopathy, oral anticoagulant use) may be considered for “prolonged” or “extended” duration of DAPT (> 1 month in SIHD with BMS, > 6 months in SIHD with DES, > 12 months in ACS with either BMS or DES) to further reduce the risk of ischemic complications, including late in-stent thrombosis, but at the expense of an increased risk of bleeding.⁽³⁾ Given the delicate balance between benefit and harm associated with prolonged DAPT following stent implantation, use of a “DAPT score” (Table 5) ^(3,27) may be useful to individualize the decision to continue DAPT by predicting patients who will derive benefit ^(3,27).

Conversely, patients who experience a significant bleeding complication, are at high risk for bleeding, or have a high risk of severe bleeding complications (eg, major intracranial surgery) may be considered for a shorter duration of DAPT (3 months for SIHD with DES, 6 months after ACS with stent implantation) ⁽³⁾. Because the risk for in-stent thrombosis is increased with premature discontinuation of DAPT, it is imperative for clinicians to educate patients on this risk, and the need for continuation of combination antiplatelet therapy for the recommended duration.

Clopidogrel, prasugrel, and ticagrelor are all indicated in combination with aspirin for at least 1 year in patients with ACS who undergo PCI ^(3,24,25). Both clopidogrel and ticagrelor are also indicated in combination with aspirin in patients with NSTEMI who do not undergo PCI. ⁽²⁴⁾ In patients with ACS, DAPT more effectively reduces the risk of death, MI, and stroke compared with aspirin alone even in patients who are managed medically (eg, in the absence of PCI). ⁽²⁸⁻³⁰⁾ Compared to clopidogrel, prasugrel and ticagrelor provide greater protection against cardiovascular events in ACS but increase risk for bleeding ^(29,30).

(Table 5)	
“DAPT” Score Calculation to Guide Prolonged Dual Antiplatelet Therapy (DAPT) Following Stent Implantation ^{(3) (27)}	
Variable	Point
Age $\geq$ 75 years	-2
Age 65 to < 75	-1
Age < 65 years	0
Current cigarette smoker	1
Diabetes mellitus	1
Myocardial infarction (MI) at presentation	1
Prior percutaneous coronary intervention (PCI) or MI	1
Stent diameter < 3 mm	1
Paclitaxel-eluting stent	1
Heart failure of left ventricular ejection fraction < 30% (0.30)	2
PCI of saphenous vein graft	1

A score of > 2 is associated with a favorable benefit:risk ratio for prolonged DAPT while a score of < 2 is associated with unfavorable benefit:risk ratio.

1.5.4.3 Statins:

Statins are the preferred drugs to lower LDL cholesterol based on their potency and efficacy in preventing cardiac events. Several studies in tens of thousands of patients with or at high-risk for SIHD demonstrate that lowering cholesterol with statins reduces the risk of MACE by 21% ⁽³¹⁾. In addition to their LDL cholesterol-lowering effect, statins likely confer additional benefits in patients with SIHD ⁽³²⁾. Prompted by evidence that patients with “normal” LDL cholesterol derive benefit from statins, studies suggest statins modulate the following characteristics thought to stabilize atherosclerotic plaques and contribute to the cardiovascular risk reduction seen with these drugs:

- Shift LDL cholesterol particle size from predominantly small, dense, highly atherogenic particles to larger, less atherogenic particles.
- Improve endothelial function leading to more effective vasoactive response of the coronary arteries.

- Prevent or inhibit inflammation by lowering C-reactive protein and other inflammatory mediators implicated in atherosclerosis.
- Possibly improve atherosclerotic plaque stability.

In summary, to control risk factors and prevent MACE, statin therapy should be considered in all patients with SIHD, particularly in those with elevated LDL cholesterol or diabetes. Statins are potent lipid-lowering agents, possess non-lipid-lowering effects that may provide additional benefit to patients with SIHD, and have been shown to reduce morbidity and mortality in patients with and at high risk for SIHD. Moreover, because statins improve outcomes in patients with SIHD and “normal” LDL cholesterol, statins should be considered in all patients with SIHD, regardless of baseline LDL cholesterol.

1.5.4.4 ACE Inhibitors and Angiotensin Receptor Blockers:

Angiotensin II, a neurohormone produced primarily in the kidney, is a potent vasoconstrictor and stimulates the production of aldosterone. Together, angiotensin II and aldosterone increase BP and sodium and water retention (increasing ventricular wall tension), cause endothelial dysfunction, promote thrombus formation, and cause myocardial fibrosis.

ACE inhibitors decrease angiotensin II production and have consistently been shown to decrease morbidity and mortality in patients with HFrEF or history of MI^(33,34). In addition, there is evidence that ACE inhibitors reduce the risk of vascular events in patients with chronic stable angina or risk factors for SIHD^(35,36). In patients with vascular disease (including SIHD) or risk factors for vascular disease, both ramipril and perindopril reduced the risk of MACE by 20% or more in separate studies^(35,36).

In the absence of contraindications, ACE inhibitors should be considered in all patients with SIHD, particularly those individuals who also have hypertension, diabetes mellitus, chronic kidney disease, left ventricular dysfunction, history of MI, or any combination of these⁽¹⁾. Additionally, ACE

inhibitors should also be considered in patients at high risk for developing SIHD based on findings from the studies summarized above. ARBs may be used in patients with indications for ACE inhibitors but who cannot tolerate them due to side effects (eg, chronic cough). ARBs also antagonize the effects of angiotensin II. In one large trial, valsartan was as effective as captopril at reducing morbidity and mortality in post-MI patients ⁽³³⁾ . However, there are far more data supporting the use of ACE inhibitors in SIHD. Therefore, ACE inhibitors are preferred over ARBs in patients with a history of MI, diabetes, chronic kidney disease, or left ventricular dysfunction. The ACE inhibitors and ARBs with indications for patients with or at risk for SIHD or IHD-related complications are listed in (Table 6) ^(33,35,36) .

Side effects with ACE inhibitors and ARBs include hyperkalemia, deterioration in renal function, and angioedema. Serum potassium increases are secondary to aldosterone inhibition and are more likely in the presence of preexisting renal impairment, diabetes, or concomitant therapy with nonsteroidal anti-inflammatory drugs (NSAIDs), potassium supplements, or potassium-sparing diuretics. Reductions in glomerular filtration may occur during ACE inhibitor or ARB initiation or up-titration due to inhibition of angiotensin II-mediated vasoconstriction of the efferent arteriole. This type of renal impairment is usually temporary and more common in patients with preexisting renal dysfunction or unilateral renal artery stenosis. Bilateral renal artery stenosis is a contraindication for ACE inhibitors and ARBs because of the risk for overt renal failure.

Angioedema is a potentially life-threatening adverse effect that occurs in less than 1% of white patients but up to 8% of African Americans treated with ACE inhibitors. Angioedema may also occur with the administration of ARBs. Patients treated with ACE inhibitors may develop a chronic cough secondary to bradykinin accumulation. For patients who develop a persistent ACE inhibitor-induced cough or mild angioedema, substitution of an ARB for an ACE inhibitor may be appropriate. Both ACE inhibitors and ARBs can cause fetal injury and death and are contraindicated in pregnancy.

(Table 6)		
Doses of Angiotensin-Converting Enzyme Inhibitors and Angiotensin Receptor Blockers Indicated in Ischemic Heart Disease (IHD) ^(33,35,36)		
Drug	Indications	Usual Dosage in IHDA
Angiotensin-Converting Enzyme Inhibitors		
Captopril	HTN, HF, post-MI, diabetic nephropathy	6.25–50 mg three times daily
Enalapril	HTN, HF	2.5–40 mg daily in one to two divided doses.
Fosinopril	HTN, HF	10–80 mg daily in one to two divided doses.
Lisinopril	HTN, HF, post-MI	2.5–40 mg daily.
Perindopril	HTN, IHD	4–8 mg daily.
Quinapril	HTN, HF, post-MI	5–20 mg twice daily.
Ramipril	HTN, high-risk for IHD, HF, post-MI	2.5–10 mg daily in one to two divided doses.
Trandolapril	HTN, HF, post-MI	1–4 mg daily.
Angiotensin Receptor Blockers		
Candesartan	HTN, HF	4–32 mg daily.
Valsartan	HTN, HF, post-MI	80–320 mg daily in one to two divided doses.
Telmisartan	HTN, high-risk for IHD	20–80 mg daily.

1.5.4.5 Nitroglycerin to Relieve Acute Symptoms:

Short-acting nitrates are first-line treatment to terminate acute episodes of angina. Patients with SIHD should have sublingual nitroglycerin tablets or spray to relieve acute ischemic symptoms. Nitrates undergo biotransformation to nitric oxide. Nitric oxide activates smooth muscle guanylate cyclase, leading to increased intracellular concentrations of cyclic guanosine monophosphate (cGMP), release of calcium from the muscle cell, and ultimately, to smooth muscle relaxation. Nitrates primarily cause venodilation, leading to reductions in preload. The resultant decrease in ventricular volume and wall tension leads to a reduction in myocardial oxygen demand. In higher doses, nitrates cause arterial dilation and reduce afterload and BP.

In addition to reducing oxygen demand, nitrates increase myocardial oxygen supply by dilating the epicardial coronary arteries and collateral vessels, as well as relieving vasospasm.

Short-acting nitrates are available in tablet and spray formulations for sublingual administration. Sublingual nitroglycerin tablets are well absorbed across the oral mucosa, produce an antianginal effect within 1 to 3 minutes, and are less expensive than the spray. However, the spray is preferred for patients who have difficulty opening the tablet container or produce insufficient saliva for rapid dissolution of sublingual tablets. At the onset of an angina attack, a 0.3 to 0.4 mg dose of nitroglycerin (tablet or spray) should be administered sublingually, and repeated every 5 minutes up to three times or until symptoms resolve. Standing enhances venous pooling and may contribute to hypotension, dizziness, or lightheadedness. Sublingual nitroglycerin may be used to prevent effort- or exertion-induced angina. In this case, the patient should use sublingual nitroglycerin 2 to 5 minutes prior to an activity known to cause angina, with the effects persisting for approximately 30 minutes. Isosorbide dinitrate, also available in sublingual form, has a longer half-life with antianginal effects lasting up to 2 hours. The use of short-acting nitrates alone, without concomitant long-acting antianginal therapy, may be acceptable for patients who experience angina symptoms once every few days. However, for patients with more frequent attacks, other antianginal therapies are recommended.

The use of nitrates within 24 to 48 hours of a phosphodiesterase type 5 inhibitor (eg, sildenafil, vardenafil, and tadalafil), commonly prescribed for erectile dysfunction, is contraindicated. Phosphodiesterase degrades cGMP, which is responsible for the vasodilatory effects of nitrates. Concomitant use of nitrates and phosphodiesterase type 5 inhibitors enhances cGMP-mediated vasodilation and can result in serious hypotension, decreased coronary perfusion, and even death. All patients with SIHD should be prescribed sublingual nitroglycerin and educated regarding its use.

1.5.5 Pharmacotherapy to Prevent Recurrent Ischemic Symptoms:

The overall goal of antianginal therapy is to allow patients with IHD to resume normal activities without symptoms of angina and to experience minimal to no adverse drug effects. The drugs used to prevent ischemic symptoms are B-blockers, CCBs, nitrates, and ranolazine. These drugs exert their antianginal effects by improving the balance between myocardial oxygen supply and demand. B-blocker, CCBs, nitrates, and ranolazine decrease the frequency of angina and delay the onset of angina during exercise. However, there is no evidence that any of these agents prevent ACS or improve survival in patients with SIHD. Combination therapy with two or three antianginal drugs is often needed.

1.5.5.1 B-blockers:

B-blockers antagonize B₁- and B₂-adrenergic receptors in the heart, reducing heart rate and cardiac contractility, and decreasing myocardial oxygen demand. B-blockers may also reduce oxygen demand by lowering BP and ventricular wall tension through inhibition of renin release from juxtaglomerular cells. By slowing heart rate, B-blockers prolong diastole, thus increasing coronary blood flow. However, B-blockers do not improve myocardial oxygen supply. B-blockers with intrinsic sympathomimetic activity (eg, acebutolol, pindolol, and penbutolol) have partial B-agonist effects and cause lesser reductions in heart rate at rest. As a result, B-blockers with intrinsic sympathomimetic activity may produce lesser reductions in myocardial oxygen demand and should be avoided in patients with SIHD. Other B-blockers appear equally effective at controlling symptoms of angina. The properties and recommended doses of various B-blockers used to prevent angina are summarized in (Table 7) ^(37,38,39). The frequency of dosing and drug cost should be taken into consideration when choosing a particular drug. Most B-blockers are available in inexpensive generic versions. B-blockers should be initiated in doses at the lower end of the usual dosing range, with titration according to symptom and hemodynamic response.

The B-blocker dose is commonly titrated to achieve the following:

- Resting heart rate between 55 and 60 beats/min.
- Maximum heart rate with exercise of 100 beats/min or less or 20 beats/min above the resting heart rate.

In the absence of contraindications, B-blockers are the preferred initial therapy to prevent symptoms of angina in patients with SIHD because of their potential cardioprotective effects (eg, after MI and/or in patients with HFrEF). The long-term effects of B-blockers on morbidity and mortality in patients with SIHD are largely unknown. Yet, B-blockers decrease the risk of reinfarction and death by 23% in patients who have suffered an MI ⁽³⁷⁾.

However, controversy exists regarding the role of B-blockers as first-line therapy for all patients with SIHD. In a recent observational study, B-blockers did not lower the risk of MACE in patients with SIHD or IHD risk factors ⁽³⁸⁾. Additionally, recommendations for using B-blockers for risk reduction in patients with SIHD or other vascular disease, in the absence of MI or heart failure, were recently downgraded ⁽³⁹⁾.

B-blockers are contraindicated in patients with severe bradycardia (heart rate < 50 beats/min) or atrioventricular (AV) conduction defects in the absence of a pacemaker. B-blockers should be used with particular caution in combination with other agents that depress AV conduction (eg, digoxin, verapamil, and diltiazem) because of the increased risk for bradycardia and heart block. Relative contraindications include asthma, bronchospastic disease, and severe depression. B1_Selective blockers are preferred in patients with asthma or chronic obstructive pulmonary disease. However, selectivity is dose dependent, and B1_selective agents may induce bronchospasm in higher doses.

There are several precautions to consider with the use of B-blockers in patients with diabetes or heart failure. All B-blockers may mask the tachycardia and tremor (but not sweating) that commonly accompany episodes of hypoglycemia in diabetes. In addition, nonselective B-blockers may alter glucose metabolism and slow recovery from hypoglycemia in insulin-dependent diabetes.

B₁-Selective agents are preferred because they are less likely to prolong recovery from hypoglycemia. Importantly, B-blockers should not be avoided in patients with SIHD and diabetes, particularly in patients with a history of MI who are at high risk for recurrent cardiovascular events. B-blockers are indicated in patients with chronic HFrEF who are euvolemic due to their mortality benefit in this population. However, B-blockers are negative inotropes (ie, they decrease cardiac contractility). Therefore, B-blockers may worsen symptoms of heart failure in patients with left ventricular dysfunction (ie, ejection fraction < 40% [0.40]) and initiation or titration should be delayed in patients with acute heart failure until symptoms have resolved. In particular, when used for the management of SIHD in a patient with heart failure, B-blockers should be initiated in very low doses with slow up-titration to avoid worsening heart failure symptoms.

Other potential adverse effects from B-blockers include fatigue, sleep disturbances, malaise, depression, and sexual dysfunction. Abrupt B-blocker withdrawal may increase the frequency and severity of angina, possibly because of increased receptor sensitivity to catecholamines after long-term B₁-blockade. If the decision is made to stop B₁-blocker therapy, the dose should be tapered over several days to weeks to avoid exacerbating angina.

(Table 7)				
Properties and Dosing of B ₁ -Blockers in Ischemic Heart Disease ^(37,38,39)				
Drug	Receptor Affinity	Usual Dose Range	Dose Adjust in Hepatic Impairment	Dose Adjust in Renal Impairment
Atenolol	B₁-Selective	25–200 mg once daily.	No	Yes
Betaxolol	B₁-Selective	5–20 mg once daily.	No	Yes
Bisoprolol	B₁-Selective	2.5–10 mg once daily.	Yes	Yes
Carvedilol	α₁, B₁ and B₂	3.125–25 mg twice daily.	Avoid in sever	No
Carvedilol phosphate	α₁, B₁ and B₂	10–80 mg once daily.	Avoid in sever	No
Labetalol	α₁, B₁ and B₂	100–400 mg twice daily.	Yes	No
Metoprolol	B₁-Selective	50–200 mg twice daily (once daily for extended release).	Yes	No
Nadolol	B₁ and B₂	40–120 mg once daily.	Yes	No
Nebivolol	B₁-Selective	5–10 mg once daily.	NO	Yes
Propranolol	B₁ and B₂	20–120 mg twice daily (60–240 mg once daily for long-acting formulation).	Avoid in sever	Yes
Timolol	B₁ and B₂	10–20 mg twice daily.	Yes	No
			Yes	Yes

1.5.5.2 Calcium Channel Blockers:

CCBs inhibit calcium entry into vascular smooth muscle and cardiac cells, resulting in the inhibition of the actin-myosin complex and contraction of the cell. Inhibition of calcium entry into the vascular smooth muscle cells leads to systemic vasodilation and reductions in afterload. Inhibition of calcium entry into the cardiac cells leads to reductions in cardiac contractility. Thus, CCBs reduce myocardial oxygen demand by lowering both wall tension (through reductions in afterload) and cardiac contractility. The nondihydropyridine CCBs, verapamil and diltiazem, slow sinoatrial and AV nodal conduction, decrease heart rate, and further decrease myocardial oxygen demand. Because of their negative chronotropic effects, verapamil and diltiazem are generally more effective antianginal agents than the dihydropyridine CCBs.

In contrast, dihydropyridine CCBs, nifedipine in particular, inhibit calcium in the vasculature and are potent vasodilators that can cause baroreflex-mediated increases in sympathetic tone and heart rate. In addition to decreasing myocardial oxygen demand, all CCBs increase myocardial oxygen supply by dilating coronary arteries, thus increasing coronary blood flow and relieving vasospasm.

CCBs are as effective as B-blockers at preventing ischemic symptoms. CCBs are recommended as alternative treatment in SIHD when B-blockers are contraindicated or not tolerated. In addition, CCBs may be used in combination with B-blockers when initial treatment is unsuccessful. The combination of a B-blocker and a dihydropyridine CCB may improve symptoms better than either drug used alone ⁽⁴⁰⁾. However, the combination of a B-blocker with either verapamil or diltiazem should be used with extreme caution because both drugs decrease AV nodal conduction, increasing the risk for severe bradycardia or AV block when used together. If combination therapy is warranted, a long-acting dihydropyridine CCB is preferred. B-blockers will prevent reflex increases in sympathetic tone and heart rate with the use of CCBs with potent vasodilatory effects. For patients with variable and unpredictable occurrences of angina, indicating possible coronary

vasospasm, CCBs may be more effective than B-blockers in preventing angina episodes. The dosing of CCBs in IHD is described in (Table 8) ⁽⁴⁰⁾. Verapamil and diltiazem are contraindicated in patients with bradycardia and preexisting conduction disease in the absence of a pacemaker. As previously noted, verapamil and diltiazem should be used with particular caution in combination with other drugs that depress AV nodal conduction (eg, B-blockers and digoxin). Because of their negative inotropic effects, CCBs may cause or exacerbate heart failure in patients with HFrEF and should be avoided in this population. The exceptions are amlodipine and felodipine that have less negative inotropic effects compared with other CCBs and appear to be safe in patients with left ventricular systolic dysfunction ^(41,42). Finally, there is some evidence that short-acting CCBs (particularly short-acting nifedipine and nicardipine) may increase the risk of cardiovascular events ⁽⁴³⁾. Therefore, short-acting agents should be avoided in the management of SIHD.

(Table 8)	
Dosing of Calcium Channel Blockers in Ischemic Heart Disease ⁽⁴⁰⁾	
Drug	Usual Dose Range
Nondihydropyridines	
Diltiazem, extended release	120–360 mg once daily; consider dose adjustment in hepatic dysfunction.
Verapamil, extended release	180–480 mg once daily; use initial dose of 120 mg in hepatic dysfunction
Dihydropyridines	
Amlodipine	5–10 mg once daily.
Felodipine	5–10 mg once daily; use initial dose of 2.5 mg once daily in hepatic dysfunction.
Nifedipine, extended release	30–90 mg once daily; dose adjust and monitor closely in hepatic dysfunction.
Nicardipine	20–40 mg thrice daily; dose twice daily in hepatic dysfunction and up-titrate slowly in both hepatic and renal dysfunction.

1.5.5.3 Ranolazine:

Ranolazine is an anti-ischemic agent indicated for the management of chronic angina. The mechanism of action is unclear, but it is believed to inhibit the late inward sodium current during the plateau phase of the cardiac action potential. Under ischemic conditions, excess sodium may enter the myocardial cell during systole. The resultant intracellular sodium overload leads to intracellular calcium accumulation (calcium overload) through a sodium/calcium exchange mechanism. Calcium overload results in increases in left ventricular wall tension and MVO_2 .

By reducing intracellular sodium concentrations in ischemic myocytes, ranolazine decreases intracellular calcium overload, left ventricular wall tension, and MVO_2 .

Similar to other antianginal drugs, ranolazine reduces angina and increases exercise capacity but does not reduce incidence of MACE. Ranolazine has minimal effects on heart rate or BP; thus, it may be an option in SIHD patients with low baseline BP or heart rate.

Ranolazine is indicated as a first-line treatment for chronic stable angina. However, it is often reserved for patients with angina refractory to other antianginal medications due to its excessive cost. Ranolazine can prolong the QT interval. However, when used at recommended doses, the mean prolongation of QT interval is minimal (2.4 milliseconds [ms])⁽⁴⁴⁾. The risk for QT interval prolongation is elevated in patients with hepatic impairment or taking other medications known to interact with ranolazine or prolong QT interval. Ranolazine should be started at a dose of 500 mg twice daily and increased to 1000 mg twice daily if needed for symptom relief. Higher doses are poorly tolerated and should be avoided. Common adverse effects with ranolazine include dizziness, constipation, headache, and nausea. Syncope may occur infrequently.

Concomitant use of ranolazine with potent CYP3A4 inhibitors (eg, ketoconazole, clarithromycin, and nelfinavir) or inducers (eg, rifampin) is contraindicated. The use of ranolazine is contraindicated in patients with significant hepatic disease.

Ranolazine is a CYP3A4 substrate, weak CYP2D6 substrate, CYP2D6 inhibitor, organic cation transporter 2 (OCT2) inhibitor, and both an inhibitor and substrate of P-glycoprotein (P-gp).

The ranolazine dose should be limited to 500 mg twice daily when combined with moderate CYP3A4 inhibitors including diltiazem and verapamil. Ranolazine should be used cautiously with P-gp inhibitors (eg, cyclosporine) and substrates (eg, digoxin).

Special Populations:

Variant Angina:

Vasospasm as the sole etiology of angina (Prinzmetal or variant angina) is relatively uncommon. As a result, treatment options are not well studied. Nevertheless, based on the pharmacology of available drugs, several recommendations can be made. First, B-blockers should be avoided in patients with variant angina because of their potential to worsen vasospasm due to unopposed B-adrenergic receptor stimulation. In contrast, both CCBs and nitrates are effective in relieving vasospasm and are preferred in the management of variant angina. Because nitrates require an 8- to 12-hour nitrate-free interval, their role as monotherapy for prophylaxis of anginal attacks due to vasospasm is limited. However, immediate-release nitroglycerin is effective at terminating acute anginal attacks due to vasospasm.

Therefore, all patients diagnosed with variant angina should be prescribed immediate-release nitroglycerin. CCBs are effective for monotherapy of variant angina. Because short-acting CCBs have been associated with increased risk of adverse cardiac events, they should be avoided ⁽⁴¹⁾. Long-acting nitrates may be added to CCB therapy if needed.

Microvascular Angina:

There are limited data on optimal therapy in patients with microvascular disease. Both ACE inhibitors and statins may produce beneficial effects on endothelial function and improve microvascular angina ⁽⁶⁾. Short-acting nitrates remain the treatment of choice for relieving acute symptoms, although they may be less effective in microvascular disease. Similar to obstructive CAD, B-blockers are first line to control symptoms of angina in patients with microvascular disease and may be more effective than CCBs and long-acting nitrates in this setting ⁽⁴⁾. Ranolazine may also produce favorable antianginal effects for patients with continued symptoms.

Elderly Patients with IHD:

Elderly patients are more likely than younger patients to have other comorbidities that may influence drug selection for the treatment of angina. As a result, polypharmacy is more common in elderly patients, increasing the risk of drug–drug interactions, and perhaps decreasing medication adherence. Additionally, elderly patients are often more susceptible to adverse effects of antianginal therapies, particularly the negative chronotropic and inotropic effects of B-blockers and CCBs. Therefore, drugs should be initiated in low doses with close monitoring of elderly patients with IHD.

Acute Coronary Syndromes:

“Acute Coronary Syndromes.” It is important to educate patients with SIHD on the signs of ACS and steps to take if signs or symptoms occur. Importantly, patients should be instructed to seek emergent care if symptoms of angina last longer than 20 to 30 minutes, do not improve after 5 minutes of using sublingual nitroglycerin, or worsen after 5 minutes of using sublingual nitroglycerin. In patients with a history of ACS, it is crucial to select appropriate pharmacotherapy to prevent recurrent ACS and death. Appropriate pharmacotherapy for patients with a history of ACS includes aspirin (often in combination with a P2Y12 inhibitor), ACE inhibitors or ARBs, B_blockers, statins, and sublingual nitroglycerin. In addition, control of cardiovascular risk factors (eg, dyslipidemia, hypertension, and diabetes) with lifestyle modifications and pharmacotherapy is critical.

1.6 Aims of the study

The present study was conducted to:

- ✿ Evaluation of Medication Use among Discharged IHD Patients at a Private and Public Hospitals in Sana’a, Yemen.
- ✿ Determine the major drug-drug interaction.
- ✿ Evaluated drugs use according to guideline (4 class).

Chapter-II

MATERIALS AND METHODS

2.1 Study area, setting, sample size and inclusion criteria

The study was conducted in the internal section of a private hospital and public hospital in Sana'a Yemen, during a period of two years (2020- 2021). The sample size was 131 patients medical chart. All patients were ≥ 20 years old of both sexes.

2.2 Study design

The study design is a retrospective cross-sectional study review to evaluation medications across discharged patients. The patient's medical charts were reviewed to obtain their medication lists. The screening tool was based on IHD Guideline 2021.

2.3 Data collection

The study reviewed patients' medical charts extracted from the files and electronic health record (EHR) database. to obtain the required data. Collected demographic data include:

(a) Age and gender, (b) lipid profile, (c) creatinine clearance, (d) heart rate, (e) systolic blood pressure (f) other chronic disease, (g) the list of discharged medications and each medication generic name, dose, frequency, dosage form, duration of medications and route of administration. The selection of these variables is based on previous similar studies.

2.4 Statistical analysis

Following data collection, data was analyzed using statistical package for social science (SPSS) version 21. Continuous variables were presented as mean and standard deviation. Categorical variables were presented as frequencies and percentages.

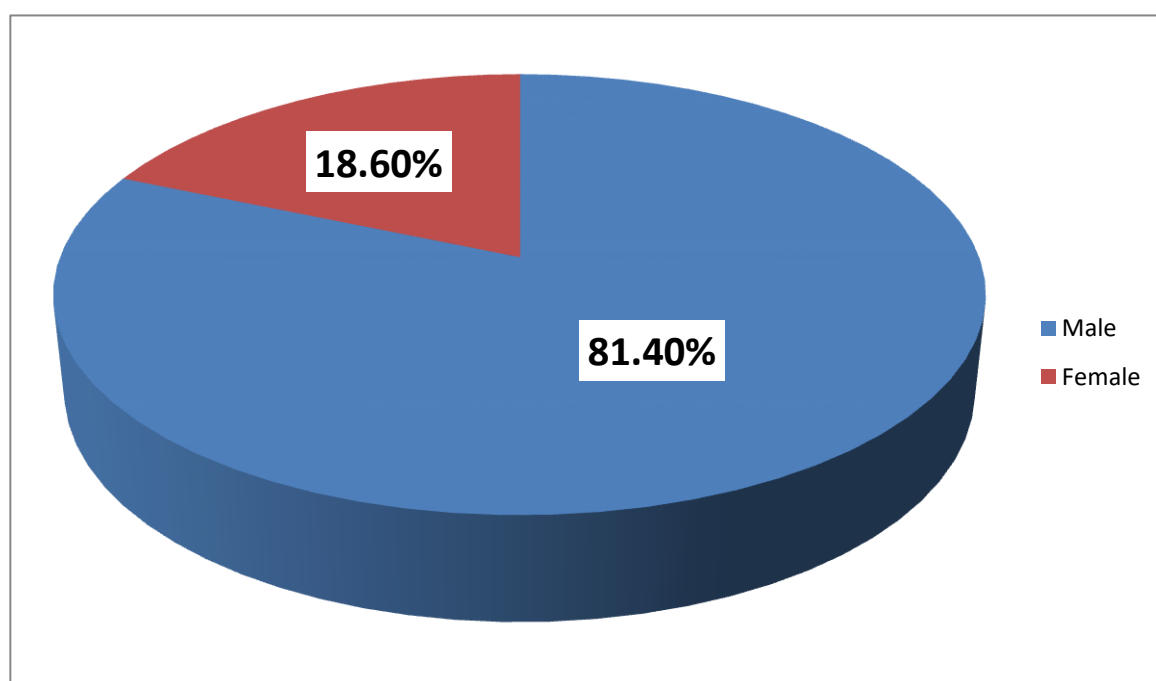
Chapter III

3.1 Study population characteristics

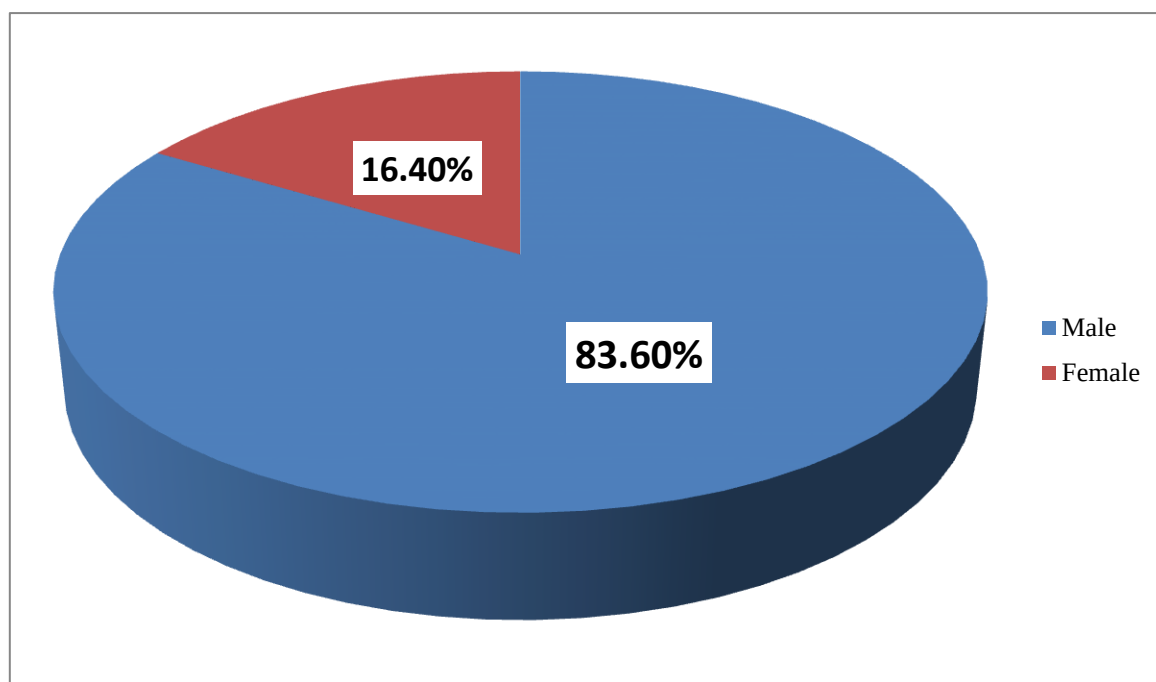
3.1.1 Gender distribution

During the period of this study a total of 131 patients records were reviewed, among which majority were males and the remaining were female [Table 9]

[Table 9] Gender distribution				
	UST		Al-Thawra	
	Frequency	Percent (%)	Frequency	Percent (%)
Male	57	81.40	51	83.60
Female	13	18.60	10	16.40
Total	70	100.0	61	100.0



[Figure 6] Gender distribution UST

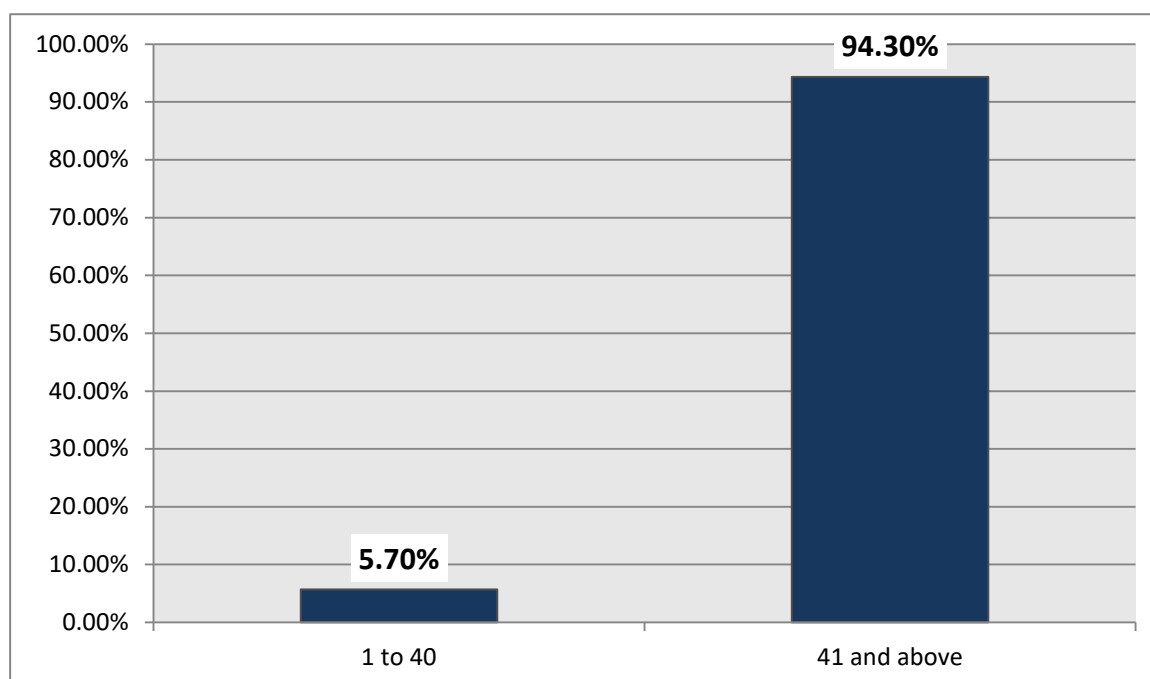


[Figure 7] Gender distribution Al-Thawra

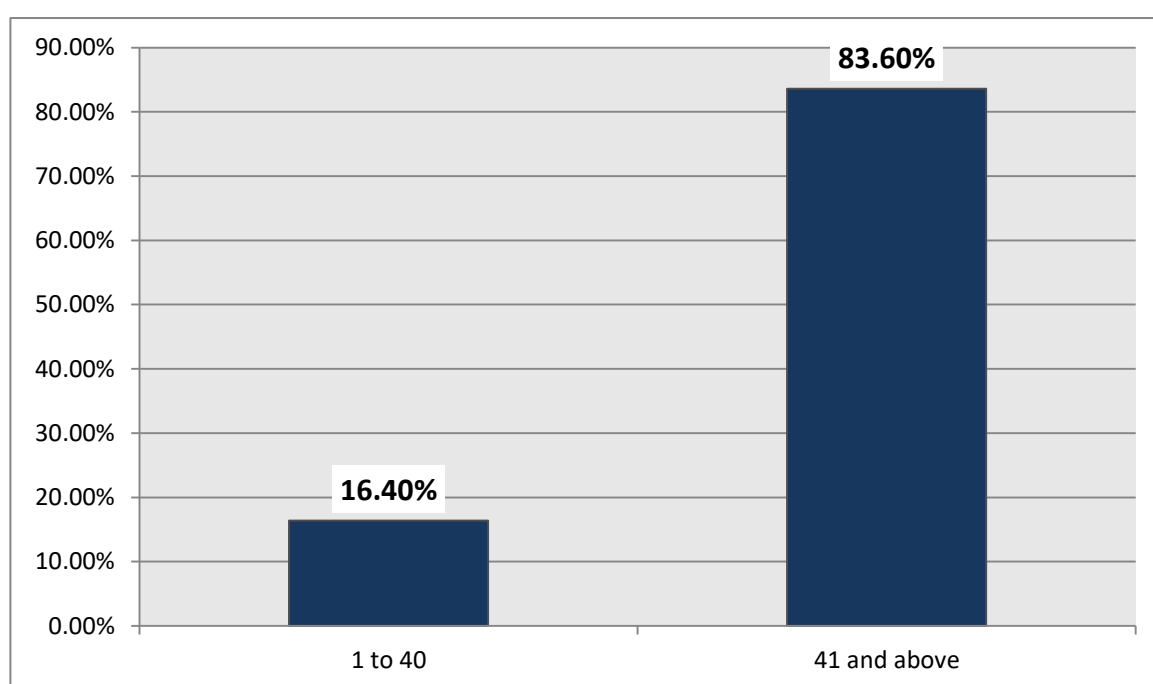
3.1.2 Age distribution

It was found that age of the patients involved varied from 24 to 80 years. The age groups were categorized into two: 1-40 years, and 41 years and above. [Table 10]

[Table 10] Age distribution				
	UST		Al-Thawra	
	Frequency	Percent (%)	Frequency	Percent (%)
1 - 40	4	5.70	10	16.40
41 and Above	66	94.30	51	83.60
Total	70	100.0	61	100.0



[Figure 8] Age distribution UST

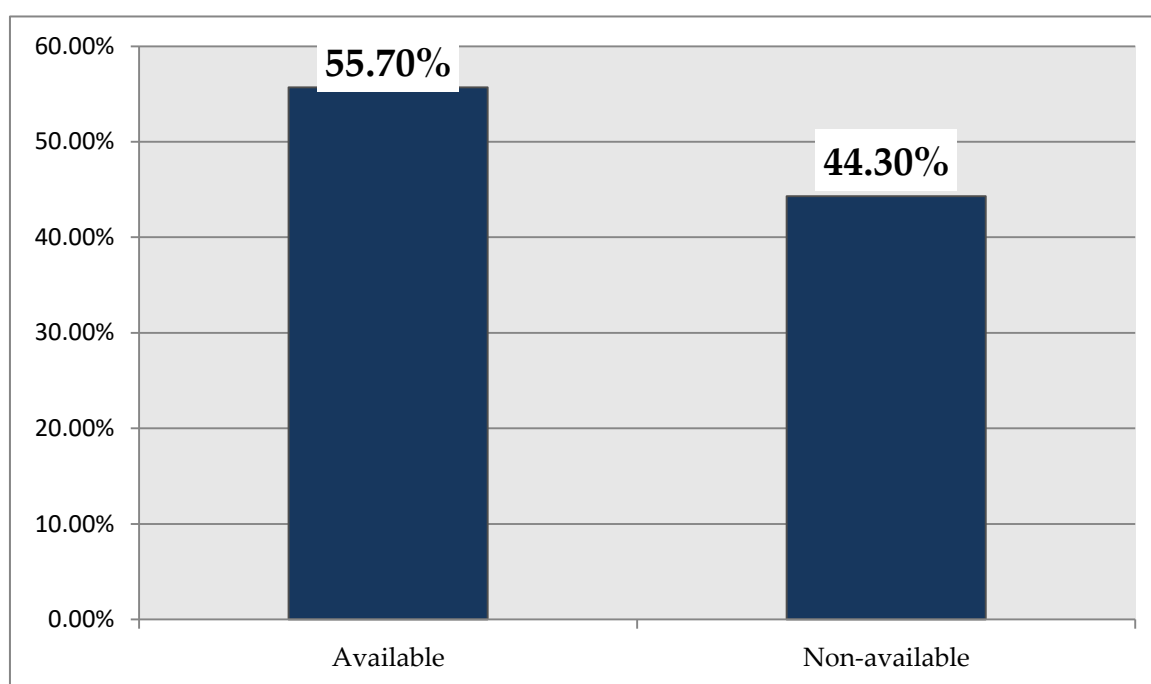


[Figure 9] Age distribution Al-Thawra

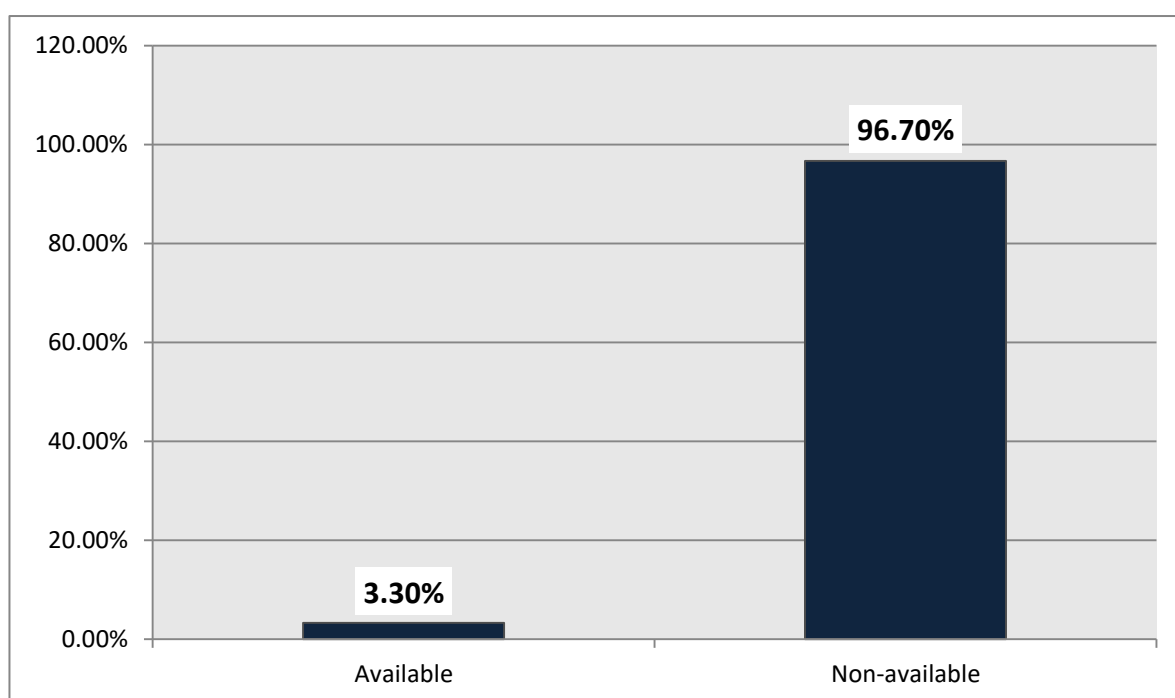
3.1.3 Lipid Profile distribution

It was found that Lipid profile of the patients ,were categorized into two: Available . and Non-available.[Table 11]

[Table 11] Lipid Profile distribution				
	UST		Al-Thawra	
	Frequency	Percent (%)	Frequency	Percent (%)
Available	39	55.70	2	3.30
Non-available	31	44.30	59	96.70
Total	70	100.0	61	100.0



[Figure 10] Lipid profile distribution UST



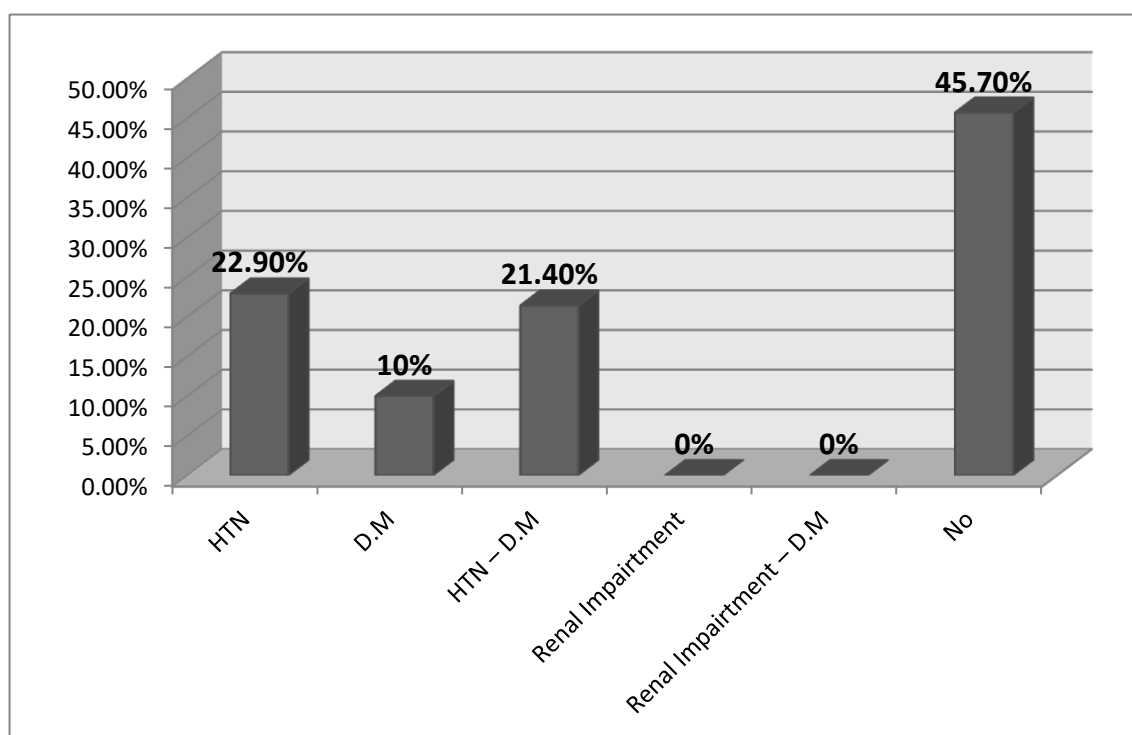
[Figure 11] Lipid profile distribution Al_Thawra

3.2 Health conditions of population

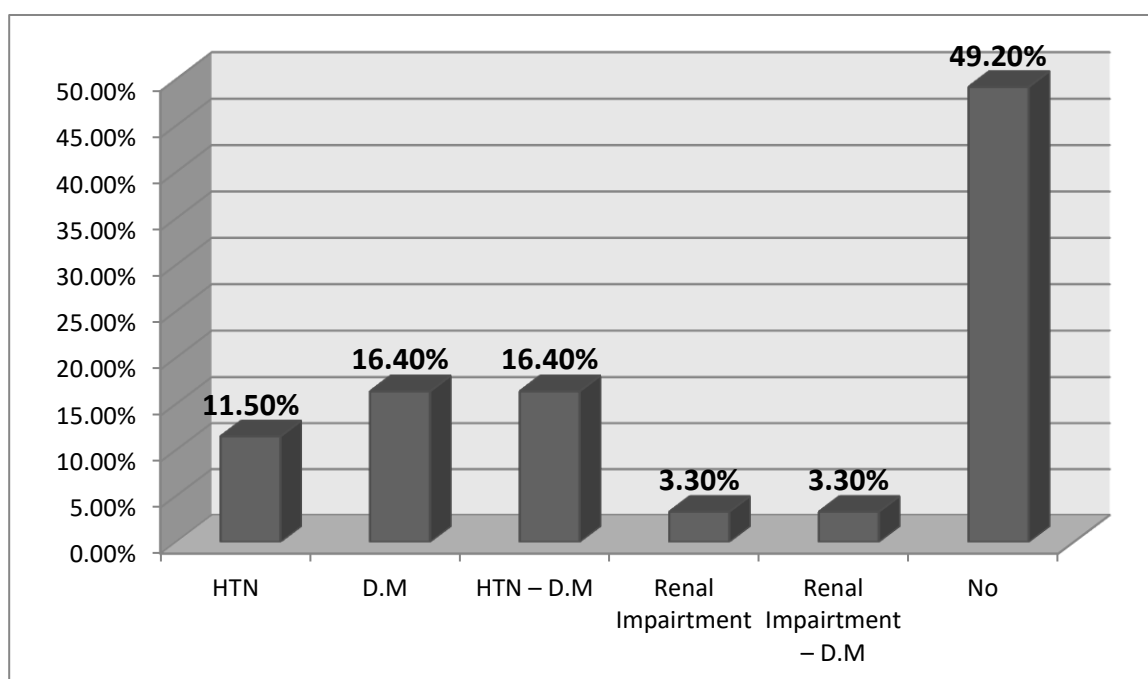
3.2.1 Morbidity pattern of the Chronic Disease patients

The major morbidity pattern was cardiovascular disease in UST (22.9%), while Al_Thawra was (11.50%) distributed between hypertension, which were the most. And (39.8%) of the morbidity included cardiovascular disease with diabetes mellitus together [Table 12].

[Table 12] Chronic Disease distribution				
	UST		Al_Thawra	
	Frequency	Percent (%)	Frequency	Percent (%)
HTN	16	22.90	7	11.50
D.M	7	10.0	10	16.40
HTN – D.M	15	21.40	10	16.40
Renal Impairment	0	0.0	2	3.30
Renal Impairment – D.M	0	0.0	2	3.30
No	32	45.70	30	49.20
Total	70	100.0	61	100.0



[Figure 12] Chronic Disease distribution UST

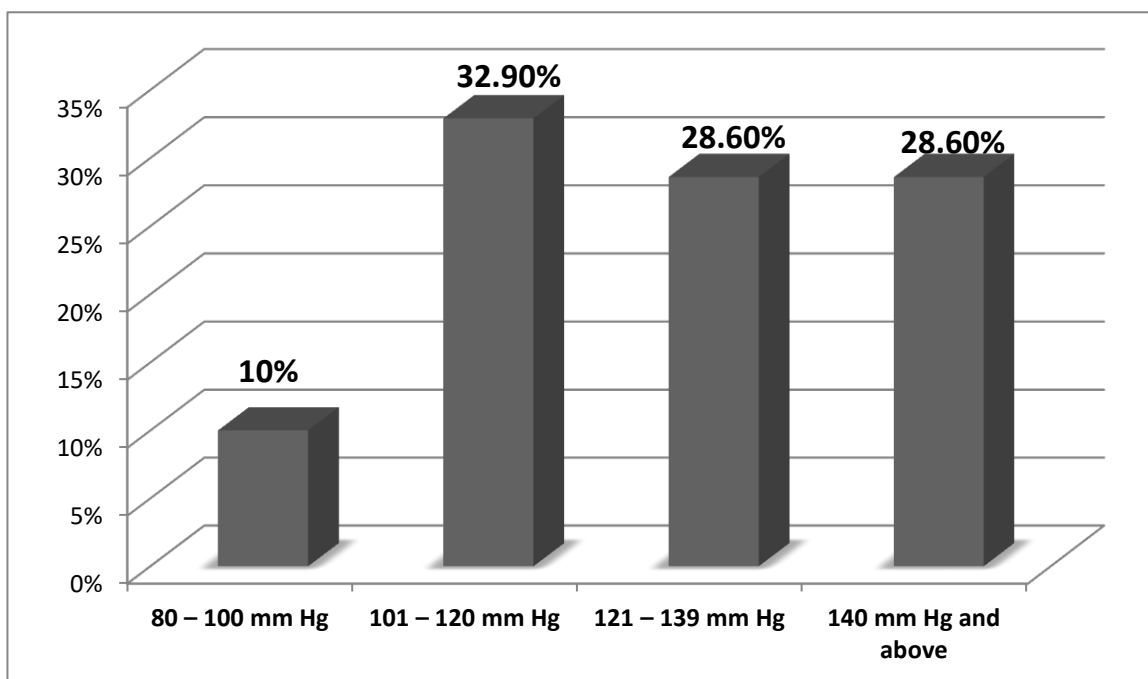


[Figure 13] Chronic Disease distribution Al-Thawra

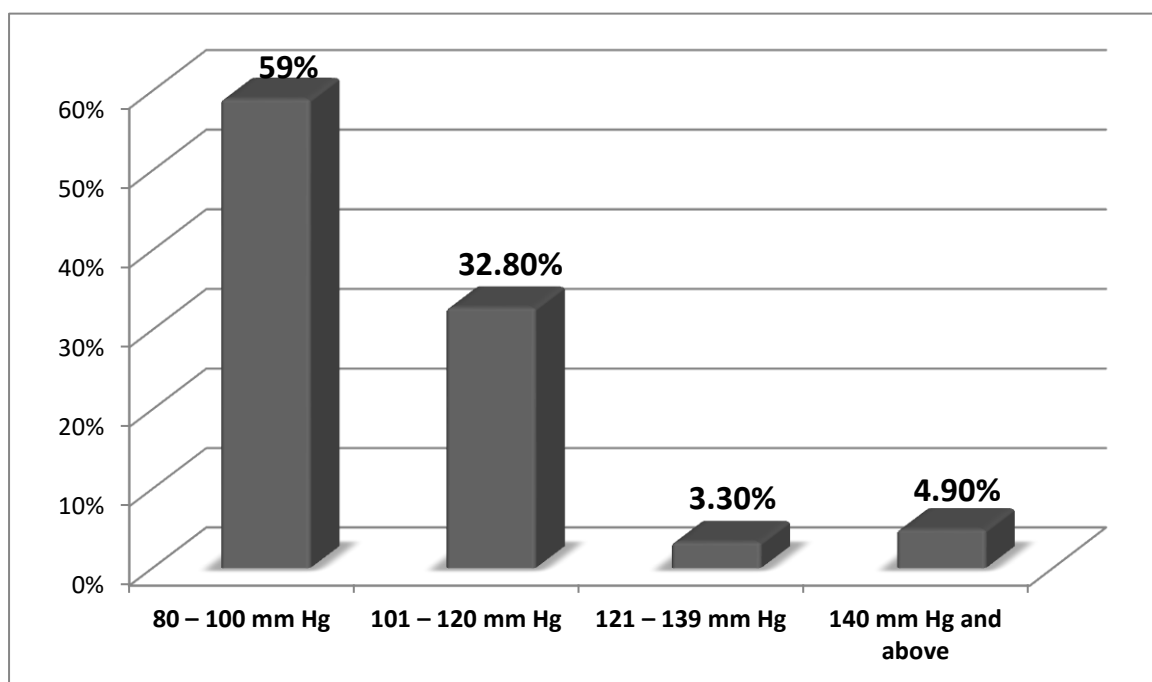
3.3 Systolic Blood pressure distribution

When were taken and follow the existing cases found that SBP of the patients involved varied from 80 to 190 mm Hg, SBP groups were categorized into four: (80-100 mm Hg) , (101-120mm Hg) , (121-139 mm Hg).And (140 mm Hg and above) [Table 13].

[Table 13] Systolic B.P distribution				
	UST		Al-Thawra	
	Frequency	Percent (%)	Frequency	Percent (%)
80 – 100 mm Hg	7	10.0	36	59.0
101 – 120 mm Hg	23	32.90	20	32.80
121 – 139 mm Hg	20	28.60	2	3.30
140 mm Hg and above	20	28.60	3	4.90
Total	70	100.0	61	100.0



[Figure 14] SBP distribution UST

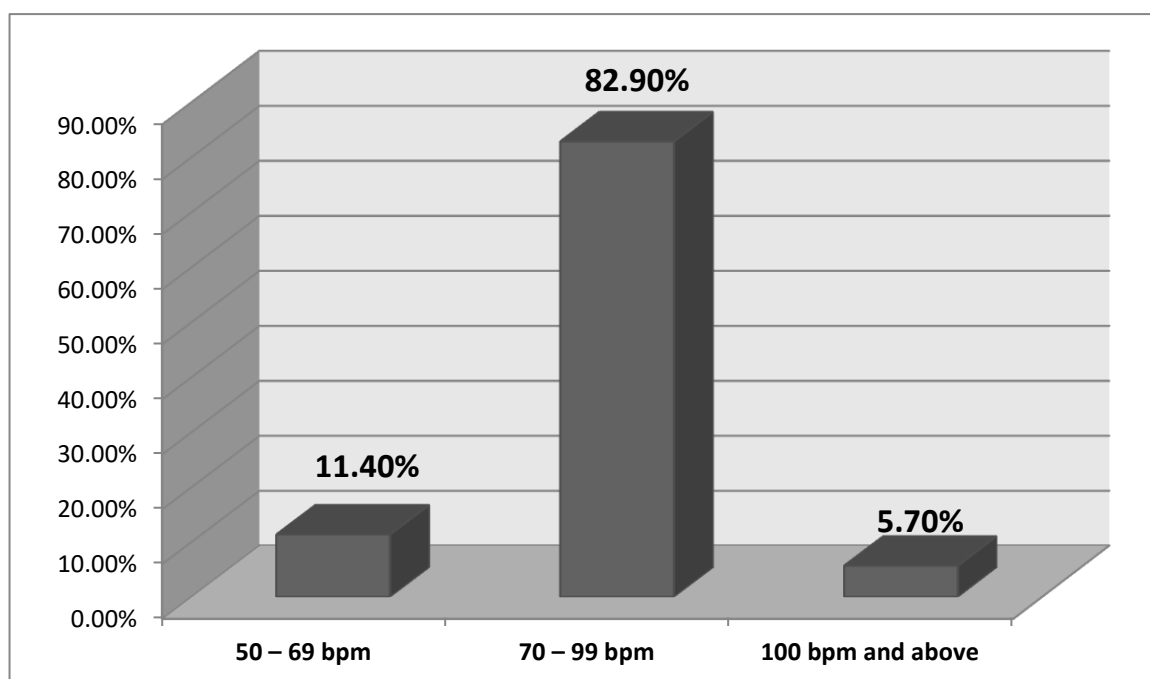


[Figure 15] SBP distribution Al-Thawra

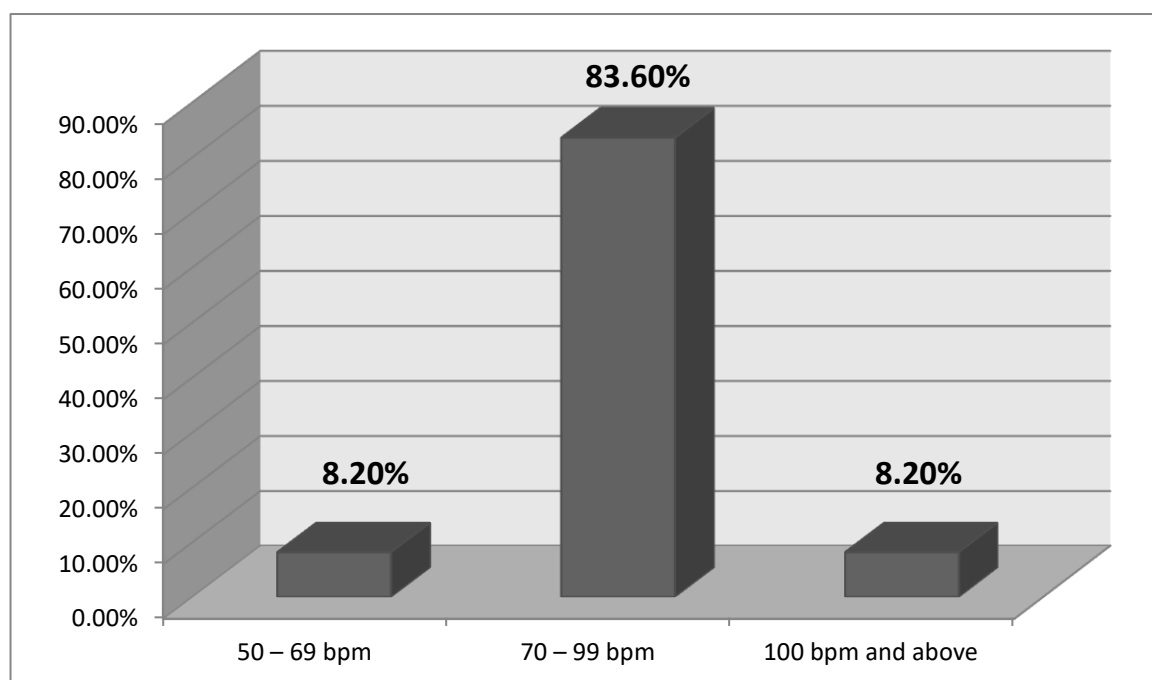
3.4 Heart Rate distribution

A total of 131 patients were prescribed of which (83.25%) were heart rate (70-99 bpm) followed by (19.6%) were heart rate (50-69 bpm), and (13.9%) were heart rate (100 bpm and above) [Table 14].

[Table 14] Heart Rate distribution				
	UST		Al-Thawra	
	Frequency	Percent (%)	Frequency	Percent (%)
50 – 69 bpm	8	11.40	5	8.20
70 – 99 bpm	58	82.90	51	83.60
100 bpm and above	4	5.70	5	8.20
Total	70	100.0	61	100.0



[Figure 16] Heart Rate distribution UST

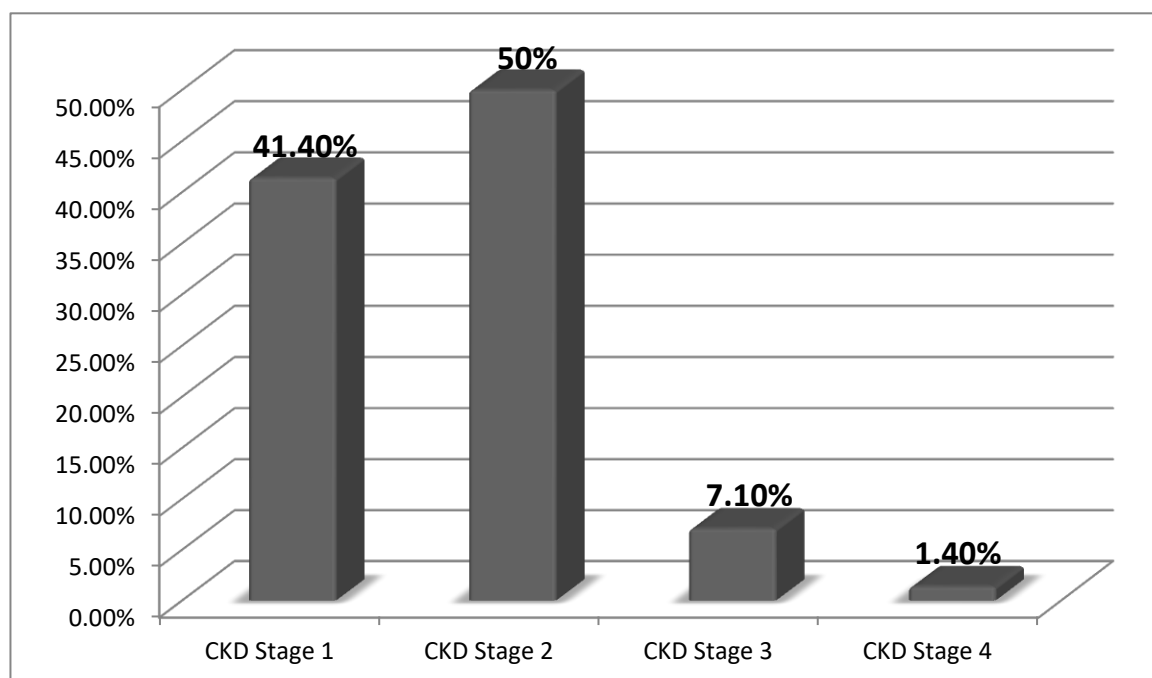


[Figure 17] HR distribution Al-Thawra

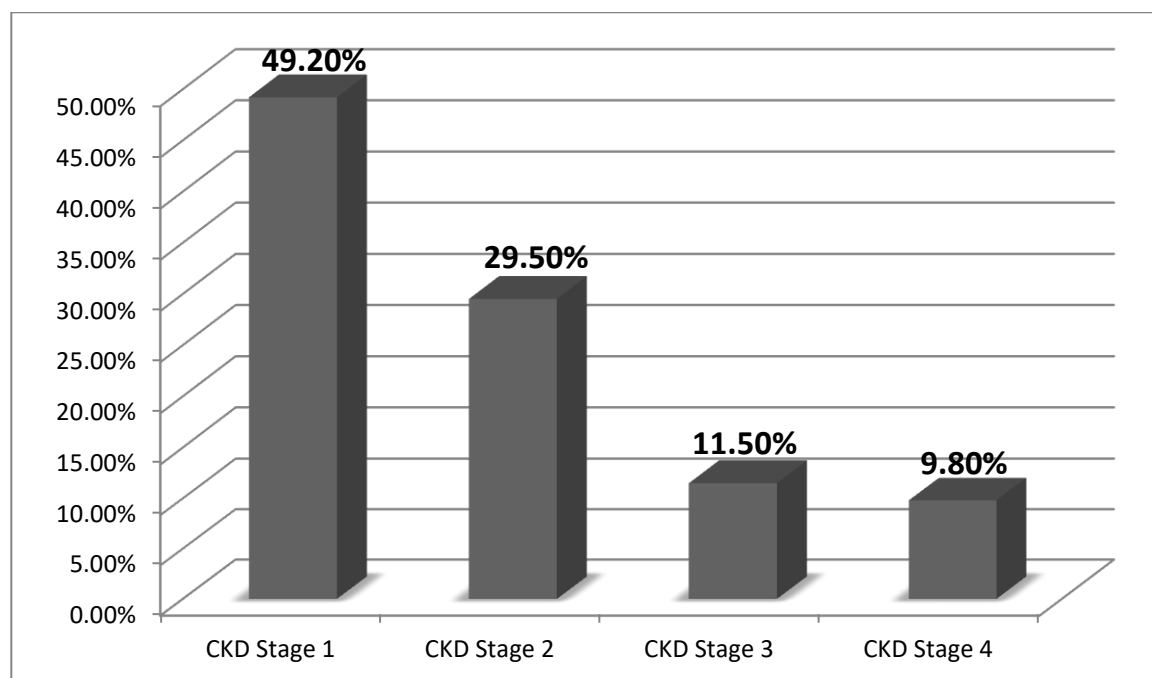
3.5 Prevalence of chronic kidney disease stages according to GFR.

Based on GFR category 131 inpatients were screened for chronic kidney disease stages and the results were as followed: [Table 15].

[Table 15] Prevalence of chronic kidney disease stages according to GFR				
	UST		Al-Thawra	
	Frequency	Percent (%)	Frequency	Percent (%)
CKD Stage 1	29	41.40	30	49.20
CKD Stage 2	35	50.0	18	29.50
CKD Stage 3	5	7.10	7	11.50
CKD Stage 4	1	1.40	6	9.80
Total	70	100.0	61	100.0



[Figure 18] CKD distribution UST

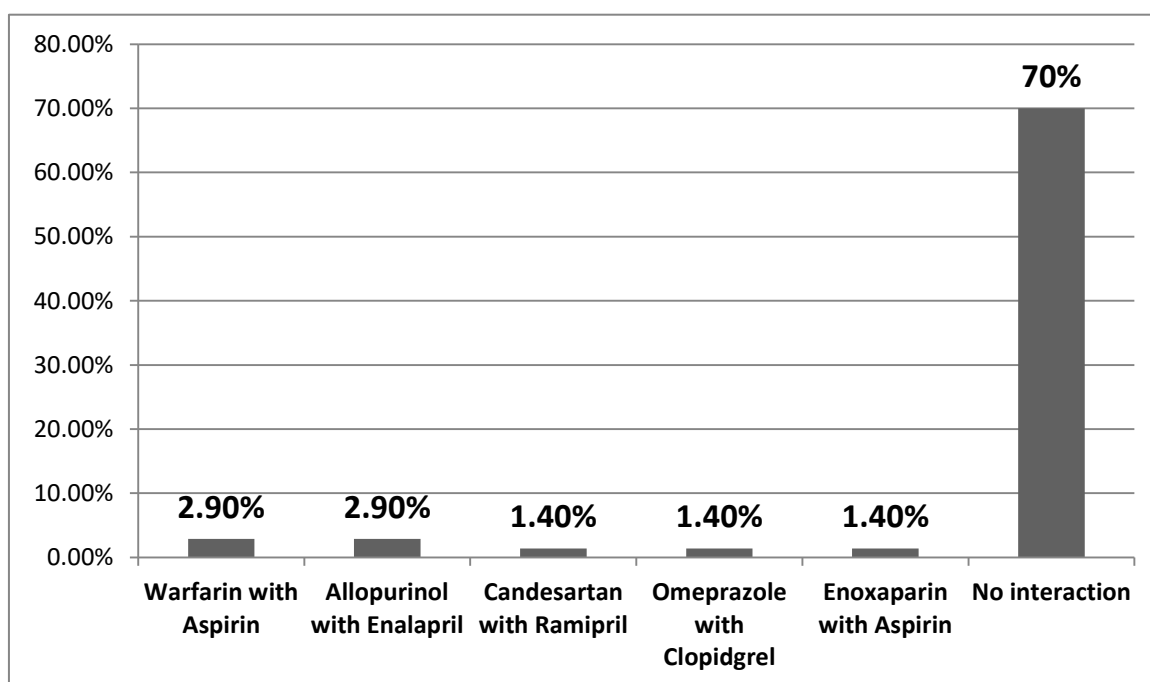


[Figure 19] CKD distribution Al-Thawra

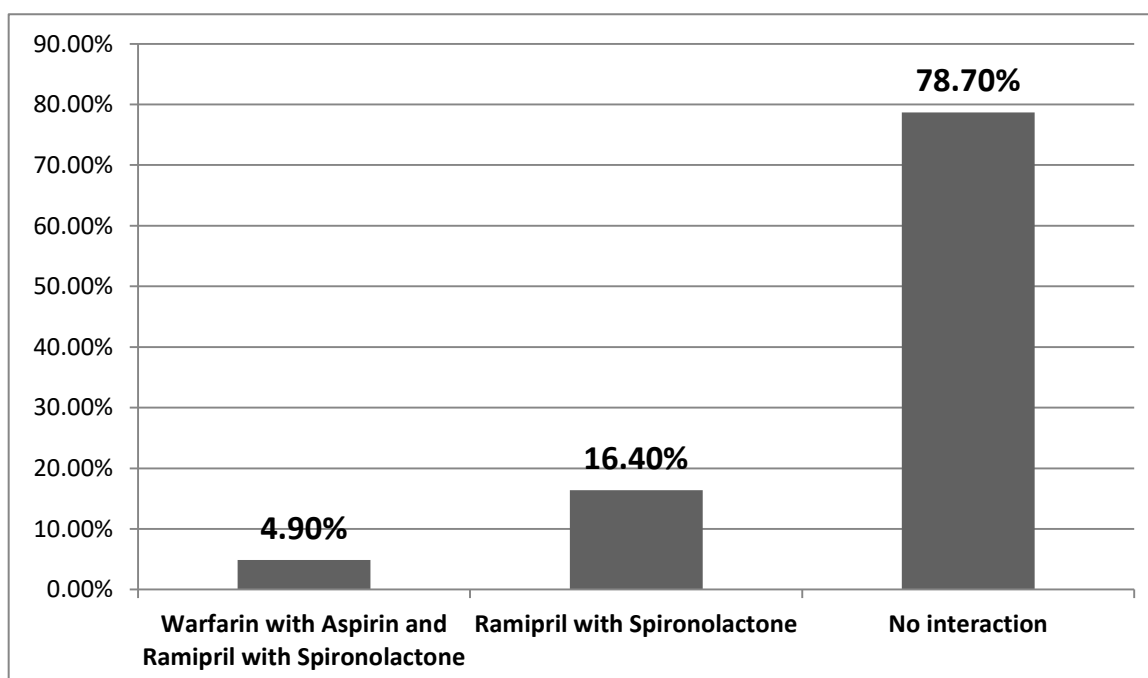
3.6 Major Drug - Drug interactions

The results also revealed that 63 (90%) of patients had no drug-drug interactions of UST hospital. While 48 (78.7%) of Al_Thawra hospital [Table 16].

[Table 16] Major Drug – Drug interactions				
	UST		Al-Thawra	
	Frequency	Percent (%)	Frequency	Percent (%)
Warfarin with Aspirin	2	2.90	0	0.0
Allopurinol with Enalapril	2	2.90	0	0.0
Candesartan with Ramipril	1	1.40	0	0.0
Omeprazole with Clopidogrel	1	1.40	0	0.0
Enoxaparin with Aspirin	1	1.40	0	0.0
Warfarin with Aspirin and Ramipril with Spironolactone	0	0.0	3	4.90%
Ramipril with Spironolactone	0	0.0	10	16.40
No interaction	63	90.0	48	78.70
Total	70	100.0	61	100.0



[Figure 20] Major D_D interaction distribution UST

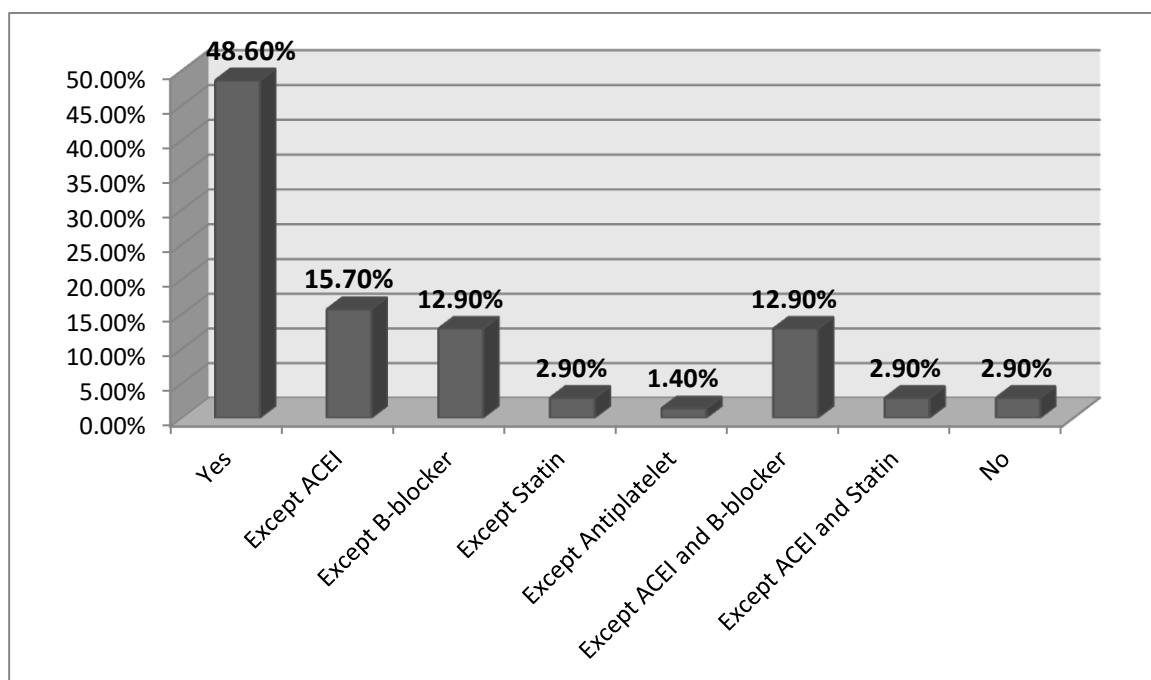


[Figure 21] Major D_D interaction distribution Al-Thawra

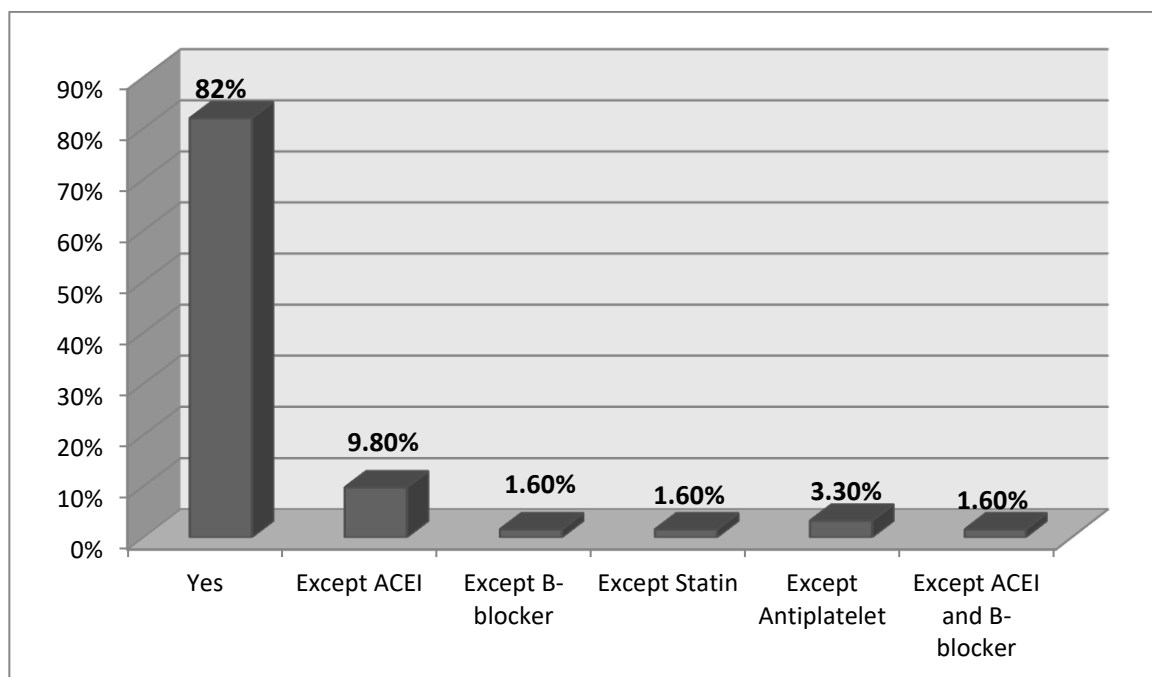
3.7 Drugs to used according to IHD Guideline 2021 (4 Class)

Based on Guideline category 131 patients were screened for drugs to be used and the results were as followed: [Table 17].

[Table 17] Drugs to used according to IHD Guideline 2021 (4 Class)				
	UST		Al-Thawra	
	Frequency	Percent (%)	Frequency	Percent (%)
Yes	34	48.60	50	82.0
Except ACEI	11	15.70	6	9.80
Except B-blocker	9	12.90	1	1.60
Except Statin	2	2.90	1	1.60
Except Antiplatelet	1	1.40	2	3.30
Except ACEI and B-blocker	9	12.90	1	1.60
Except ACEI and Statin	2	2.90	0	0.0
No	2	2.90	0	0.0
Total	70	100.0	61	100.0



[Figure 22] IHD Guideline 2021 (4 Class) distribution UST



[Figure 23] IHD Guideline 2021 (4 Class) distribution Al-Thawra

Chapter-IV

4- Discussion

This is the first Yemeni study and one of the few international studies that evaluates of medications use in discharge patients with IHD based on the latest version of guideline Study have demonstrated a enhancing prescribing of guideline recommended medication for ischemic heart disease ⁽⁴⁷⁾.

It was found that Lipid profile of the patients in UST hospital which majority were Available and the remaining were Non-available. While in Al-Thawra hospital which majority were Non-available and the remaining were available [Table 11].

In this study, the presence of certain chronic conditions. The most common morbidity resulting in hospitalization was HTN, D.M and CKD [Table 12].

The systolic blood pressure of patients in UST hospital were minimum 80 mm Hg, and maximum 190 mm Hg. While in Al-Thawra hospital were minimum 90 mm Hg, and maximum 140 mm Hg [Table 13].

Heart rate of patients in UST hospital were minimum 50 bpm, and maximum 114 bpm. While in Al-Thawra hospital were minimum 58 bpm, and maximum 110 bpm [Table 14].

For patients in this study major drug-drug interactions on discharge as mentioned in [Table 16]. In drug – drug interactions the majority of interactions found in UST hospital between Warfarin with Aspirin may cause you to bleed more easily .You need a dose adjustment based on your prothrombin time or International Normalized Ratio (INR).

And other major D_D interactions between Candesartan with Ramipril may increase the risk of side effects such as low blood pressure, kidney function impairment, and condition called hyperkalemia (high blood potassium). In sever cases, hyperkalemia can lead to kidney failure, muscle paralysis, irregular heart rhythm, and cardiac arrest. Omeprazole decreases effects of clopidogrel by affecting hepatic enzyme CYP2C19 metabolism so clopidogrel efficacy may be reduced.

At Al-Thawra hospital interaction between Ramipril with Spironolactone that using together may increase the level of potassium in your blood (hyperkalemia), especially if you are dehydrated or have kidney disease, diabetes, heart failure, or if you are an old adult. More and more drugs interaction had been found in this study and needs more focusing and care when prescribing drugs to patients.

The prevalence of drugs used based on guideline in UST hospital were 34(48.60%) yes to following guideline, 11(15.70%) were except ACEI. and 9(12.90%) were except B-blocker and except ACEI with B-blocker. While drugs used in Al-Thawra hospital 50 (82%) were yes to following guideline, 6 (9.8%) were except ACEI. Of the results the that are more hospitalized and directed were Al-Thawra hospital [Table 17] .

The role of health care providers should expand in patient to take the necessary precautions when managing IHD patient's conditions to avoid medical errors, adverse events and other misadventures associated with IHD patients. Additionally, pharmacists can play a major role in improving the appropriateness of medications use by the recommendation for either medication discontinuation, medication review, the clinical application of tools to assess patients such as guideline.

Chapter-V

5 Conclusion & Recommendation

The study showed a high prevalence of patients in private hospital were not use drugs according to guideline that should be correct and increase knowledge about use of guideline to physicians.

According to the results obtained in this work, the following recommendations are proposed:

- o The studies should be done on all departments of Yemen's hospitals.
- o Physicians should be aware and given more attention during prescribing of drugs.
- o Clinical pharmacists should be present in all branches of the hospitals in order to correct any errors.

Limitations of the study

The study is subjected to some limitations. One of the study limitations is that not all recommendation of guideline are available in the hospital formulary. Also, this study is done over two hospital only, one department and the study duration was short so, the results may be not representative of all IHD in Yemen.

This study only used guideline as the tool for evaluation medications used in discharged patients with IHD. Comparing between guideline and other validated tools in evaluated drugs used may be suggested. The reason was not used in the evaluation is the lack of sufficient information about patient history because of incomplete documentation

Principle investigator statement

I certify that the information provided in this article is accurate. I have ultimate responsibility for the conduct of the study, the ethical performance, and strict adherence to the study protocol.

Ethical approval

The hospital research ethics committee and 21 September approved this study.

Data availability statements

The data are not publicly available because of ethical restriction as it contains information that could compromise the privacy and public of patients. As per the ethical approval certificate, the data is the property of hospital.

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الخلاصة

المقدمة: يعتبر السبب الرئيسي للوفاة في جميع أنحاء العالم . على الرغم من وصفها وفقاً للقايد لان لتحسين النتائج الصحية، قمنا بتقييم ما إذا كان التدخل المستهدفة من محترفي الرعاية الصحية تعتبر فعالة لتعزيز وصف النتائج الصحية في مرضى الذين يعانون من تصلب الشرايين.

أهداف الدراسة: أجريت هذه الدراسة لتقييم الادوية المستخدمة في وصفات الخروج الطبية للمرضى في مستشفى حكومي وخاص في مدينة صنعاء – اليمن وفقاً للقايد لان ٢٠٢١ لمرضى تصلب الشرايين بالإضافة لتحديد أهم التدخلات الادوية مع بعضها.

مواد وطرق العمل: أجريت دراسة مرجعية على وصفات الخروج للمرضى، حيث تضمنت المرضى من عمر ٢٤ عاماً و ما فوق ومن كلا الجنسين. وقد جمعت البيانات المتعلقة بالدراسة من ملفات و سجلات كمبيوتر المستشفى، وتم تقييم ومراجعة وفقاً للقايد لايين ومن ثم حلت البيانات باستخدام برنامج الإحصاء الوصفي (SPSS) النسخة ٢١.

النتائج: كان اجمالي الوصفات ١٣١ وصفة طبية ل ١٣١ مريض في المستشفى خلال عامين ٢٠٢١ و ٢٠٢٠ . ١٠٨ وصفة طبية كانت للذكور بما يعادل ٨٣% و ٢٣ وصفة طبية من للإناث بما يعادل ١٧% . وقد أظهرت الدراسة نمط الأمراض الأكثر انتشارا كان مرضى ارتفاع ضغط الدم بعدد ٢٣ حالة بما يعادل ٢٣% ، و مرضى السكر بعدد ١٧ بما يعادل ٢٦.٤%، واعتمادا على القايد لايين في الادوية الموصوفة، أظهرت النتائج في المستشفى الخاص كانت بعدد ٣٤ حالة بما يعادل ٤٨.٦% من المرضى كانوا مطبقين لل ٤ اجزاء تبع للقايد لايين . بينما في المستشفى الحكومي كانت بعدد ٥٠ حالة بما يعادل ٨٢% كانوا مطبقين لل ٤ اجزاء تبع للقايد لايين. أيضا كشفت النتائج بأن ٧ المرضى بما يعادل ١٠% ظهرت في وصفات خروجهم تداخل دوائي في المستشفى الخاص. بينما في المستشفى الحكومي كشفت النتائج بأن ١٣ من المرضى بما يعادل ٢١.٣% ظهرت في وصفات خروجهم تداخل دوائي .



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والتطبيقية
كلية الصيدلة السريرية

تقييم استخدام ادوية الخروج لمرضى تصلب الشرايين في مدينة صنعاء-اليمن

إشراف

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