Prescription Survey on Cardiovascular Disease in National Institute of Cardiovascular Disease (NICVD)

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DISSERTATION

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DISSERTATION ACCEPTANCE FROM

DAFFODIL INTERNATIONAL UNIVERSITY

PHARMACY DEPARTMENT

Dedicated to……...

MY PARENTS & SISTER FOR THEIR DEEP FRIENDLINESS

Certificate

This is to certify that the results of the investigation that are embodied in this project are original and have not been submitted before in substance for any degree or diploma of this university. The entire present work submitted as a project work for the partial fulfillment of the degree of bachelor of pharmacy, is based on the result of author's (Md. Mehedi Hasan, ID NO: 111-29-256) own investigation.

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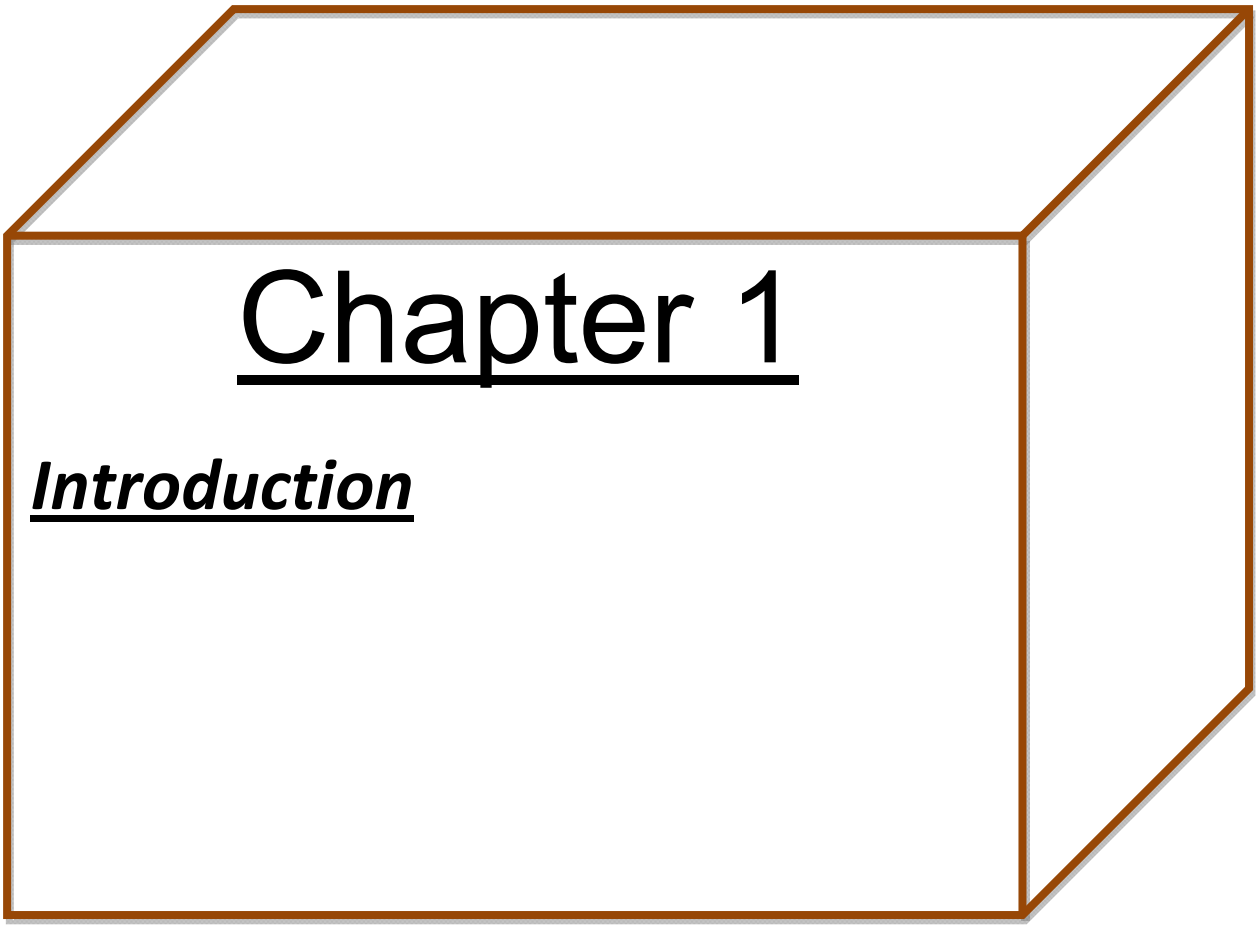
ABSTRACT

Cardiovascular drugs encompass a large number of prescription medications that are used to control heart disease. It is a complicated group of drugs with many being used for multiple heart conditions. The main objective of this survey was to analyze the prescribed cardiovascular drugs with their brand name marketed by different local and international companies of Bangladesh. This was a descriptive cross-sectional survey involving patients, physicians and pharmacy in-charge based on questionnaire with answer and followed up by prescription monitoring.

This study carried out at the outdoor of National Institute of Cardiovascular Disease (NICVD) (Shera-e- Bangla Nagar Dhaka-1207,Bangladesh) from March -20 to May-10. A total number of 219 prescription were collected among them 200 prescriptions is selected that were completely cardiac disease drugs content, 99% prescriptions were prescribed by the specialist but only 1% by general physicians.

Out of the total patients with a male, female ratio of 160:40, all patients were over 12 years and approximately 70 % the patients were demographically from urban area whereas 30% patients came from rural area. Individual patients got different classes of drugs including Beta-blockers, Organic nitrates, anticoagulant, antiplatelet and thrombolytic agents, Calcium channel blockers, Diuretics, ACE inhibitors, Lipid lowering agents and Miscellaneous agents were respectively 19.00, 26.00, 35.00,15.00,7.50,12.50 and 1.00 percent.

This study may be guidelines for optimizing rational use of cardiovascular drugs and also a new statistical approach for effective cardiovascular disease management in Bangladesh.



Chapter 1

Introduction

1. INTRODUCTION:

Cardiovascular disease is not properly curable. According to World Health Organization (WHO), cardiovascular disease (CVD) is a common cause of premature morbidity and mortality. An estimated 17.3 million people died from CVDs in 2008, representing 30% of all global deaths. Of these deaths, an estimated 7.3million were due to coronary heart disease and 6.2 million were due to stroke. By 2030, almost 25 million people will die from CVDs, mainly from heart disease and stroke. Over 80% of CVD deaths take place in low- and middle-income countries and occur almost equally in men and women.

At the same time they often do not have the benefit of prevention programs compared to people in high-income countries. A study in Bangladesh revealed that 27.93%, 21.08% and 13.41% stroke patients with lipid disorder had high cholesterol, low density lipoprotein (LDL) and triglycerides (TG) level respectively. 42.67% patients had low high density lipoprotein (HDL) level showed in the same study. Trend of cardiovascular disease management and drug use are changing day by day. In recent days, the Approach of cardiovascular disease management is more preventive than cure. For example antioxidant, Antilipidemic agents are used to reduce the factors. During this study, it was marked that cardiovascular drugs are effectively life saving one. These are projected to remain the single leading cause of death. The most prescribed drugs for the management of CVD'S in Bangladesh are Beta-adrenoceptor blocker, Organic nitrates, Anticoagulant, anti-platelet and thrombolytic drug, Calcium channel blocker, Diuretics, Renin-angiotensin system drugs, Lipid lowering drugs, Miscellaneous drugs etc. This study was conducted to compare various cardiovascular drugs prescribed by physicians in Bangladesh. The aim of this study was to compile data on prescribed marketed cardiovascular drugs by their brand names of various local and multinational companies of Bangladesh for assessment on effective cardiovascular disease management.[1]

1.1 Cardiovascular Diseases

Cardiovascular disease remains the leading killer of adult women and men globally. However, as substantial gains in reducing acute cardiovascular mortality have been realized the prevalence of persons living with cardiovascular disease has increased significantly. Without systematic access to formal and informal programs of chronic cardiovascular disease prevention such as cardiac rehabilitation, these individuals will suffer multiple recurrent acute care events and/or unnecessarily premature death.

Heart disease is a type of cardiovascular disease. In addition to heart disease, the term cardiovascular disease encompasses a variety of heart conditions, such as high blood pressure and stroke. Coronary heart disease (CHD) is caused by a narrowing of the coronary arteries, which results in a decreased supply of blood and oxygen to the heart. CHD includes myocardial infarction, commonly referred to as a heart attack, and angina pectoris, or chest pain. A heart

attack is caused by the sudden blockage of a coronary artery, usually by a blood clot. And chest pain occurs when the heart muscle does not receive enough blood. Another type of heart disease is a heart rhythm disorder, which includes rapid heart, heart murmurs, and other unspecified disorders. Congestive heart failure (CHF), which is often the end-stage of heart disease, is another disease of the heart.[2]

1.2 Different Cardiovascular disorder-

- Angina (Stable angina, unstable angina)
- Non-ST segment elevation myocardial infarction
- Chronic Rheumatic Heart Diseases
- Diastolic Murmur
- Hypertension
- Left Ventricular Failure
- Chronic Obstructive Pulmonary Diseases.
- Ischemic (or ischemic) heart disease
- Acute myeloid leukemia (AML)
- Cerebrovascular disease (Ischemic Stroke)

1.3 Angina (Stable angina, unstable angina)

What is angina?

Angina–also sometimes called angina pectoris—is a symptom of an underlying heart condition. It means that the heart is not getting enough blood and as a result, not enough oxygen. This decrease of oxygen being delivered to the muscle of the heart happens if one or more coronary arteries are narrowed or blocked, a condition called atherosclerosis.

This type of blockage may result in chest pain. And while angina does not usually damage the heart, and the pain might only last a few minutes, it is a warning sign that you should not ignore. Your body is telling you that your risk for a heart attack or cardiac arrest is increased. Very simply, angina is your heart's way of getting your attention.

An angina attack is not the same as a heart attack, although many of the symptoms are the same. An angina attack may be provoked by extremes in emotion (being very angry or upset), eating a large meal or eating it very quickly, doing more exercise than usual (overexerting yourself), being exposed to extremes in temperature (too hot or too cold), or smoking. If the angina is a result of physical activity, stopping the activity generally stops the pain. But no matter what the cause of the chest pain or discomfort, it is important that you get medical attention as soon as possible. [2]

1.3.1 There are several different kinds of angina. These are explained here.

Different types of angina

Five different kinds of angina have been identified, with the two most common being stable angina and unstable angina.

Stable angina

Stable angina occurs when the heart has to work harder than normal, during exercise, for example. It has a regular pattern, and if you already know that you have stable angina, you will be able to predict the pattern. Once you stop exercising, or take medication (usually nitroglycerin) the pain goes away, usually within a few minutes.

Un Stable angina

Unstable angina is more serious, and may be a sign that a heart attack could happen soon. There is no predictable pattern to this kind of angina; it can just as easily occur during exercise as it can while you are resting. It should always be treated as an emergency. People with unstable angina are at increased risk for heart attacks, cardiac arrest, or severe cardiac arrhythmias (irregular heartbeat or abnormal heart rhythm).[2]

Less common kinds of angina include:

• Variant angina

• Microvascular angina

• Atypical angina

Variant angina is also known as Prinzmetal's angina. It often occurs while someone is resting (usually between midnight and 8:00 in the morning), and it has no predictable pattern—that is, it is not brought on by exercise or emotion. This kind of angina may cause severe pain, and is usually the result of a spasm in a coronary artery. [2]

Micro vascular angina

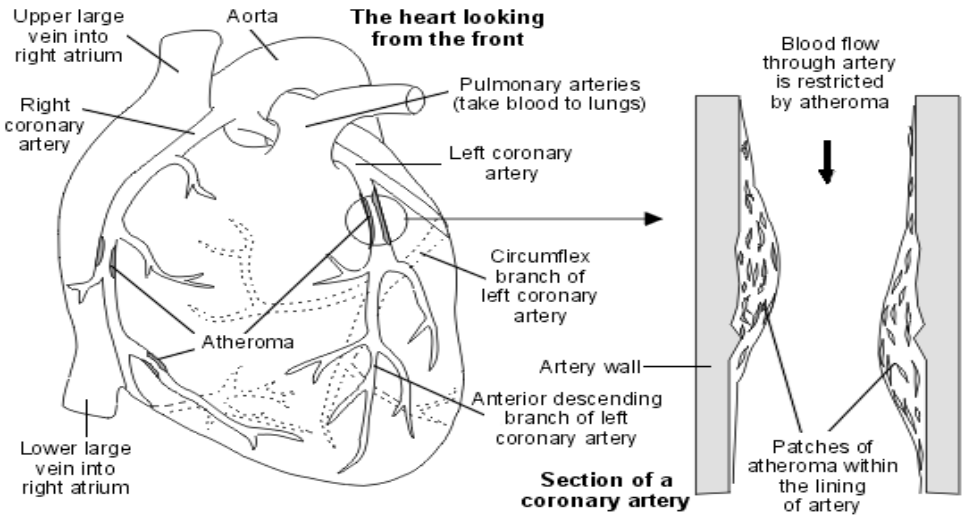
Micro vascular angina sometimes referred to as Syndrome X—occurs when tiny vessels in the heart become narrow and stop functioning properly, even if the bigger arteries are not blocked by plaque. Usually it is treated with common angina medications.

Atypical angina

Atypical angina often doesn't cause pain, but you may feel a vague discomfort in your chest, experience shortness of breath, feel tired or nauseous, have indigestion, or pain in your back or neck. Women are more likely than men to have feelings of vague chest discomfort.

In order to understand what causes angina, it might be helpful to first understand a little bit about how your heart works.[3]

1.3.2 What causes angina?



If you have angina, one or more of your coronary arteries is usually narrowed. This causes a reduced blood supply to a part, or parts, of your heart muscle. The blood supply may be good enough when you are resting. When your heart works harder (when you walk fast or climb stairs and your heart rate increases) your heart muscle needs more blood and oxygen. If the extra blood that your heart needs cannot get past the narrowed coronary arteries, the heart responds with pain.The narrowing of the arteries is caused by atheroma. Atheroma is like fatty patches or plaques that develop within the inside lining of arteries. (This is similar to water pipes that get furred up with limescale.) Plaques of atheroma may gradually form over a number of years. They may be in one or more places in the coronary arteries. In time these can become bigger and cause enough narrowing of one or more of the arteries to cause symptoms. The diagram shows three narrowed sections as an example, but atheroma can develop in any section of the coronary arteries.[3]

1.3.3 What treatments are available for angina?

You may have heard people talk about nitrates (nitroglycerin is the most common of these) or know someone who is taking them. This class of medications is the most commonly prescribed for the treatment of angina, and they work by relaxing and widening your blood vessels so that your blood can flow to your heart more easily, and so that your heart doesn't have to work as hard to keep the flow steady.

Nitrates, as discussed above, are available in a number of different forms and your doctor will determine which one is best for you. Headache is a possible side effect. You should not use medications for erectile dysfunction like Viagra for example.

Beta-blockers are a class of medicines used to treat several kinds of heart disease. They work by lowering blood pressure, and slowing your heart rate which means your heart doesn't have to work as hard.

Calcium channel blockers or **calcium antagonists** also work by lowering blood pressure and slowing your heart rate, and are often used if you cannot take a beta-blocker. They may be useful to treat coronary artery spasm.

Antiplatelet medications are blood thinners that work by preventing blood clots from forming and blocking your arteries. The most commonly used antiplatelet medication is aspirin, which works by preventing platelets from sticking to blood vessel walls. An enteric-coated aspirin is generally recommended because it is easier on the stomach. Other medications can be used to stop platelets from sticking together. They may be used to reduce the risk of clot-induced heart attacks or strokes.[4]

1.4 Myocardial infarction

Myocardial infarction is a medical term, commonly known as a heart attack that occurs when a portion of the heart muscle (myocardium) is damaged because of sudden occlusion of one of the coronary arteries that supply the oxygen rich blood to the heart muscle.

1.4.1 Pathophysiology of myocardial infarction:

An acute myocardial infarction occurs by developing a thrombus also called blood clot in a coronary artery previously affected by atherosclerosis. Cholesterol deposition in the wall of the artery is the main mechanism of atherosclerosis. This deposited cholesterol ultimately forms a plaque in the wall of the artery called atherosclerotic plaque. Many years are required to establish a plaque. Sometimes atherosclerotic plaque may rupture or erode and can trigger clotting mechanism in the blood to form a blood clot. This blood clot impaired blood flow to heart muscle lasts long enough to damage myocardial cells.[5]

1.4.2 Types of myocardial infarction:

1.4.3 ST-segment elevation myocardial infarction (STEMI):

It occurs by complete occlusion of a major coronary artery that produces an entire thickness damage of heart muscle. STEMI is also called transmural infarction due to its full thickness involvement. This entire thickness damage of heart muscle produces an ECG (electrocardiography) change of ST-segment elevation. It can be sub classified into anterior, antero-septal, posterior, inferior, lateral, high lateral or antero- lateral myocardial infarction (according to left ventricular wall damage), and RV type (according to right ventricular wall damage).

1.4.4 Non ST-segment elevation myocardial infarction (NSTEMI):

NSTEMI is usually due to complete occlusion of a minor coronary artery or partial occlusion of a major coronary artery that produces a partial thickness damage of heart muscle. Here, the damage of heart muscle is confined to the inner ⅓ rd – ⅔ rd of the left ventricular wall. For this reason, it is also called subendocardial infarction. ST-segment elevation in ECG is not developed in this myocardial infarction because of partial thickness damage of heart muscle. Here, this muscle damage is demonstrated by an elevation of cardiac markers (CK-MB or Troponin) in the blood.[5]

1.4.5 Symptoms of myocardial infarction:

Chest pain: Chest pain is the cardinal symptom of acute myocardial infarction. Pain is constricting, choking, squeezing or heavy in character, develops gradually over several minutes, usually located in the center of the chest, but may radiate to neck, jaw, shoulder, back, and arms (most commonly left arm). Occasionally, pain may be felt only at the sites of radiation. In older patient patients or those with diabetes mellitus, painless myocardial infarction may occur (also called silent myocardial infarction). Pain conducting nerve fiber is degenerated (autonomic neuropathy) in old age and in diabetes.

Shortness of breath: It may develop due to ischemic left ventricular dysfunction or dynamic mitral regurgitation.

Nausea, vomiting, and sweating: Due to upset of autonomic nervous system.

Syncope (sudden loss of consciousness): Sometimes patients may present with syncope, usually due to an arrhythmia or severe hypotension.

Tachycardia (high pulse rate): Due to sympathetic nerve activation.

Bradycardia (low pulse rate): Patients with inferior myocardial infarction may present with bradycardia due to vagus nerve activation.

Carcinogenic shock: Some patients may present with shock due to impaired myocardial function.[5]

1.4.6 Treatment of myocardial infarction:

A myocardial infarction requires immediate hospitalization and medical attention.

Basic treatment:

(1) Complete bed rest with continuous monitoring by ECG.

(2) Inhaled oxygen therapy.

(3) Aspirin and clopidogrel

A 300 mg tablet of aspirin is given orally as early as possible. It can be given intravenously to patients who cannot swallow or who are unconscious. Aspirin reduces the mortality rate of

myocardial infarction by approximately 25%. In combination of aspirin, clopidogrel 300 mg should be given orally as early as possible. Small mortality benefit is seen in combination of aspirin and clopidogrel. Ticagrelor 150 mg orally may be given instead of clopidogrel. Aspirin, clopidogrel and ticagrelor all are antiplatelet drugs that prevent the aggregation of platelets within the blood vessels.

Pain relief

Nitrate and opiate analgegic are used to relief pain. Nitrate act as a vasodilator and relief pain. Nitrate should first be given buccally or by sublingual (under tongue) spray. If the patient experiencing persistent ischemic chest pain after 3 doses given 5 minutes apart, then intravenous glyceryl trinitrate 0.6-1.2 mg/hour or isosorbide dinitrate 1-2 mg/hour can be given until pain relieved or systolic blood pressure falls to less than 100 mgHg. Intravenous opiate analgegic such as morphine 10 mg or diamorphine 5 mg is usually used for severe pain and may have to be repeated.

Treatment to restore blood supply in the occluded coronary artery (re-perfusion therapy):

The part of the heart muscle does not die immediately after occlusion of coronary artery. If blood supply is restored within a few hours, much of the affected area of the heart muscle will be saved from damaging. There are two therapies that can restore blood supply back through the occluded coronary artery:

Primary percutaneous coronary intervention (PCI):

It is treatment of choice for ST-segment elevation myocardial infarction. It should be done within 120 minutes of symptoms starting. Where PCI is not available or primary PCI cannot be achieved within 120 minutes of diagnosis, thrombolytic therapy should be administered.

On the other hand, medium to high risk patients with non-ST segment elevation myocardial infarction should be considered for early coronary angiography and revascularization, either by PCI (percutaneous coronary intervention) or by CABG (coronary artery bypass grafting). Early medical treatment is appropriate in low risk patients, and coronary angiography and revascularization reserved for those who fail to settle with medical treatment. (By using grace score or timi score, patients with non-ST segment elevation myocardial infarction can be categorized into low, medium or high risk group).[5]

Thrombolytic therapy:

It is only indicated for ST-segment elevation myocardial infarction. It is also called clot-busting drug. Available clot-busting drugs are streptokinase, alteplase, tenecteplase and reteplase. These drugs break the blood clot within the coronary artery and clear lumen. It should be given within three hours of onset of chest pain but may be given in patients who present within 12 hours of pain. After 12 hours, thrombolytic therapy appears to be a little benefit and may be harmful. In non-ST segment elevation myocardial infarction, this therapy is totally harmful and should be avoided.

Antiplatelet therapy:

Aspirin 75 mg daily should be given indefinitely if there are no side effects occur. Clopidogrel 75 mg daily should be given orally in combination of aspirin. Both drugs inhibit platelet aggregation and prevent further occlusion. Ticagrelor 90 mg two times daily may be given instead of clopidegrol. Ticagrelor is more effective than clopidegrol. Patients who undergo percutaneous coronary intervention, glycoprotein receptor blocking drug such as tirofiban, abciximab, or eptifibatide are the best antiplatet drug.

Anticoagulant therapy:

Anticoagulant therapy is recommended in patients not receiving reperfusion therapy. This therapy prevents reinfarction, and reduces the risk of thromboembolic complications. Anticoagulation can be achieved by using unfractionated heparin, low molecular weight heparin or fractionated heparin (enoxaparin, dalteparin), or a penta saccharine (fondaparinux). Comparatively low molecular weight heparin is more safety and efficacious than unfractionated heparin, and pentasaccharide is more safety and efficacious than low molecular weight heparin. The dose regimens are:

- **Enoxaparin:** 1 mg/kg body weight two times daily usually for 8 days by subcutaneous injection.
- **Dalteparin:** 120 units/kg body weight two times daily usually for 8 days by subcutaneous injection.
- **Fondaparinux:** 2.5 mg daily usually for 8 days by subcutaneous injection.

Beta-blocker:

Beta-blocker diminishes myocardial oxygen demand by reducing heart rate, blood pressure and myocardial contractility, and reduces chest pain and ventricular arrhythmias. Reduction in heart rate prolongs the diastolic period of the heart and may augment blood flow to the heart muscle. In patients not receiving thrombolytic therapy, early beta-blocker reduces in infarct size and mortality rate. In patients receiving thrombolytic therapy, recent trials have not found a mortality rate reduction, although recurrent ischemia and re-infarction rates were reduced. Oral beta-blocker atenolol 25-50 mg twice daily, metoprolol25-50 mg twice daily, or bisoprolol 5 mg once daily are usually adequate. Beta-blockers should be avoided if there is heart failure, heart block, hypotension, or bradycardia.[5]

Nitrate:

Oral or sublingual nitrate can be used if patients feel chest pain.

Statin:

Irrespective of serum cholesterol level, all patients should receive statin such as atorvastatin, rosuvastatin, simvastatin or pitavastatin.

ACE (angiotensin converting enzyme) inhibitor or ARB (angiotensive receptor blocker):

An ACE inhibitor such as ramipril, enalapril, captopril, or lisinopril is started 1 or 2 days after myocardial infarction. ACE inhibitor therapy reduces ventricular remodeling, prevent the onset of heart failure, and reduce recurrent infarction. An ARB (valsartan, candesartan, losartan or olmesartan) is suitable alternative in patients intolerant of ACE inhibitor.[5]

1.5.1 What is Ischemic Heart Disease and Stroke?

Ischemia means a "reduced blood supply". Ischemic Heart Disease (IHD) is where a waxy substance called plaque builds up inside blood vessels, and restricts the normal flow of blood. When plaque builds up in the arteries, the condition is called atherosclerosis. Atherosclerosis can affect any artery in the body, including arteries in the heart, brain, arms, legs, pelvis, and kidneys. As a result, different diseases may develop based on which arteries are affected. Ischemic Vascular Disease (IVD) is a term that includes a group of diseases caused by the build-up of plaque. Ischemic heart disease is where atherosclosis affects the coronary arteries in the heart. If the flow of oxygen-rich blood to your heart muscle is reduced or blocked, angina or a heart attack may occur. Angina is chest pain or discomfort. It may feel like pressure or squeezing in your chest. The pain also may occur in your shoulders, arms, neck, jaw, or back. Angina pain may even feel like indigestion. A heart attack, also know at myocardial infarction occurs if the flow of oxygen-rich blood to a section of heart muscle suddenly becomes blocked. If blood flow isn't restored quickly, the section of heart muscle begins to die. Without quick treatment; a heart attack can lead to serious problems and even death.[6]

1.5.2 Stroke Caused by Blocked Arteries

A frequent cause of stroke is plaque building up in the arteries on each side of your neck (the carotid arteries). These arteries supply oxygen-rich blood to your brain. If blood flow to your brain is reduced or blocked, even for a few minutes, the lack of oxygen may cause damage, or even death of brain cells. If brain cells die or are damaged because of a stroke, symptoms occur in the parts of the body that these brain cells control. Examples of stroke symptoms include

Sudden weakness; paralysis or numbness of the face, arms, or legs (paralysis is an inability to move); trouble speaking or understanding speech; and trouble seeing.

1.5.3 What Causes Ischemic Heart Disease?

All types of ischemic heart disease are caused by atherosclerosis. Atherosclerosis may start when certain factors damage the inner layers of the arteries. These factors include:

- Smoking
- High amounts of certain fats and cholesterol in the blood
- High blood pressure
- High amounts of sugar in the blood due to insulin resistance or diabetes

1.5.4 What Are the Signs and Symptoms of Ischemic Heart Disease?

Ischemic heart disease usually doesn't cause signs and symptoms until it severely narrows or totally blocks an artery. Many people don't know they have the disease until they have a medical emergency, such as a heart attack or stroke. Some people may have signs and symptoms of the disease. Signs and symptoms will depend on which arteries are affected.[6]

1.5.5 How Is Ischemic Heart Disease Treated?

The most effective treatments for all ischemic heart diseases are quitting smoking, and medications to control high blood pressure and high cholesterol. Some people also need medications to prevent blood clots.

The goals of treatment include:

- Relieving symptoms
- Reducing risk factors in an effort to slow or stop the buildup of plaque
- Lowering the risk of blood clots forming
- Widening or bypassing plaque-clogged arteries
- Preventing atherosclerosis-related diseases

Following a healthy diet, being physically active, and maintaining a healthy weight and managing stress are also important to stopping or reducing the build-up of plaque and avoiding medical emergencies, complication and disability. If you have severe atherosclerosis, your doctor may also recommend a medical procedure or surgery.[6]

1.6.1 Diastolic Murmur

Diastole is the part of the cardiac cycle when the heart refills with blood following systole (contraction). Ventricular diastole is the period during which the ventricles are filling and relaxing, while atrial diastole is the period during which the atria are relaxing. The term diastole originates from the Greek word meaning dilation. Diastole is closely related to the phenomenon of recoil within ballistics.

Diastolic heart murmurs are heart murmurs heard during diastole. Diastolic murmurs start at or after S2 and end before or at S1.

Many involve stenosis of the atrioventricular valves or regurgitation of the semilunar valves.

Heart sounds are the noises generated by the beating heart and the resultant flow of blood through it. Specifically, the sounds reflect the turbulence created when the heart valves snap shut. In cardiac auscultation, an examiner may use a stethoscope to listen for these unique and distinct sounds that provide important auditory data regarding the condition of the heart.

In healthy adults, there are two normal heart sounds often described as a lub and a dub (or dup), that occur in sequence with each heartbeat. These are the first heart sound (S_1) and second heart

sound (S_2), produced by the closing of the AV valves and semilunar valves, respectively. In addition to these normal sounds, a variety of other sounds may be present including heart murmurs, adventitious sounds, and gallop rhythms S_3 and S_4.

Heart murmurs are generated by turbulent flow of blood, which may occur inside or outside the heart. Murmurs may be physiological (benign) or pathological (abnormal). Abnormal murmurs can be caused by stenosis restricting the opening of a heart valve, resulting in turbulence as blood flows through it. Abnormal murmurs may also occur with valvular insufficiency (regurgitation), which allows backflow of blood when the incompetent valve closes with only partial effectiveness. Different murmurs are audible in different parts of the cardiac cycle, depending on the cause of the murmur.[7]

1.7.1 Hypertension

Most instances of hypertension (=chronically elevated blood pressure) result from increased arterial resistance, while cardiac output is in the normal range. Hence, the some of the most effective antihypertensive drugs act by decreasing this resistance, but not necessarily via direct vasodilatation, though the end result is just that. One basic problem in treating hypertension is that in the vast majority of cases (90%) the basis for the chronically elevated blood pressure is unidentified, termed primary, or essential, hypertension. Many of the interacting control mechanisms mentioned above are altered and often adjusting two or more of these processes is required to lower the BP. In a minority of cases, the hypertension is a consequence of a disease state; hence it is termed secondary hypertension. Obviously, the disease is addressed first, and its resolution will usually alleviate the secondary hypertension. If not, additional, direct intervention would then be considered.[8]

1.7.2 Classification of hypertension based on blood pressure:

Status	Systolic pressure mm Hg	Diastolic pressure mm Hg	Risk
Normal	< 130	< 85	None
Prehypertension	120 to 139	80 to 90	Slight
Hypertension:			
Stage 1 (Mild)	140 to 159	90 to 99	Long-term
Stage 2 (Moderate)	160 to 179	100 to 109	50% in 5 years
Stage 3 (Severe)	180 to 209	110 to 119	40% in 2 years
Stage 4 (Very severe)	> 210	> 120	Emergency

Risk = stroke, cardiac failure, renal insufficiency or failure

1.7.3 Classes of Antihypertensive Drugs

- Diuretics
- Centrally-acting Sympathetic Inhibitors
- Peripherally-acting Sympathetic Inhibitors
- Vasodilators
- Calcium channel Inhibitors
- ACE Inhibitors

1.7.4 Basic Physiology of Blood Pressure Control:

BP =Heart Output X Peripheral Vascular Resistance

The control of blood pressure requires constant adjustment of cardiac output and peripheral resistance. Output of blood from the heart via the aorta is dependent upon ventricular filling pressure, which is a direct function of atrial pressure (=preload), ventricle contractility (=force of contraction), heart rate, and vascular resistance (=after load). Though it may be confusing that output is dependent upon vascular resistance while the product of output and resistance determines blood pressure, simply think of the output of blood from the heart as a force and the blood-filled narrow vessels into which the newly oxygenated blood is pumped as a variably opposing force (or a variable volume of liquid <=different mass> having momentary inertia, for those of you more inclined toward physics).More important to the present discussion, there are two structural components to the vascular system essential for determining overall resistance: narrow arterioles, which exert the major force of resistance to cardiac output, and the large capacitance venules, which determine the volume and pressure of the blood returning to the heart. Overall blood volume, the third essential component in determining blood pressure, is regulated by electrolyte balance by the kidney via aldosterone whose level is controlled by angiotensin II, generated by the renin-angiotensinogen system, and K+.[8]

Objectives:

1. To understand the basic controls in regulating blood pressure
2. To know the categories of various antihypertensive drugs
3. To know the actions and uses of antihypertensive drugs

1.8.1. Acute Left Ventricular Failure

Acute left ventricular failure presents as pulmonary edema due to increased pressure in the pulmonary capillaries. It is important to realize though that left ventricular failure and pulmonary edema are not always synonymous as there are other causes for pulmonary edema e.g. acute renal failure, acute respiratory distress syndrome (ARDS).

Acute heart failure may be de novo or it may be a decomposition of chronic heart failure.

1.8.2 Causes of Acute Heart Failure

- Acute myocardial infarction / ischemia
- Acute mitral regurgitation papillary muscle rupture
- Chordae rupture
- Arrhythmias
- Aortic dissection
- Cardiac tamponade
- Valve destruction (e.g. endocarditis)
- Myocarditis
- "Flash" pulmonary edema e.g. renal artery stenos is, phaeochromocytoma

Initial Management

The patient should be sitting upright and assessed by the ABC approach.

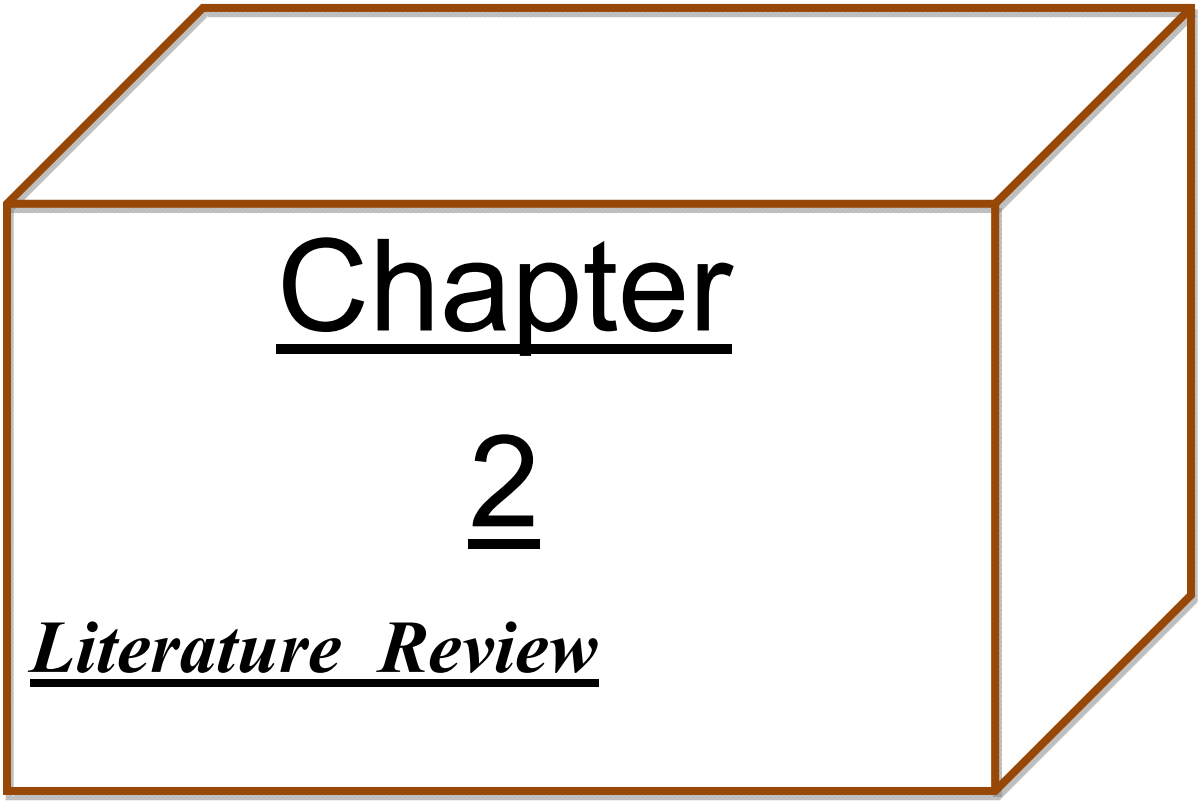
A. Check the patient's airway and administer high flow oxygen through a reservoir bag (also known as trauma mask).
B. Monitor the patient's breathing and look for evidence of fatigue (if concerns then an urgent anaesthetic/ICU opinion should be sought). Pulse oximetry should be used.
C. Assess the patient's circulation by measuring pulse and blood pressure and feeling their peripheries to check perfusion. The patient should be on a cardiac monitor to identify any arrhythmias. Insert an intravenous cannula.

Carcinogenic shock has a very high mortality (up to 90%) and the patient should be reassessed at regular intervals to identify evidence of developing shock so that appropriate management can be initiated quickly.[9]

1.9.1 Tobacco use risk factor of cardiovascular diseases:[12]

All over the prescription survey discuss the doctor maximum patient are addicted Tobacco smoke. The epidemiology of smoking-induced cardiovascular disease and the mechanisms by which tobacco smoke is thought to cause CVD. The discussion includes use of biomarkers to diagnose smoking-induced CVD and treatment implications of the pathophysiology of the disease.

1. There is a nonlinear dose response between exposure to tobacco smoke and cardiovascular risk, with a sharp increase at low levels of exposure (including exposures from secondhand smoke or infrequent cigarette smoking) and a shallower dose-response relationship as the number of cigarettes smoked per day increases.
2. Cigarette smoking leads to endothelial injury and dysfunction in both coronary and peripheral arteries. There is consistent evidence that oxidizing chemicals and nicotine are responsible for endothelial dysfunction.
3. Tobacco smoke exposure leads to an increased risk of thrombosis, a major factor in the pathogenesis of smoking-induced cardiovascular events.
4. Cigarette smoking produces a chronic inflammatory state that contributes to the atherogenic disease processes and elevates levels of biomarkers of inflammation, known powerful predictors of cardiovascular events.
5. Cigarette smoking produces an atherogenic lipid profile, primarily due to an increase in triglycerides and a decrease in high-density lipoprotein cholesterol.
6. Smoking cessation reduces the risk of cardiovascular morbidity and mortality for smokers with or without coronary heart disease.
7. The use of nicotine or other medications to facilitate smoking cessation in people with known cardiovascular disease produces far less risk than the risk of continued smoking.
8. The evidence to date does not establish that a reduction of cigarette consumption (that is, smoking fewer cigarettes per day) reduces the risks of cardiovascular disease.
9. Cigarette smoking produces insulin resistance and chronic inflammation, which can accelerate macrovascular and microvascular complications, including nephropathy.



Chapter

2

Literature Review

2.1. Title: Drug Utilization Pattern in Cardiovascular Diseases: A Descriptive Study in Tertiary Care Settings in Pakistan.

Authors: Zafar F1, Ali H1, Naveed S2*, Korai OU3, Rizvi M1, Naqvi GR3 and Siddiqui S ,Faculty of Pharmacy, Ziauddin University Karachi, Pakistan ,Jinnah University for Women, Pakistan ,Federal Urdu University of Arts, Science and Technology, Pakistan. Zafar et al., J Bioequiv.

Abstract

The correct use of cardiovascular drugs in patients has been shown to decrease the risk associated with cardiovascular morbidity and mortality. The objective of this study was to determine the drug utilization pattern in cardiovascular diseases. For this purpose we conducted this study in various tertiary care setups located in various areas of Karachi, from January to March, 2014. We collected the data from 100 patients having different age groups. The collected data was assessed to determine the prescribing trends. Results indicated that hypertension and ischemic heart diseases were mostly diagnosed and mostly diseases were treated by giving the drugs in combinations. The use of Beta Blockers, Diuretics, Calcium Channel Blockers and Angiotensin Converting Enzymes (ACE) inhibitors was very common. Also prescribing errors related to dosing frequency and prescribed dose were also determined. Results were analyzed with SPSS 20 using Chi square model to calculate the study outcome. Furthermore standard error and statistical variance for the given data is also calculated by descriptive analytical procedure. Present study will help the health practitioners to optimize the appropriate use of cardiovascular drugs.

2.2. Title: Cardiovascular disease prevalence and prescription patterns at a tertiary level hospital in Bangladesh.

Authors:Md. Abdul Muhit, Md. Obaidur Rahman, Sheikh Zahir Raihan, Muhammad Asaduzzaman, Mohammad Ahasanul Akbar, Nahid Sharmin and A. B. M.Faroque. Journal of Applied Pharmaceutical Science 02 (03); 2012: 80-84.

ABSTRACT

Demographic study of cardiovascular diseases (CVDs) and drug utilization in population is the basis for assessment of cardiovascular disease management. Aim of this study was to analyze the prevalence of CVDs with drug utilization and current trends in Bangladesh.

A cross-sectional type of descriptive study was carried out at the outdoor of National Institute of Cardiovascular Diseases (NICVD), Dhaka from July'09 to August'09. A total of 780 patients, who acquiesce with the inclusion and exclusion criteria, were interviewed with structured questionnaire and followed up by prescription monitoring. Out of the total patients with a male,

female ratio of 5.3: 4.7, 45.90% patients were over 55 years and 69.62% patients had come from urban area. The patients had lipid level disorder (47.05%), hypertension (28.05%), heart failure (27.25%), ischemic heart disease (21.55%) and 40.39% were associated with diabetes. Individual patient got 6.35 ± 1.56 no. of drug of different class of which most frequently prescribed drugs were antiatherogenic (97.67%), lipid lowering agents (95.35%), antianginal (79.07%), beta-blockers (51.16%), ACE inhibitors (30.23%), diuretics (37.21%), anxiolytics (81.4%) etc. This data may be propitious for the general physicians for optimizing rational use of cardiovascular drugs and also accessible in formulating strategy for effective cardiovascular disease management.

2.3. Title: A Statistical Analysis of Hypertension as Cardiovascular Risk Factor

Authors: Razia Iqbal, Zahoor Ahmad, Faheem Malik, Saleha Mahmood, Noreen Shahzadi, Sharoona Mehwish and Anum Zahra Department of Zoology, University of Gujrat, Gujrat, Pakistan. Middle-East Journal of Scientific Research 12 (1): 19-22, 2012.

Abstract:

There was a continuous, strong and graded relation between blood pressure and cardiovascular disease but no clear threshold value separated hypertensive patients who would experience future cardiovascular events from those who will not. 600 patients were examined. T test, Chi square test and Logistic Regression were applied. According to T test 253 were hypertensive and 357 were not. The % age of hypertension was 42.2%. The frequency of cardiovascular patient was 344 out of 600. The % age of cardiovascular patients was 57.3%. The present study suggests that numerous factors definitely increase cardiovascular risk including age, sex, family history, raised cholesterol, smoking, diabetes mellitus, obesity, sedentary lifestyle and left ventricular hypertrophy.

2.4. Title: The Role of Clinical Pharmacists in Modifying Cardiovascular Disease Risk Factors

Authors:

Autumn Bagwell, PharmD.; Jessica W. Skelley, PharmD; Lana Saad, PharmD; Thomas Woolley, PhD; and Dee Ann Dugan, PharmD,

Abstract

Objective: Assess the effect of intensive clinical and educational interventions aimed at reducing risk factors for Cardiovascular Disease (CVD), implemented by clinical pharmacists, on modifying risk factors in targeted patients at high risk for CVD.

Design: Patients with at least two risk factors for CHD were identified at two clinics by conducting a pre-intervention survey and were monitored over a period of 6 months with follow up conversations conducted every 4 weeks by phone and at subsequent physician visits. A post-intervention survey was conducted at the end of the study period to detect modified risk factors.

Setting: The Jefferson County Public Health Department (JCHD)

Participants: We followed a total of 47 patients over 6 months. The average age at baseline was 51 years old and 80% of the participants were female. The baseline average number of modifiable cardiovascular disease risk factors was 3.7.

Measurements: We assessed total number of CVD risk factors, smoking behavior, blood pressure, LDL, A1C, weight, and level of physical activity (major modifiable risk factors by the American Heart Association).

Results: Over a 6 month follow-up of 47 patients, statistically significant reductions occurred in total number of CVD risk factors, systolic and diastolic blood pressures, and A1C. Reductions also occurred in LDL level, weight, and changes in smoking behavior and physical activity were identified.

Conclusions: Results showed that increased patient counseling on adherence and lifestyle changes along with increased disease state monitoring and medication adjustment led by a clinical pharmacist can decrease risk factors in patients with multiple risk factors for cardiovascular disease.

2.5. Title: Premature Coronary Heart Disease and Traditional Risk Factors-Can We Do Better.

Authors: Roxana Sadeghi Nadia Adnani, Azam Erfanifar, Latif Gachkar, Zohre Maghsoomi. Cardiovascular Research Center, Shahid Beheshti University of Medical Sciences, Tehran.

ABSTRACT:

Background:

Traditional cardiovascular risk factors are strong predictors of an increased likelihood for premature CHD. Considering the benefits of risk factors' management, it is imperative to find and treat them before looking for more unknown and weak risk factors.

Objectives:

Limited information is available about the demographic and historical characteristics of the patients with premature Coronary Heart Disease (CHD) in IR Iran. The main objective of this study was to determine the prevalence of the traditional risk factors in these patients. Also, the researchers hypothesized that there are insufficient risk assessment and preventive intervention methods for the asymptomatic adult population.

Methods:

This study was conducted on 125 patients with premature CHD (age<50 years) who were admitted in two academic hospitals with acute coronary syndromes. The patients were accepted since they had a definite CHD on the basis of acute myocardial infarction (elevated cardiac enzymes) or documented CAD in coronary angiography.

Results:

The mean age of the study population was 42.50±5.65 (26 to 49 years). Among the patients,92 (73.6%) were male, 113 (90.4%) were married, 58 (46.4%) were smokers,19 (15.2%) were opium users, 97 (77.6%) had dyslipidemia, 44 (35.2%) had hypertension, and 33 (26.4%) had diabetes mellitus. In addition, family history was presented in 54 patients (43.2%).

Among the study population, 120 patients (96%) had at least one of the traditional risk factors, including dyslipidemia, hypertension, diabetes mellitus, cigarette smoking, and family history of CHD. However, none of the dyslipidemic patients had controlled total cholesterol, LDL, HDL, and triglyceride. Also, none of the diabetic patients had hemoglobin A1C<7%. Among the 44 hypertensive patients, blood pressure of 15 ones (34%) was within the normal range. Besides, only 3 patients (2.4%) had regular physical activity (at least 30 minutes, three times a week).

Conclusions:

Premature Coronary Heart Disease is a public health problem. However, there is lack of effective and intensive treatments of well-defined traditional risk factors and prevention methods for the majority of the patients experiencing premature CHD. In sum, there is still plenty of room for improvement of risk management in IR Iran.

2.6.Title: National survey on prescription of cardiovascular drugs among outpatients with coronary artery disease in Switzerland.

Authors: Jörg Muntwyle, Giorgio Noseda, Roger Darioli, Christiane Gruner, Felix Gutzwiller, Ferenc Follath, Department of Internal Medicine, University Hospital Zurich, Switzerland.

ABSTRACT:

Background: Secondary prevention of coronary artery disease markedly reduces cardiovascular mortality and non-fatal endpoints. Outpatient care of subjects with coronary artery disease has been assessed in several European countries, but no current data is available for Switzerland.

Methods: A random sample of office-based physicians across Switzerland recorded current drug prescription of outpatients with coronary artery disease in the years 2000/2001 by means of a mail questionnaire. We assessed treatment frequencies according to different patient characteristics.

Results: 565 patients were included (mean age 68 ± 11 years, 75% male). There was no evidence for differences in drug utilization among the regions. Drug prescription rates for antithrombotic agents, beta-blockers, ACE-inhibitors/angiotensin receptor blockers and lipid lowering drugs were 91%, 58%, 50% and 63% respectively. Lower treatment rates were observed among patients >70 years and in those without a history of myocardial infarction or coronary revascularization. Forty-nine percent of the patients had a blood pressure >140/>90, and 60% had lipid readings above the intervention cut-off according to the Swiss recommendations.

Among those without a history of myocardial infarction or coronary revascularization, the respective figures were 60% and 80%.

Conclusions: Compared to former surveys evidence based drug prescription has improved in Switzerland. Despite this, therapeutic goals for cholesterol levels and blood pressure are not being reached in a large proportion of patients. A high risk group for under use of evidence based drugs are patients without a history of myocardial infarction or coronary revascularization.

Chapter 3

Significance (purpose)of the Study

3.1 Purpose of the Study:

The purpose of this study was to describe the prevalence of cardiovascular and other circulatory diseases and associated utilization of cardiovascular medicines. [10]

The study evaluated:

- The prevalence of any cardiovascular disease, circulatory disease, coronary artery disease, congestive heart failure, or cerebrovascular disease among adults in Bangladesh;
- Identification of various age group who are "at risk" for cardiovascular disease due to a diagnosis of hypertension or angina.
- Identification of various therapeutic classes of medications which are used to treat cardiac diseases.
- For assessing variable types of cardiovascular drugs and matter relating to about near future prospects of other new cardiovascular agents in Bangladesh.
- Inpatient, outpatient emergency department, and office-clinic visit utilization rates specific to cardiovascular diagnosis.
- Identify the guidelines for the prevention, detection and management of chronic heart failure
- Identify clinical guidelines for stroke management.
- Use of medications to treat cardiovascular disease.
- Age-specific and age-standardized rates;
- Prevalence, utilization and payment rates by geographical location (Health Analysis Area) of member residence; and
- Prevalence of several coexisting conditions.

Chapter 4

Methodology

4.1 Methodology

To perform this part of Project protocol, the methodology, involved for the under taking of a number of steps. A randomized representative sample was determined before the required date was collected. Over the 1.5 months collection period we selected randomly ideal 200 prescriptions from National Institute of Cardiovascular Disease (NICVD).

219 prescriptions was collected from National Institute of Cardiovascular Disease (NICVD) Hospital outdoor and finally 200 prescriptions selected that were completely cardiac disease drugs content which were prescribed by 99% specialist and 1% general physician.

This was absolutely essential for the purpose of obtaining information that actually represented the real scenario. Among the 200 prescriptions 160 were male and 40 were female, all were adults of more than thirty years of age. Some confidential information was collected orally and some was collected in written form. Besides some information was collected observation. Two sources were basically used to collect the data. Here, all data was collected from the representative drug house, hospital and direct interview of patient.

Chapter 5

Result&Discussion

5.1 Results and Discussion:

Out of 200 patients who came to visit National Institute of Cardiovascular Disease (NICVD) Hospital, patients were male 80% and female were 20%.Approximately maximum patients were urban area whereas minimum patients came from rural area and the difference was found to be statically insignificant. The patients above thirty years of age were 97%.

Table 1: Prevalence of cardiovascular disease in Patients' ≥12 years of age.

Age(Years)	Prescriptions
12-19	8
20-39	24
40-59	80
60-79	70
80+	18

Table 2: Comprehensive list of all types of prescribed cardiovascular drugs alone (n=200)

Therapeutic class	**No. of prescriptions**	**Percentage (%)**
Organic nitrates	52	26
Beta-adrenoceptor blocker	38	19
Anticoagulant, antiplatelet and thrombolytic drug	70	35
Calcium channel blocker	30	15
Diuretics	15	7.5
Renin-angiotensin system drugs	25	12.5
Lipid lowering drugs	15	7.5
Miscellaneous	2	1

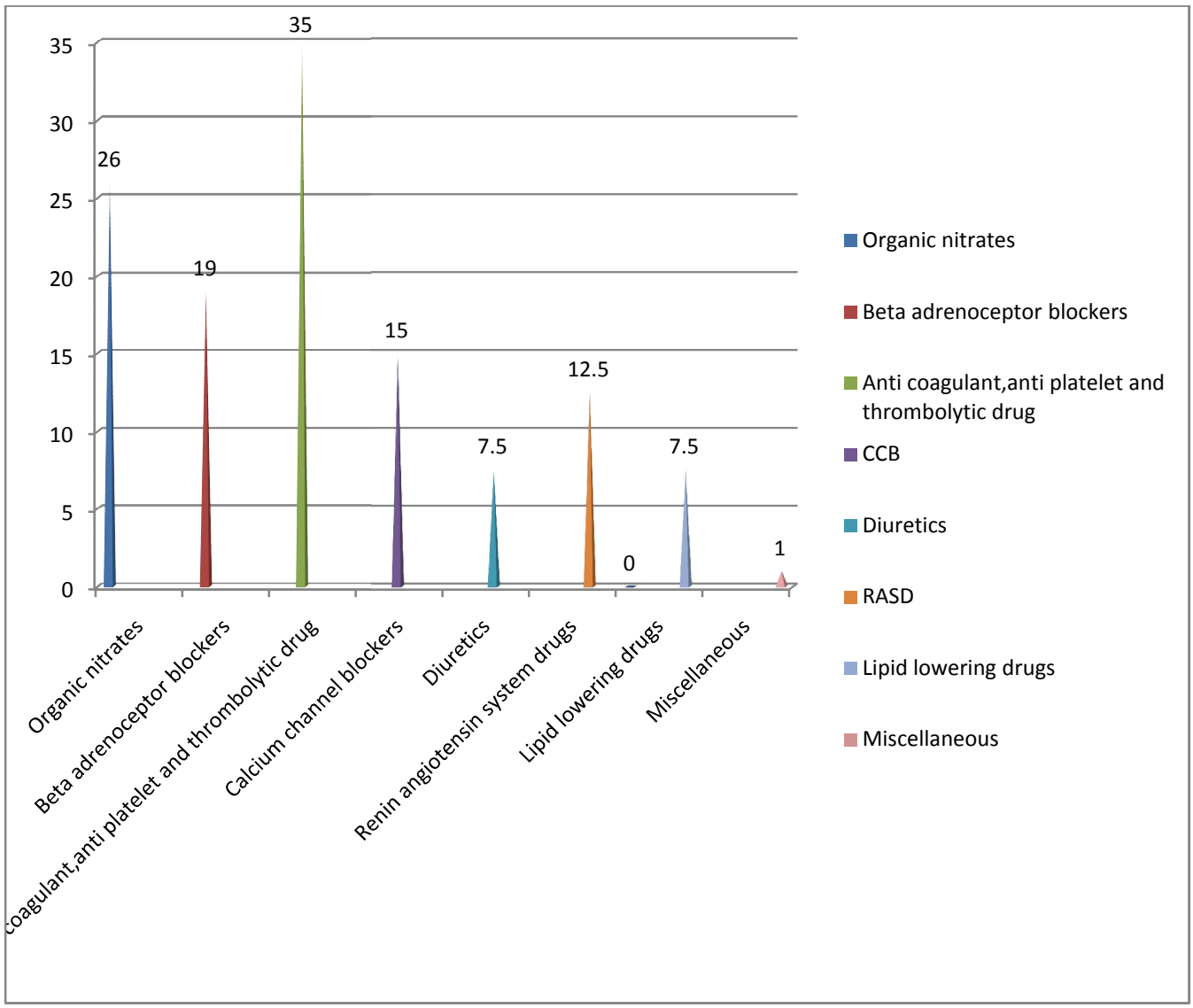


Fig-1. Prescribed different cardiac drugs.

Table 3: Different cardiac disorder (n=200)

Disease name	**No. of Patients(n=200)**	**Percentage (%)**
Angina(Stable angina, Unstable angina)	**33**	**16.5**
Non-STsegment elevation myocardial infarction	**22**	**11**

Chronic Rheumatic Heart Diseases	**15**	**7.5**
Diastolic Murmur	**16**	**8**
Hypertension	**36**	**18**
Left Ventricular Failure	**28**	**14**
Chronic Obstructive Pulmonary Diseases.	**12**	**6**
Ischaemic (or ischemic) heart disease	**8**	**4**
Acute myeloid leukemia (AML)	**30**	**15**

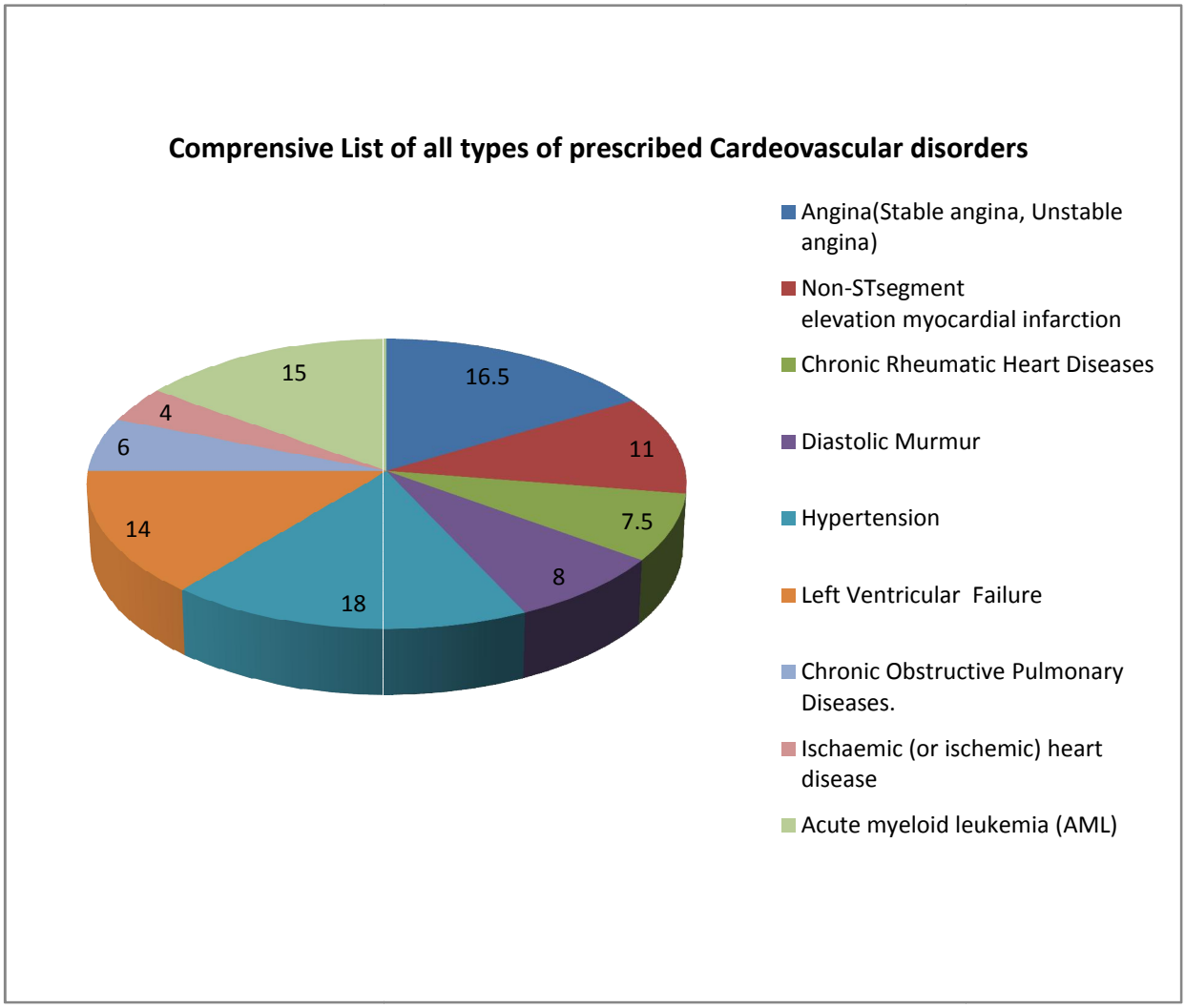


Fig-2: Comprehensive list of all types of cardiovascular disorders (n=200)

Table 4: Various generics of Anticoagulant, antiplatelet and Fibrinolytic agents

Generic name of Drugs	No. of prescription for Anticoagulant, ant platelet and thrombolytic drug alone(n=200)	Percentage (%)
Aspirin	120	60
Clopidogrel	140	70
Warfarin	1	0.5

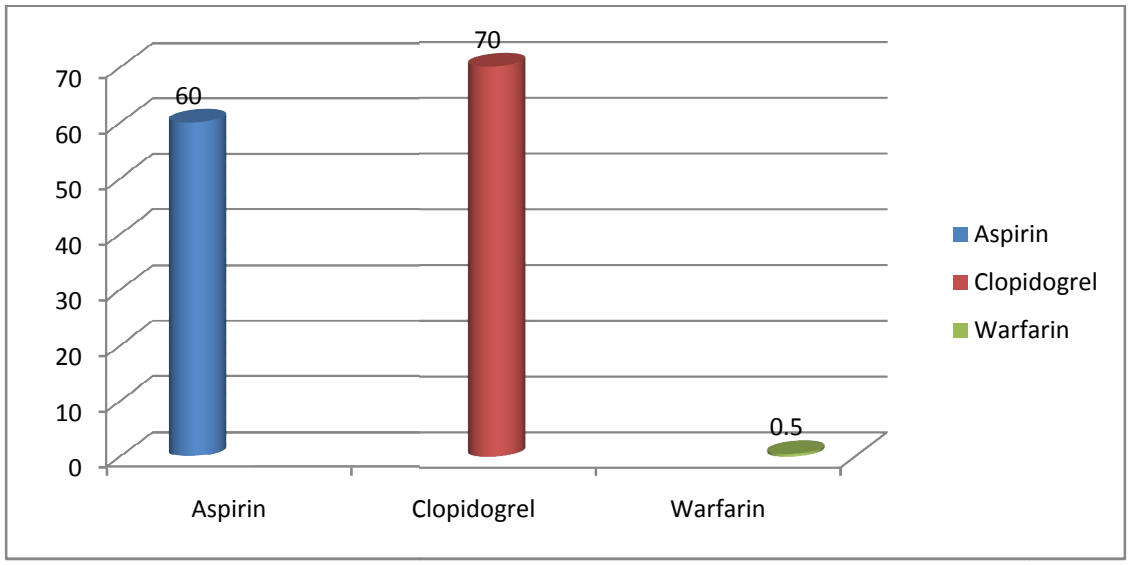


Fig-3. Widely used Anticoagulant, antiplatelet and Fibrinolytic drugs.

Table 5: Various Generics of Beta blockers

Generic name of Drugs	No. of prescription for Beta adrenoceptor blocker alone (n=160)	Percentage (%)
Atenolol	65	32.5
Metoprolol	35	17.5
Propranolol	43	21.5
Carvedilol	18	9

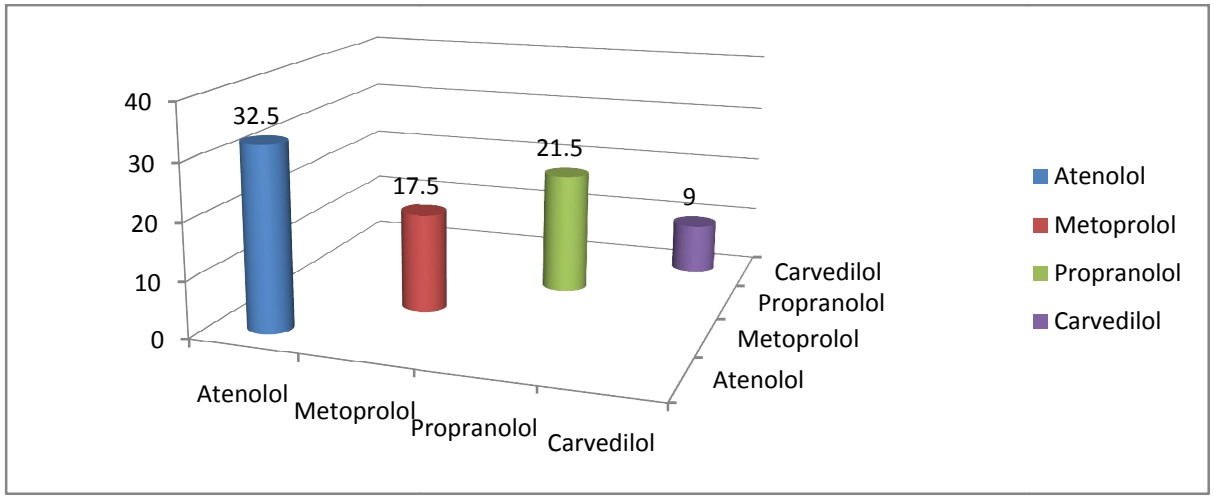


Fig-4. Comparison of various Beta blockers according their generic name.

Table 6: Various generics of Calcium channel blockers

Generic name of Drugs	**No. of prescription for Calcium channel blockers alone(n=71)**	**Percentage (%)**
Amlodipine	25	12.5
Diltiazem	23	11.5
Verapamil	18	9
Nifedipine	5	2.5

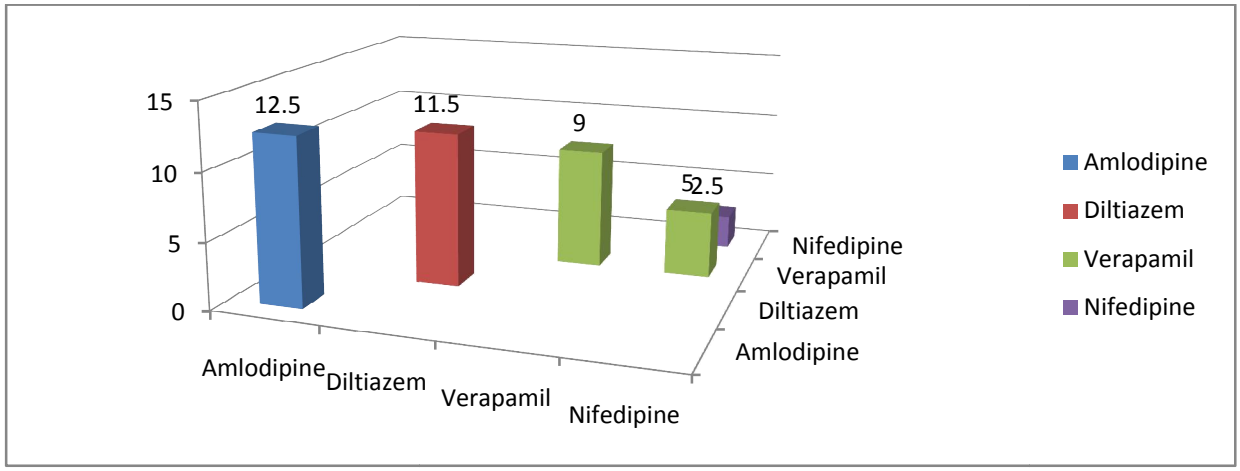


Fig. 5: Calcium channel blocker according to their generic name

Table 7: Various generics name of angiotensin converting enzyme inhibitor

Generic name of Drugs	No of prescriptions for Renin angiotensin system drugs alone (n=76)	Percentage (%)
Captopril	**10**	**5**
Lisinopril	**7**	**3.5**
Ramipril	**45**	**22.5**
Enalapril	**5**	**2.5**

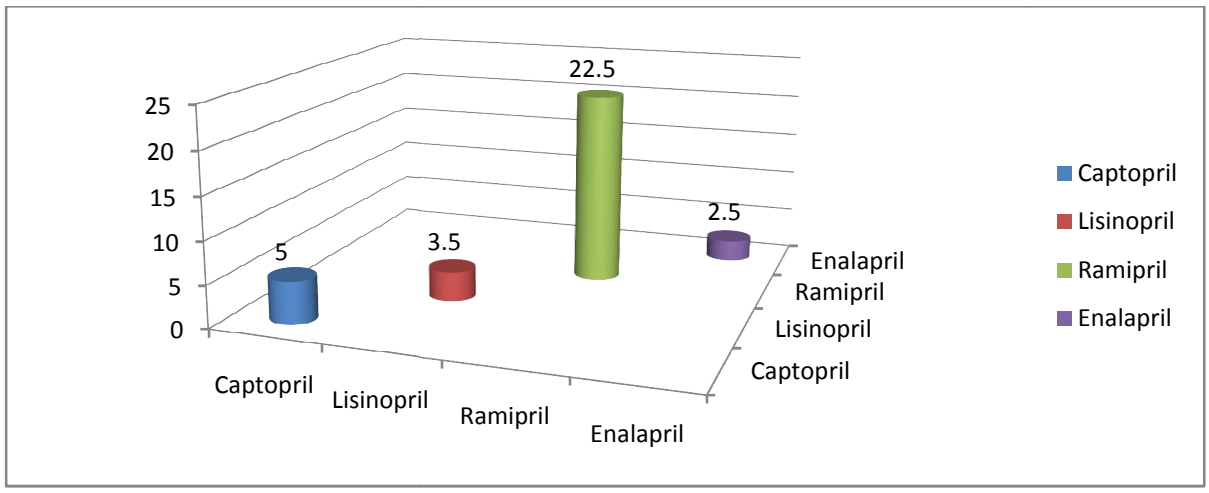


Fig-6. Comparison of available ACE-Inhibitors

Table 8: Various generics of Lipid lowering drugs

Generic name of Drugs	No. of prescription for Lipid lowering drugs alone(n=82)	Percentage (%)
Fluvastatin	14	7
Atorvastatin	30	15
Simvastatin	20	10
Fenofibrate	12	6
Gemfibrozil	6	3

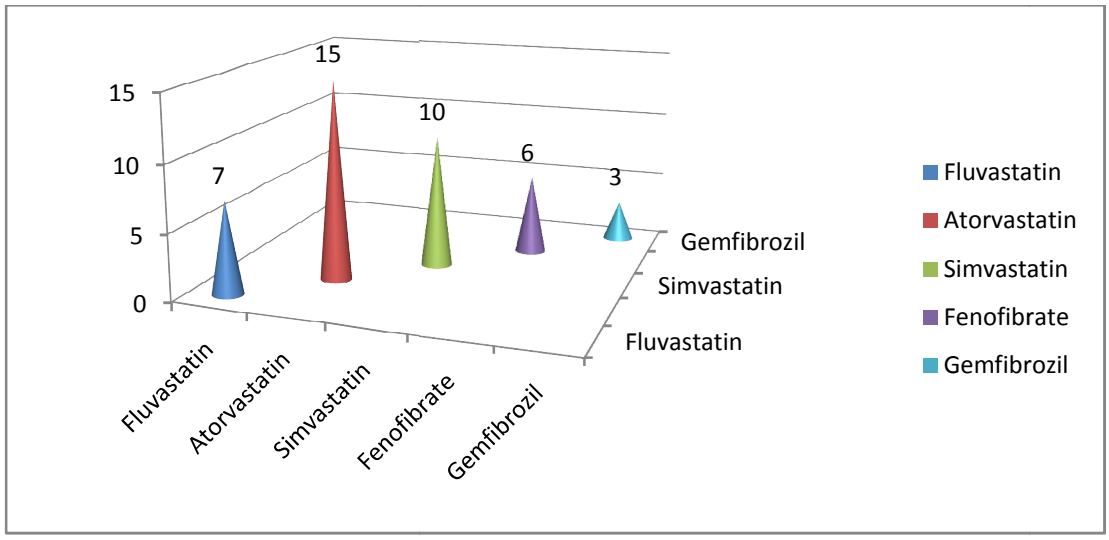


Fig-7. Comparison between various generic classes of lipid lowering drugs

Table 9: comparison of available K+- sparing diuretics

Generic name of Drugs	No. of prescription for K+-sparing diuretics.(N=61)	Percentage (%)
Spironolactone	35	17.5
Triamterene	15	7.5
Amiloride	11	5.5

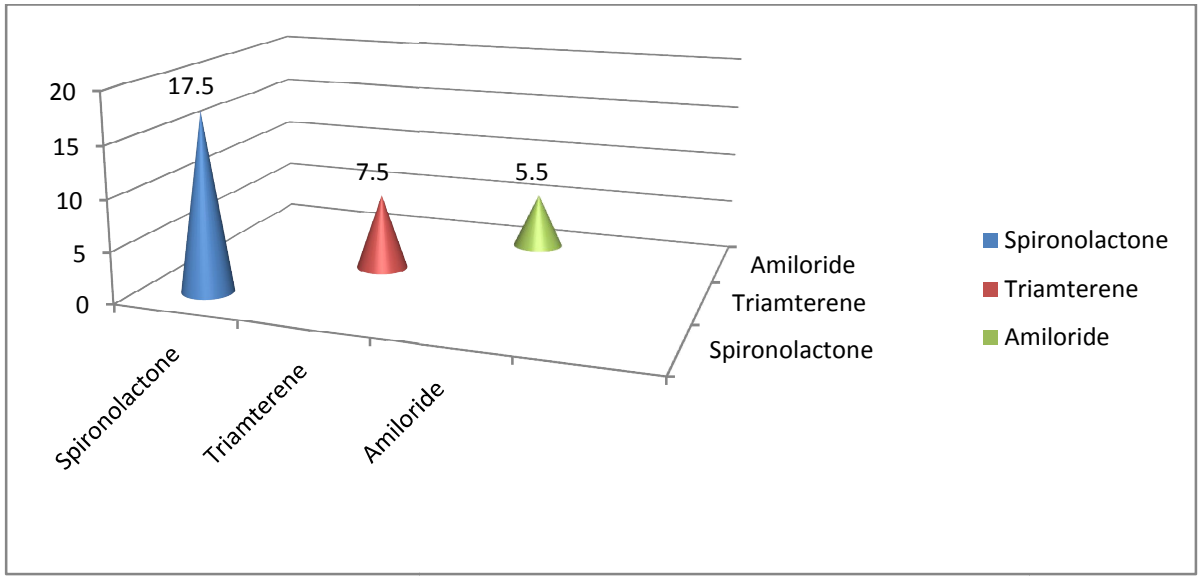


Fig-8. Presentation of available K+ sparing diuretics

Table-10: Comparison of available loop diuretics

Generic name of Drugs	No. of prescription for loop diuretics.(N=60)	Percentage (%)
Frusemide	35	17.5
Torsemide	25	12.5

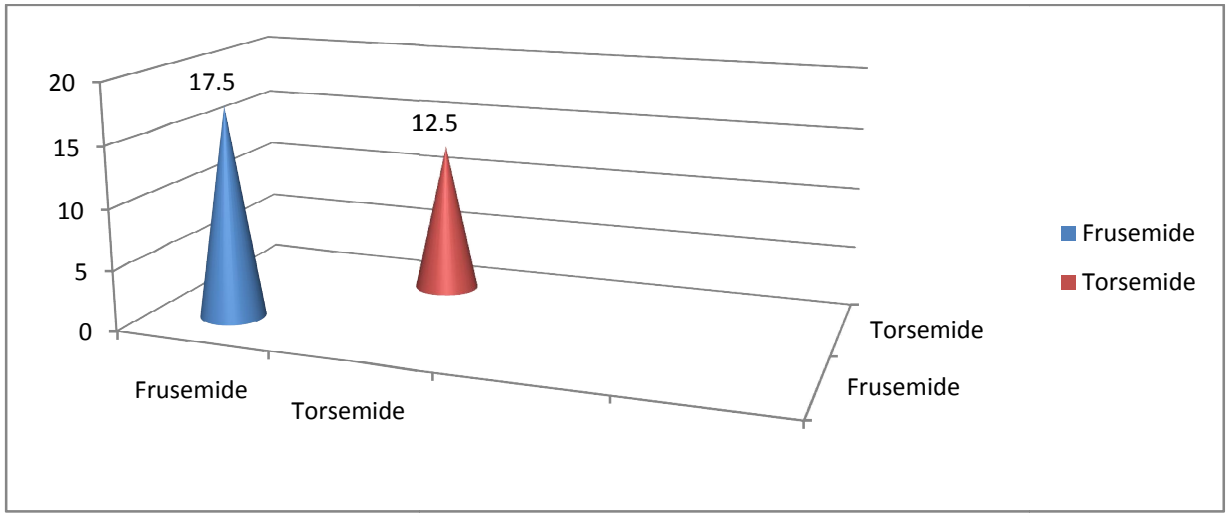


Fig-9. Presentation of available loop diuretics.

Chapter 6

Conclusion

Conclusion

Statistical analysis reveals that Hypertension & Angina occurs most prominently which is found in 36% and 33% patients respectively. Among all types of cardiovascular drugs Organic nitrates, Beta-adrenorecpor blockers and Anticoagulants are prescribed mostly than other class of drugs, which are used 26%, 19% and 35% respectively. Changes in patterns of cardiovascular disease management and drug use are changing day by day. Anti-platelet and thrombolytic drug, Calcium channel blocker, Diuretics, Renin-angiotensin system drugs, Lipid lowering drugs are also used prominently. This Survey has many draw backs such as many times it was not possible to collect latest information about the drugs due to demand a charge for the new journals, medical representatives of different companies do such type presentation survey almost every time so drug housekeeper feels disturb in this events. Hence sometimes prescription goes in irregular fashion, Patient feels disturb to collect prescription; Professor level doctor's interview was very difficult. [13]

Now a day in cardiovascular disease the approach is much more preventive than cure. For example antioxidant, antilipidemic agents are used to reduce the incidence of diseases. Cardiovascular disease is not totally curable. So patients should be conscious about using the drug. There is a trend when it feels good patient stop taking medication. This approach hampers the therapy. During this survey it was observed that though the cardiovascular drugs are so potentially lifesaving one. Moreover, this survey based on demographic data and statistical approach collected from National Institute of Cardiovascular Disease (NICVD) (Shera-Bangla Nagar Dhaka1207, Bangladesh)

Bangladesh. Moreover, the study based on a tertiary level hospital, may not accord with the data to other generalized hospitals. Furthermore, this study protocol will also ascertain the further evaluation and practice of cardiovascular drugs practice in cardiac disease management in Bangladesh.[13]

Statistically significant results showed that increased patient counseling on adherence and lifestyle changes along with increased disease state monitoring and medication adjustment led by a clinical pharmacist can decrease risk factors in patients with multiple risk factors for cardiovascular disease. Clinical pharmacists are in an optimal position to provide effective interventions aimed at decreasing risk factors for cardiovascular disease. Increased monitoring for cardiovascular disease presence and progression is recommended in both symptomatic and asymptomatic adults. Further research and consideration into the role of the clinical pharmacists in implementing interventions targeted at decreasing risk factors for cardiovascular disease is warranted.[14]

Cardiovascular disease is the leading cause of death. Importantly, it remains the foremost cause of preventable death globally. Public health efforts to improve lifestyles, controlling lifestyle-related major cardiovascular risk factors, will certainly contribute to cardiovascular disease prevention.[16]

Chapter 7

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