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A Study on Prescribing Drugs of Heart Failure and Patient Compliance

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I. INTRODUCTION

Heart Failure

Heart failure is a clinical syndrome with signs or symptoms as your heart gets weakness unable to pump enough blood to body; caused by structural or functional abnormality resulting in elevated natri-uretic peptide levels and objective evidence of pulmonary or systemic congestion.

This condition includes hypertension, coronary heart disease, heart inflammation, cardiomyopathy. The signs and symptoms of heart failure may not show up right immediately. However, over time, you can have fatigue, shortness of breath, and a buildup of fluid in your lower body, stomach, or neck.[1]

STAGES

1) Stage A

There are no symptoms of known in stage A heart failure. Certain risk factors, such as genetics or a family history are uncontrollable. Among the risk factors modifiable are:[2]

- Hypertension;
- Diabetes
- Obstructive heart disease
- Being obese

In addition, to the above there is an increased risk of heart failure if you are exposed to certain medications or toxic substances like alcohol cocaine or methamphetamines, as well as treatments for cancer such as radiation and specific chemotherapeutic agents.[3]

Treatment of stage A

- Control of the stage A progressive lifestyle changes are necessary to treat heart failure. These include controlling blood pressure, blood sugar, and cholesterol as well as following healthy diet, exercise and quiting from excessive alcohol and smoking.
- Excessive filling pressures can be demonstrated by non-invasive imaging techniques like Doppler echocardiography and invasive hemodynamic testing.
- Individuals with risk factors, elevated BNP levels or persistently elevated cardiac troponin in the absence of cofounding conditions like myopericarditis, CKD, acute coronary syndrome, or pulmonary embolism.

2) STAGE-B

It also referred as pre-heart failure, but they are not yet severe enough to produce symptoms.

Evidence of structural or functional irregularities in the heart can be found in the following ways:

- Issues with valves
- Decreasing heart wall thickness
- Heart filling issue (diastolic dysfunction)

- A lower pumping function as determined by the ejection fraction (a condition known as systolic heart failure, which occurs when the heart muscle is unable to contract properly and unable to pump enough blood that is rich in oxygen throughout the body).
- Increased markers of the heart, like B-type natriuretic peptide (BNP),Creatinine kinase enzyme,Troponin levels.

Treatment of Stage B Heart Failure

Similar to stage A heart failure, progressive life style modifications are part of the treatment for stage B heart failure. Furthermore, patients with stage B heart failure who have a history of heart attacks or a reduced ejection fraction may benefit from specific medications.

- Angiotensin receptor blockers (ARBs) or angiotensin converting enzyme (ACE) inhibitors • Beta blockers.[4]

3) STAGE C

Most people consider Congestive Heart Failure to be in stage C, also known as symptomatic heart failure,which is characterized by cardiac impairment leading to the following symptoms derived by Heart Association of America as follows :

- Dyspnea
- Dyspnea upon awakening from sleep
- Coughing
- Lower extremity or abdominal swelling
- Weight gain
- Palpitations
- Dizziness
- Decreased appetite or feeling full sooner after eating.[5]

Treatment of stage C heart failure:

- ACE inhibitors, ARBs, or ARB + neprilysin inhibitor Diuretics.
- SGLT-2 inhibitor, aldosterone antagonist, and beta-blockers.

4) STAGE D

Stage D is also reffered as advanced heart failure or end stage heart failure which is the most severe type of heart failure and frequently requires hospitalization with medical treatment.

People with stage D heart failure have a poor prognosis, with survival rate of only 20% in the patients.

Treatment of Stage D Heart Failure

The same drugs used to treat stage C heart failure are also used to treat stage D heart failure, though their tolerance may be compromised by low blood pressure or adverse effects. The following extra therapies may be taken into consideration for heart failure in stage D:

Mechanical support devices, like the left ventricular assist device (LVAD)

- Inotropes, which are drugs administered through a continuous intravenous infusion that help the heart muscle contract. These medications may improve quality of life but can also cause arrhythmias and lower overall survival.
- Heart replacement therapy
- Palliative care.[6]

How to stop the Progression of Heart Failure

Although we can't stop heart failure from progressing, there are few steps are considered to maintain the best possible health for our hearts.

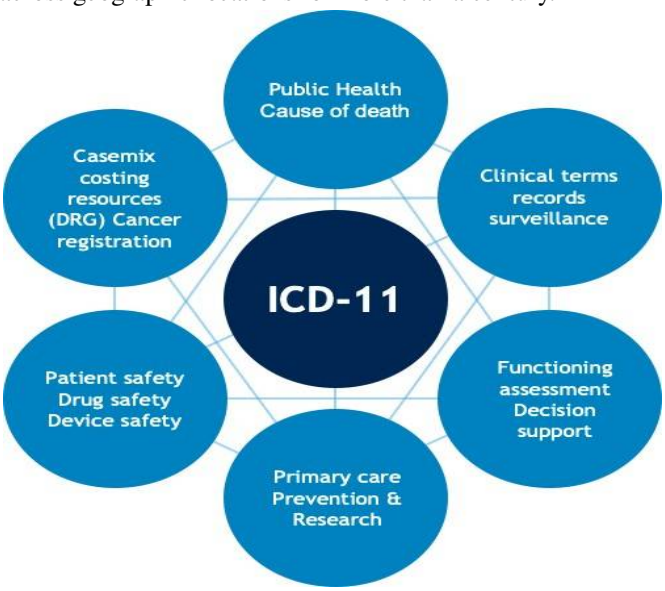
Among them are:

- Exercise on a regular basis (approximately 30 min/day).

- Consuming a diet low in sodium, processed foods, trans fats,sugars, fruits, vegetables, legumes, beans, and whole grains to promote heart health.
- Cessation of smoking.
- Regulating blood sugar, cholesterol, and blood pressure.
- Taking prescription drugs as prescribed by physician.[7]

a) INTERNATIONAL CLASSIFICATION OF DISEASES (ICD)

ICD is used for a wide range of purposes around the world and, through data that is reported and coded with the ICD, offers vital information about the scope, causes, and effects of human disease and death globally. ICD-coded clinical terms serve as the primary foundation for health records, disease statistics, and cause-of-death certificates in primary, secondary, and tertiary care settings. Payment systems, service planning, the management of quality and safety, and health services research are all supported by these data and statistics. Large-scale research is made possible and data collection is standardized through the use of diagnostic guidelines connected to ICD categories. The International Classification of Diseases (ICD) has provided consistent statistics on the causes of death and morbidity over time and across geographic locations for more than a century.



- *ICD-11 USES*

For more than a century, the International Classification of Diseases (ICD) has consistently provided statistics on the causes of death and morbidity over time and across geographic locations.

- *Certification and reporting of Causes of Death*

ICD standards are followed in the recording, analysis, and reporting of death cause information on standard forms. A population's long-term health trend is reliably provided by the data.

- *Epidemiological data during a pandemic or epidemic*

Reporting and coding morbidity, such as first aid ICD records and reports precise and accurate information about the illnesses that people suffer from and the treatments that they receive. This covers all health care levels, ranging from primary to secondary and tertiary.

This data is also useful for planning, designing policies, and keeping track of every facet of a population's health.

- *Casemix and Diagnosis-Related Grouping (DRG)*

Information that has been ICD-coded is used to allocate resources or make lump sum payments to statistically equal groups.

- *Assessing and monitoring the safety, efficacy, and quality of care:*

According to WHO recommendations for patient safety incident reporting and learning systems, quality of care uses ICD-coded information to describe the patient's condition, the treatment's outcome, and incidents or near-incidents, including mechanisms and involved objects like an infusion pump failing or a patient accidentally giving themselves the wrong dosage of a medication.

- *Cancer registries*

Over time, cancer registration offers comprehensive data on cancer patients. ICD codes information about the kind of cancer, where it is, how it spreads, and how it behaves.

- *Anti-Microbial Resistance*

Antimicrobial resistance (AMR) affects international trade and security while posing a major risk to public health. ICD provides the data recommended by the Global Antimicrobial Resistance and Use Surveillance System (GLASS). This makes it easier to record and share information about the infection, the specific agent, and the type and extent of drug resistance.

- *Reasearching and performing clinical trials and epidemiological studies*

For research and trials, information from various sites can be pooled both locally and globally when coded data in multiple languages and with common diagnostic approaches are comparable. Across languages, borders, and contexts, ICD offers all the levels of detail needed for thorough clinical and research documentation.

- *Assessing functioning*

The ability to complete tasks on one's own and engage in daily activities can be used to summarise how well a person functions overall. Additionally, it can be used to monitor the state of affairs generally or both before and after therapy. Users can compute a functioning score using the functioning categories included in ICD, which are based on the WHO disability assessment scheme (WHODAS2).[8] Some of the ICD codes which explains the severity of the heart failure as follows :

b) ICD CODE CLASSIFICATION OF HEART FAILURE

- ICD CODE OF HEART FAILURE-I50.9
- ICD CODE OF EJECTION FRACTION-I50
- ICD CODE OF REDUCED EJECTION FRACTION-I50.2
- ICD CODE OF PRESERVED EJECTION FRACTION-I50.3

➢ PATIENT COMPLIANCE:

The patient compliance and willingness of a person to adhere to medical advice,following the prescription medication as directed, show up for planned clinic appointments, and carry out advised investigations is referred to as patient compliance. There are serious health consequences, including levels of morbidity, death, and cost usage. The most frequent reason for drug non-response has been cited as poor compliance. Evidence suggests that patients who follow treatment recommendations have better health outcomes than those who do not, even when taking a placebo. Evidence-based practice guidelines, which are built on clinical, behavioral, and educational principles, offer a way to measure outcomes related to health status, patient satisfaction, and cost-benefit considerations. They may also help to guarantee that compliance is shared by the healthcare professional and the subject.[9]

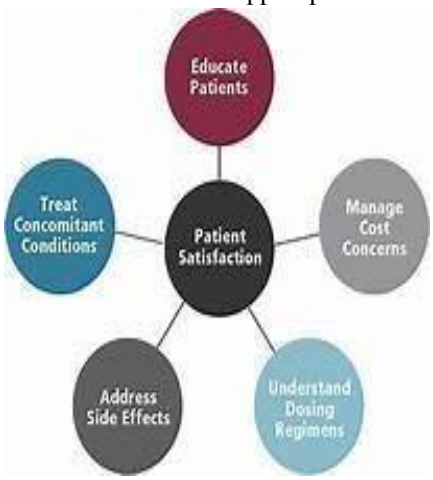
Eg : For chronic disease management to be successful, patient compliance is essential. Better clinical outcomes in chronic illnesses and disease control are facilitated by patient compliance with prescriptions and health guidelines. Regretfully, research has revealed low patient compliance rates . Complications and a decline in health are caused by low patient compliance. Readmissions of patients with chronic illnesses who have previously been hospitalized are common due to factors such therapy non-compliance and lifestyle modifications.

Non- compliance can result in inefficient treatment outcomes, greater hospitalization rates, lower discharge rates, complications, higher healthcare expenses, and even mortality.

But the term "patient compliance" has been used in multiple disciplines over the years, it is still unclear in the healthcare industry. The specific distinguishing traits might facilitate clear communication within the healthcare industry. Therefore, it is imperative to thoroughly investigate the concept of patient compliance . Improved understanding and clarification of the concept of patient compliance will benefit understanding of patient compliance as a complex idea encompassing a range of situations, behaviors, and

environments. The development of reliable and accurate scales and measuring techniques for evaluating different aspects of patient compliance is aided by concept analysis. It helps identify the crucial indicators and factors that need to be taken into account when gauging compliance.

Particular obstacles to adherence by comprehending the fundamental elements . In addition, a concept analysis of patient compliance can help researchers, medical professionals, and policymakers comprehend the idea better and develop interventions that will improve patient outcomes and healthcare interventions to support patient compliance.[10]



- ETIOLOGY

Heart failure is commonly caused by coronary artery disease, which includes an earlier MI (myocardial infarction), hypertension, atrial fibrillation, valvular heart disease, excessive alcohol consumption, infection, and cardiomyopathy of unknown cause. Furthermore, viral infections of the heart can cause inflammation in the heart's muscle layer, which in turn can aid in the onset of heart failure. The influence of genetic predisposition is significant.

There are numerous causes of heart damage, such as drug induced disorders, systemic viral infections (like HIV), chemotherapeutic agents (like daunorubicin, cyclophosphamide, and trastuzumab), and alcoholism methamphetamine, cocaine. Exposure to certain toxins, like lead and cobalt, is an uncommon cause.

Other reasons like connective tissue diseases like systemic lupus erythematosus and
infiltrative disorders like amyloidosis. Heart failure is thought to have an independent cause, namely obstructive sleep apnea, a condition of sleep in which abnormal breathing overlaps with obesity, hypertension, and/or diabetes. Variations in blood pressure have been connected to heart failure in recent reports from clinical trials.[11]

- EPIDEMIOLOGY

Over 64 million people worldwide affected from heart failure in 2022. About 2% of adults worldwide suffered from heart failure. From all over of the population people over age 75 rates greater than 10%. An increase in rates is anticipated. Growing life span is the primary cause of these rates, but other contributing factors include elevated risk factors (obesity, diabetes, dyslipidemia, and hypertension) and better survival rates from other forms of cardiovascular disease (arrhythmias, valvular disease, and myocardial infarction). For those over 65, heart failure is the most common reason for hospitalization.

- SEX

Men have a higher incidence of heart failure, but the overall prevalence rate is similar in both sexes since women survive longer after the onset of heart failure. Women tend to be older when diagnosed with heart failure (after meopause), they are more likely to have diastolic dysfunction, and seem to experience a lower overall quality of life than men after diagnosis.[12]

- PATHOPHYSIOLOGY

The pathophysiology of heart failure is due to reduced heart muscle efficiency due to injury or overloading is the primary pathophysiology of heart failure. Because of this, a variety of conditions can leading to, such as cardiac amyloidosis, hypertension,

and myocardial infarction, which can cause the heart muscle to stiffen due to misfolded proteins depositing in it and the muscle being starved of oxygen resulting in death. These increases in workload will eventually cause modifications to the heart itself.

Due to ventricle overload, a person with heart failure may have less force when their heart contracts. The Frank-Starling law of the heart states that in a healthy heart, increased ventricle filling leads to increased contraction force and, consequently, an increase in cardiac output. This mechanism breaks down in heart failure because the ventricle is so full of blood that the heart's muscles can no longer contract as effectively. This results from the overstretched heart muscle's decreased capacity to cross-link actin and myosin filaments.[13][

Failure of the diastole, systole, or both can result in a decreased stroke volume. Reduced contractility is typically the cause of increased end systolic volume. Impaired ventricular filling causes a decrease in end diastolic volume; this happens when the ventricle's compliance decreases (i.e. when the walls stiffen). In periods of increased oxygen demand (such as exercise), the amount of cardiac output can increase as the heart works harder to meet normal metabolic demands. This adds to the exercise intolerance that heart failure patients frequently experience. This corresponds to a reduction in cardiac reserve, or the heart's capacity to pump blood during demanding physical activity. The heart cannot keep up with normal metabolic demands because it must pump more blood.

An elevated heart rate, induced by elevated sympathetic activity, is a common observation in heart failure patients in order to maintain a sufficient cardiac output. At first, this keeps blood pressure and perfusion levels stable, which helps offset the effects of heart failure. However, it also puts additional strain on the myocardium and raises the need for coronary perfusion, which can exacerbate ischemic heart disease. Abnormal heart rhythms that have the potential to be fatal can also be caused by sympathetic activity. There could be a physical enlargement of the cardiac muscle layer. The cause of this is the heart's terminally differentiated muscle fibers getting bigger in an effort to become more contractile. This could be a factor in the increased stiffness and consequent reduction in diastolic relaxation capacity.

Reduced cardiac output and increased heart strain are the overall effects. This decreases blood flow to the rest of the body and raises the risk of cardiac arrest, particularly from abnormal ventricular heart rhythms. The decreased cardiac output in chronic illness results in various alterations in the body, some of which are physiological responses etc.....

Vasopressin, also referred to as antidiuretic hormone, or ADH, is secreted by the posterior pituitary in response to elevated sympathetic stimulation, which results in renal fluid retention. As a result, the blood pressure and volume rise.

- ✓ Heart failure also impairs the kidneys' capacity to excrete water and salt, which exacerbates edema. Renin is released when the blood supply to the kidneys is reduced, and this enzyme is responsible for producing the powerful vasopressor angiotensin. Angiotensin and its metabolites increase the vasoconstriction and stimulate the adrenal glands to secrete more aldosterone, a steroid. This encourages the kidneys to retain fluid and salt.
- ✓ Atrophy of the muscle fibers is brought on by decreased skeletal muscle perfusion. Exercise intolerance can be exacerbated by this, leading to weakness, increased fatiguability, and decreased peak strength.

Cardiogenic pulmonary edema that can develop in the lungs as a result of left-sided heart failure. This narrows the air-blood gap, stiffens the lungs, and decreases spare capacity for ventilation. It also lowers gas exchange efficiency. This leads to orthopnea, paroxysmal nocturnal dyspnea, and dyspnea (shortness of breath) which side of the heart fails largely dictates the symptoms of heart failure. Blood is pumped by the left side into the systemic circulation and by the right side into the pulmonary circulation.

Although the pulmonary circulation is impacted by left-sided heart failure, this does not always translate into a reduction in cardiac output to the systemic circulation at the beginning of symptoms. The ejection fraction is lowered in systolic dysfunction, which results in an abnormally high volume of blood in the left ventricle. The end-diastolic ventricular pressure will be elevated in diastolic dysfunction. The left atrium and pulmonary veins receive this surge in volume or pressure first. Pulmonary edema results from an increase in volume or pressure in the pulmonary veins, which hinders the alveoli's natural drainage and promotes the fluid's movement from the capillaries to the lung parenchyma. Thus, gas exchange is hampered. Thus, orthopnea, paroxysmal nocturnal dyspnea, and dyspnea are common respiratory symptoms associated with left-sided heart failure.

Patients with severe cardiomyopathy may experience symptoms such as cyanosis, claudication, cold and clammy extremities, dizziness, and fainting. These symptoms are more evident due to reduced cardiac output and poor perfusion.[14]

- ➢ SYSTOLIC DYSFUNCTION OR HFrEF:

Systolic dysfunction-related heart failure is more easily identified. Simplistically put, it can be explained as the heart's pump function failing. It is distinguished by an ejection fraction that is lower than 45%. Insufficient ventricular contraction strength leads to insufficient stroke volume, which in turn causes insufficient cardiac output. Myocytes that are congenital diseases, like Duchenne muscular dystrophy, have altered molecular structures.

Both inflammation (myocarditis) and infiltration (amyloidosis) can harm myocytes and the parts that make them up. Both pharmacological and toxic substances can induce oxidative stress and intracellular damage.

Examples of these include cocaine, ethanol, doxorubicin, and amphetamines. The most frequent mechanism of injury is ischemia, which results in infarction and the formation of scars. Following myocardial infarction, scar tissue replaces dead myocytes, adversely influencing myocardial function. This is shown on an echocardiogram as either absent or abnormal wall motion (akinesia or hypokinesia).[15]

➢ DIASTOLIC DYSFUNCTION OR HFpEF:

Diastolic dysfunction-related heart failure is commoly characterized by the ventricle's inability to relax sufficiently in the backward direction, which usually indicates a stiffer ventricular wall. Elevated end-diastolic pressures are a consequence of the failure of ventricular relaxation, and the outcome is the same as in the case of systolic dysfunction (peripheral edema in right heart failure, pulmonary edema in left heart failure).

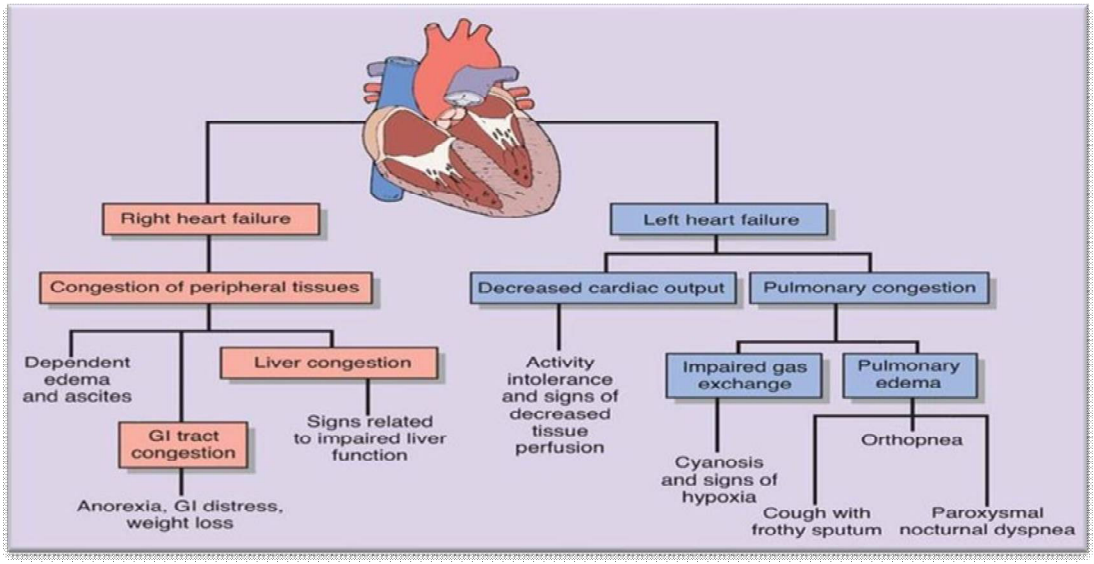
Similar processes that result in systolic dysfunction can also cause diastolic dysfunction; these include processes that impact cardiac remodeling.

If systolic function is maintained, diastolic dysfunction might not show up until physiologic extremes. At rest, the patient might not have any symptoms at all. They are, nevertheless, extremely sensitive to elevations in heart rate, and abrupt episodes of tachycardia—which may be brought on by pathological tachyarrhythmias like atrial fibrillation with rapid ventricular response, or simply by the body's reaction to exertion, fever, or dehydration—may cause flash pulmonary edema.

Therefore, it is crucial to maintain adequate rate control (usually with a pharmaceutical agent like a beta-blocker or calcium channel blocker that slows down AV conduction) in order to avoid acute decompensation.

Through echocardiography, a number of parameters, including the E/A ratio (early-to-atrial left ventricular filling ratio), the E (early left ventricular filling) deceleration time, and the isovolumic relaxation time, can be measured in order to determine the left ventricular diastolic function.[16]

c) FLOW CHART



- SIGNS AND SYMPTOMS: -

✓ Fatigue
✓ Activity limitation

- ✓ Congestion
- ✓ Edema or Ankle swelling, Shortness of breath, weight loss.
- ✓ Shortness of breath
- ✓ Weight loss

- PHYSICAL EXAMINATION

Upon physical examination, patients with heart failure need to have a thorough evaluation. Patients experiencing acutely decompensated heart failure or severe chronic heart failure may present with anxiety, diaphoresis, and poor nutritional status. In cases of acute decompensated heart failure, wheezing may occur. Sputum that is frothy and has a red tint may be observed as the severity of pulmonary congestion increases. It is crucial to understand that pulmonary congestion does not always result from the absence of rales. Another classic finding in all HF patients that needs to be evaluated is jugular venous distention.[17] There may be a Kussmaul sign, or paradoxical increase in jugular venous distention with respiration. In individuals whose left-sided filling is elevated Hepatojugular reflux, or distention of the jugular vein following application of pressure over the liver while the patient is lying at a 45° angle, is observed in patients with elevated left-sided filling pressures. When there is a significant level of volume overload, peripheral edema—which is present in severe heart failure—will be evident.

- ✓ Accentuation of P2, pulsus alternans, and S3 gallop are cardiac findings in HF patients. The most important and early discovery related to HF is an S3 gallop. There will be mitral and tricuspid regurgitation murmurs in patients with decompensated dilated cardiomyopathy.[18]

- LABORATORY INVESTIGATIONS

➢ Blood tests:

To determine the levels of vitals like including albumin ,hemoglobin ,RBC,WBC,Platelets, creatinine,Egfr (related to kidney function), sodium, chloride, potassium (electrolytes) and biomarkers like Creatine kinase-myoglobin binding,Troponin-T few other tests are performed in the diagnosis and prognosis of heart failure.

➢ Chest X-rays:

X-ray shows that whether the heart is enlarged or not.
Does the lungs have any congestion?
Whether your symptoms are caused by something other than heart failure

➢ Electrocardiogram (EKG or ECG):

An EKG shows that:

- ❖ If you've experienced a heart attack
- ❖ If the heart's muscle wall is enlarged and the left ventricle is thicker
- ❖ If there is an irregular cardiac rhythm (noting any arrhythmias like atrial fibrillation, or AFib)
- ❖ The regularity and force of your heartbeat.

➢ 2D -ECHO :

An echocardiogram shows that:

- ❖ The thickness of the heart muscle.
- ❖ The heart's pumping capacity.
- ❖ The size and shape of your heart.
- ❖ Your heart's ejection fraction.
- ❖ The state and operation of the tricuspid, pulmonic, mitral, and aortic heart valves.

➢ Exercise stress test:

- ❖ The results of an exercise stress test show:
- ❖ How your heart reacts to exercise.
- ❖ If the arteries supplying your heart have less blood flowing through them.
- ❖ The appropriate type and intensity of exercise for you.

- Which course of action is most likely to be effective for you.

➢ Multiple-gated acquisition scanning (MUGA) :

MUGA scans produce a film of several cardiac cycles by taking multiple images of your heart at various times. With the exception of an IV (intravenous drip line) at the beginning, the test is a painless process. The radionuclides used in the test have no long-term effectsThose who are at risk of developing heart failure as a result of receiving chemotherapy for cancer can benefit most from this test. By doing the test again, your medical team will be able to better understand how your heart works prior to, during, and following chemotherapy.

➢ MUGA scan shows:
- A clearer picture of the anatomy and physiology of your heart;
- The degree of blood flow to the heart muscle.
- The state of the heart's chambers.
- Whether part of the heart has been damaged by heart attack.
- The flow of blood through the heart
- How well your heart performs under stress and at rest.

➢ Cardiac catheterization:

The results of a cardiac catheterization include:
- Coronary artery blockages.
- Whether a lack of blood has weakened or damaged the areas of your heart that are supplied by blocked or narrowed arteries.
- The oxygen levels and blood pressure in your heart chambers and pulmonary arteries. [18]

➢ Non-pharmacological Therapy:
- Fluid restrictions in stage D patients < 2L/day
- Low salt diet <2g/day
- Exercise progression and rehabilitation program
- Lifestyle modifications : smoking cessation, alcohol limitations.
- Coronary revascularization
- Resynchronization

➢ Pharmacological Treatment:
- DRUG CLASSES IN THE STUDY

✓ ACE INHIBITORS:

- Angiotensin-converting enzyme inhibitors, or ACE inhibitors, are a class of drugs mainly used to treat heart failure and high blood pressure. This class of medication acts by reducing blood volume and relaxing blood vessels, which lowers blood pressure and lessens the heart's need for oxygen.
- The renin-angiotensin system, which hydrolyzes bradykinin and converts angiotensin I to angiotensin II, is facilitated by the angiotensin-converting enzyme, whose activity is inhibited by ACE inhibitors. Consequently, bradykinin, a peptide vasodilator, is increased and angiotensin II, a vasoconstrictor, is decreased in formation when ACE inhibitors are taken.In Blood pressure can be lowered more effectively by this combination.In The bradykinin system's ACE enzyme is inhibited by ACE inhibitor medications, which prevent bradykinin from being degraded and instead increase its levels. Prostaglandin is generated by bradykinin. The two most frequent adverse effects of ACE inhibitors can be explained by this mechanism: coughing and angioedema.
- ACE inhibitors such as benazepril, zofenopril, perindopril, trandolapril, captopril, enalapril, lisinopril, and ramipril are often prescribed.[19]

✓ Adverse effects :

Low blood pressure, coughing, hyperkalemia, headaches, fatigue, nausea, and kidney impairment are common side effects.[20]

✓ Blood:

Blood Hematologic side effects have been reported with ACE inhibitor therapy, particularly in individuals with additional risk factors. These side effects include neutropenia, agranulocytosis, and other blood dyscrasias.[21]

✓ Pregnancy:

It has been documented that ACE inhibitor use throughout all trimesters of pregnancy can result in congenital abnormalities, stillbirths, and neonatal deaths. Fetal abnormalities that are frequently reported include incomplete ossification of the skull, hypotension, renal dysplasia, anuria/oliguria, oligohydramnios, intrauterine growth retardation, pulmonary hypoplasia, and patent ductus arteriosus. In general, ACE inhibitor exposure causes negative side effects in about half of newborns, including birth defects.[22]

✓ Dosage :

All ACE inhibitors are administered orally, with the exception of enalapril, which is administered intravenously. The initial IV dose of enalapril is 0.625 to 1.25 mg every 6 hours. Updating the dosage to 5 mg IV every 6 hours is possible. It is imperative to commence geriatric dosing at the lower end of the adult dosage range.

Patients with heart failure, individuals with low salt levels, and/or patients with renal impairment should have their dosage reduced.

The only ACE inhibitors that work without the body having to activate them are captopril and lisinopril. Since they are prodrugs, all other ACE inhibitors must be activated. Most people reach their peak serum levels an hour after eating. It is better to use a non-prodrug form because the liver is where most of the activation takes place which is preffered in patients with underlying liver issues.[23]

✓ Contraindications:

ACE inhibitors are contraindicated in patients with a history of angioedema or hypersensitivity related to treatment with an ACE inhibitor and those with hereditary or idiopathic angioedema. [24]

➢ Angiotensin Receptor Blockers (ARBs)

Angiotensin receptor blockers, or ARBs, are a class of medication used to treat hypertension, congestive heart failure. ARBs bind to and inhibit the angiotensin type II receptor.They are frequently used to replace ACE inhibitor therapy in patients who are unable to tolerate ACE inhibitors due to a chronic, ineffective cough produced by the ACE inhibitor.

ARBs acts on RAAS system blockade can take place at several levels. RAAS-blockers include direct renin inhibitors (DRIs), which block the production of renin, ACEIs block conversion of AT1 to AT2 by blocking the angiotensin-converting enzyme, **ARBs** antagonize the effect of AII on AT1 receptors, and aldosterone antagonists block the effect of aldosterone. [24]

✓ Adverse Effects :

ARBs rarely cause side effects and are typically well tolerated. Because ARBs do not raise bradykinin levels, the incidence of angioedema and cough is lower with ARBs when compared with with ACEIs. Patients whose arterial blood pressure or renal function is heavily dependent on the RAAS may experience hypotension or renal failure when taking ARBs. Because of this, patients with hypotension from heart failure or bilateral renal artery stenosis should not take these medications.[25]

✓ Administration :

For lowering cardiovascular death in patients who are clinically stable but have a history of left ventricular dysfunction or failure (LVD) in the following ways:

Heart attack: 20 mg twice a day as the starting dose, and 160 mg bid as the maximum amount per day.

✓ Contraindications:

ARB medication during pregnancy decreases the fetal kidney's perfusion and is associated with renal dysgenesis, oligohydramnios, fetal anuric or oliguric renal failure, skeletal or skull abnormalities, pulmonary hypoplasia, and fetal death.
According to the FDA, ARBs pose a category D risk when used during pregnancy.

When prescribing ARBs to patients with renal disease or those taking agents that can cause hyperkalemia (K+ supplements, K+ sparing diuretics, ACEIs, DRIs, non-steroidal anti- inflammatory agents), caution must be taken or the drug should be avoided completely.[26]

- ARNIs(Angiotensin Receptor Neprilysin Inhibitor)

Angiotensin receptor neprilysin inhibitors (ARNI) are a new class of drugs, the first of which is sacubitril/valsartan. For the treatment of patients with chronic heart failure with reduced ejection fraction (HFrEF) and NYHA classes II, III, or IV, the drug has FDA approval. In addition to other common treatments for heart failure (beta-blocker, aldosterone antagonist), sacubitril/valsartan is to be used in place of ACEIs or angiotensin II receptor blockers (ARBs).[27]
The product sacubitril/valsartan is a combination. Pro-drug sacubitril functions as an inhibitor of neprilysin once it is activated. The mechanism of action involves inhibiting neprilysin's ability to break down natriuretic peptides, thereby prolonging the beneficial effects of these peptides.
By blocking the RAAS system, valsartan, an angiotensin receptor blocker, functions. Angiotensin II is broken down by neprilysin, so blocking neprilysin will cause angiotensin II to build up instead. The effect of the excess angiotensin II is therefore blocked when a neprilysin inhibitor is used in conjunction with an ARB; it cannot be used alone.
Bradykinin, an additional significant material metabolized by neprilysin, accumulates when neprilysin is inhibited. Since ACEI and ARNI dosages should not be taken together or within a short period of time, sacubitril cannot be used with an ACEI due to the increased risk of angioedema. To reduce the risk of angioedema, a 36-hour washout period must be completed by the patient when switching between ACEI and sacubitril/valsartan.[28]

- Administration:

Individuals who have never taken an ARB or ACEI before or who are on low-dose ARB or ACEI start off taking sacubitril 24 mg/valsartan 26 mg twice day. Up to sacubitril 97 mg/valsartan 103 mg taken twice daily orally, double the dosage every two to four weeks as tolerated.
Patients starting on a moderate to high dosage of ACEI or ARB should take 49 mg of sacubitril and 51 mg of valsartan twice a day. Up to sacubitril 97 mg/valsartan 103 mg orally twice day, double the dosage every two to four weeks as tolerated.[29]

- Adverse effects :

Adverse effects include hypotension, hyperkalemia, renal failure, cough, and angioedema.

- Contraindications:

Individuals who suffer from the following ailments shouldn't take valsartan or sacubitril:
Intolerance to any component present in the product.
A prior history of angioedema following an ACEI or ARB.
In diabetic patients taking renin inhibitors, valsartan (or any ARB) should not be used in combination with aliskiren due to an increased risk of hypotension, hyperkalemia, and renal impairment.
Patients who have taken an ACE inhibitor within the previous 36 hours and are at increased risk of developing angioedema.[30]

- Beta Blockers:

Patients with compensated congestive heart failure are treated with beta-blockers. Metoprolol succinate, bisoprolol, and carvedilol are specifically the beta-blockers that are frequently prescribed.
When the catecholamines, norepinephrine, and adrenaline, bind to B1 receptors, cardiac conduction velocity and automaticity are increased. Additionally, renin release is induced by B1 receptors, and this raises blood pressure. On the other hand, smooth muscle relaxation and enhanced metabolic effects like glycogenolysis are brought about by binding to B2 receptors.

These effects are blocked by beta-blockers once they attach to the B1 and B2 receptors. As a result, the heart rate decreases due to the inhibition of the chronotropic and inotropic effects on the heart. Moreover, beta-blockers lower blood pressure in a number of ways including decreased renin and reduced cardiac output. The negative chronotropic and inotropic effects lead to a decreased oxygen demand.[31]

- ❖ DOSAGE:

Beta-blockers are taken as orally,intramuscular,intra venous route of administration.

Outpatients are prescribed with daily once dosage of longer-acting beta-blockers, such as metoprolol succinate 25,50mg. Conversely, the majority of beta blockers is necessary at least twice daily. Propranolol, for example, has a half-life of approximately 4 hours, so it is possible to dose it up to 3 or 4 times a day depending on the indication and dosage.[32]

- ❖ Adverse effects:

The following side effects of beta blockers include bradycardia, hypotension, heart failure, heart block, alopecia (hair loss), exhaustion, dizziness, abnormal vision, hallucinations, insomnia, nightmares, sexual dysfunction, erectile dysfunction, nausea, diarrhea, bronchospasm, dyspnea, cold extremities, increases in the severity of Raynaud's syndrome, and/or modifications in the metabolism of glucose and lipids.

Orthostatic hypotension is often associated with treatment with mixed α1/β-antagonists. Ecdyma is often associated with carvingilol treatment. Metoprolol and propranolol, two lipophilic beta blockers with a high blood-brain barrier penetration, are more likely to induce sleep disturbances like insomnia, vivid dreams, and nightmares than other less lipophilic beta blocker.

- ❖ Contraindications :

In the past, patients with asthma have not been allowed to take beta-blockers. Though not nonselective beta-blockers, guidelines now support the use of cardio-selective beta-blockers, also referred to as beta-1 selective, in asthmatics. Patients suffering from asthma should not be treated with non-selective beta-blockers.

Patients with hypotension, either acute or chronic, and bradycardia are relative contraindications for beta-blocker use. [33]

- ❖ Selective Agents :

β1-selective beta blockers are also known as cardioselective beta blockers.Pharmacologically, cAMP will be affected by the beta-blockade of the heart's β1 receptors. In the heart cell, cAMP acts as a second messenger by phosphorylating the ryanodine receptor and the LTCC, which raises intracellular calcium levels and induces contraction. Medications may act on β1 receptors exclusively in the heart, or they may be cardioselective, but they may still have intrinsic sympathomimetic activity.

Eg: Metoprolol Succinate, Bisoprolol, Carvedilol

- ❖ Mineralocorticoid receptor antagonist:

An aldosterone antagonist, also known as a mineralocorticoid receptor antagonist (MRA), is a diuretic medication that inhibits the effects of aldosterone at mineralocorticoid receptors.

These medications are frequently used in conjunction with other medications as adjunctive therapy to treat chronic heart failure. The first drug in the class, spironolactone, is also used to treat hyperaldosteronism, which includes Conn's syndrome, and female hirsutism because it has extra antiandrogen effects. Spironolactone is one of the majority of anti-mineralocorticoids that are steroidal spironolactone. A nonsteroidal anti-mineralocorticoid is finerenone.[34]

- ❖ Dosage:

Oral tablets containing 25 mg, 50 mg, or 100 mg of ALDACTONE are the recommended dosage for this medication. The maximum daily dose is directed by the Physician.

- Each day, 50 mg for HFrEF.
- For resistant hypertension,100 mg per day.
- 200 mg per day for edema brought on by nephrotic syndrome or 400 mg per day for edema brought on by cirrhosis.
- For primary hyperaldosteronism, 400 mg per day.
- 200 mg per day for hypokalemia brought on by diuretics.

❖ Adverse Effects:

One of the most often reported side effects is increased urination; this is usually temporary and goes away with continued treatment. It is especially noticeable in the early stages after starting treatment.

Symptoms of antimineralocorticoids that are frequently experienced include nausea, vomiting, cramping in the stomach, and diarrhea. It is possible to have clinically significant hyperkalemia, which calls for routine serum potassium monitoring. The pathophysiology of hyperkalemia is due to the reduction in potassium (K) excretion caused by antimineralocorticoid medications.

❖ Contraindications:

Side effect of spironolactone is hyperkalemia. Patients who already have hyperkalemia or who are more likely to develop it should avoid this medication. Individuals with renal impairment is preferred for discontinuation.[35] SGLT2 inhibitors (sodium-glucose transport protein 2) :

SGLT-2 inhibitors,acts by proteins expression in the proximal convoluted tubules, are antihyperglycemic agents. These medications work by stopping the tubular lumen's filtered glucose from being reabsorbed.

The Food and Drug Administration (FDA) has approved four SGLT-2 inhibitors for adult use:

canagliflozin, dapagliflozin, empagliflozin, and ertugliflozin.[36]

- FDA-approved indications for SGLT-2 Inhibitors
- Glycemic control in type 2 diabetes mellitus is improved when diet and exercise are combined.
- Lowering the risk of significant adverse cardiovascular events in patients with type 2 diabetes and established cardiovascular disease, such as nonfatal myocardial infarction, nonfatal stroke, and cardiovascular death.
- Reducing heart failure patients' risk of cardiovascular hospitalization and death (HFrEF, or heart failure with reduced ejection fraction; NYHA class II–IV).
- Lowering the chance of hospitalization and eGFR decline in individuals with chronic kidney disease who are at risk of the condition getting worse.
- Better cardiovascular outcomes for those suffering from heart failure with preserved ejection fraction, or HFpEF.
- The FDA has approved dapagliflozin for the treatment of heart failure encompassing all LVEF ranges, including HFrEF, HFpEF, and HFmrEF (heart failure with mildly reduced ejection fraction, or LVEF of 40–49%).[37]

❖ Mechanism of action:

The kidneys' proximal convoluted tubules express SGLT-2 proteins, which carry out their physiological role by reabsorbing filtered glucose from the tubular lumen. The renal threshold for glucose (RTG) is lowered, urine glucose excretion is encouraged, and the reabsorption of filtered glucose is decreased by all four SGLT-2 inhibitors.

Inhibitors of SGLT2 reduce HbA1c by 0.7%.[38]

❖ Dosage of TYPE 2 Diabetes Mellitus :

- Canagliflozin: A 100 mg daily starting dose may be increased to a 300 mg daily dose. (If eGFR is less than 30 mL/min/1.73 m², it is not advised to enhance glycemic control.)
- Dapagliflozin: If eGFR is less than 45 mL/min/1.73 m², do not take this medication. The starting dose is 5 mg once daily; increase to 10 mg once daily to reach the desired glycemic goal.
- Empagliflozin: To reach the desired glycemic response, the starting dose of 10 mg once daily may be increased to 25 mg daily.

To reach the glycemic target, the starting dose of entretugliflozin is 5 mg once daily. This is subsequently increased to 15 mg daily. (It is not advised to enhance glycemic control if eGFR is less than 45 mL/min/1.73 m².)

❖ HFrEF (Heart failure with reduced ejection fraction):

According to AHA/ACC/HFSA(2022) guidelines, guideline-directed medical therapy (GDMT) for heart failure with reduced ejection fraction (HFrEF) includes sodium-glucose cotransporter-2 inhibitors. SGLT2 inhibitors, independent of type 2 diabetes, are

recommended to reduce hospitalization associated with heart failure and cardiovascular mortality in patients with chronic symptomatic HFrEF.

Preferred as follows:
10 mg of dapagliflozin and 10 mg of empagliflozin once daily

- HFpEF (Heart Failure with preserved ejection):
- All patients with HFpEF should take SGLT2 inhibitors, per the 2023 ACC Expert Consensus. The suggested agents as follows:
- 10 mg of dapagliflozin once a day.
- 10 mg of empagliflozin once day.[39]

❖ Adverse Effects:

Constipation, nausea, increased urination, female genital mycotic infections, and urinary tract infections, urosepsis ,diabetic ketoacidosis, are the most commonly reported side effects associated with SGLT-2 inhibitors.Hyperkalemia: In patients with renal impairment, canfliglozin is linked to an increased risk of hyperkalemia, particularly when combined with ACE inhibitors or ARBs.[40]

❖ Contraindications:

It is not recommended for patients undergoing dialysis to receive any of the four SGLT-2 inhibitors. Additionally, there is an absolute contraindication for any hypersensitivity reaction to any of the four agents, including angioedema or anaphylaxis.[41]
Examples :Dapagliflozin,Empagliflozin,Canagliflozin,Ertgliflozin.

II. AIM AND OBJECTIVES

A. Aim

1) To identify the compliance of the drug in the cardiology department.
2) To analyse the reasons for discontinuation of therapy.

B. Objectives

1) To evaluate the demographic details of the population.
2) To evaluate the past history and risk factors of individuals with Heart Failure.
3) To study the prescription pattern and to determine most frequently prescribed drugs.
4) To evaluate the patient compliance towards the Heart Failure.

III. NEED FOR THE STUDY

1) To assess the Safety, Efficacy and Tolerability of prescribed drug.
2) Assessment of subject quality of life is as important as treatment outcome.
3) Cost-effectiveness of the treatment should be considered for the subject convenience.

IV. REVIEW OF LITERATURE

1) Ponikowski P, Voors AA, Anker SD et al.,(2016 Jul 14) practice guideline study shows that in order to help health professionals choose the best management strategies for a specific patient with a given condition, guidelines under[2012-16] synthesize and assess all available evidence at the time they are written, taking into account the impact on outcome as well as the risk-benefit ratio of various diagnostic or therapeutic approaches. Health professionals should find guidelines and suggestions helpful when making judgments in their day-to-day work. The responsible health professionals must, however, make the ultimate choices pertaining to a specific patient after consulting with the patient and caregivers if necessary.
2) Yancy CW, Jessup M, Bozkurt et al., (2013 Oct 15) prospective observational study Guidelines for cardiovascular disease have been created jointly by the American College of Cardiology Foundation (ACCF) and the American Heart Association (AHA). since 1980. The Task Force of the ACCF/AHA Task Force on Practice Guidelines is responsible for creating, revising, and updating the practice recommendations for cardiovascular disorders and supervises, coordinates, and procedures this

endeavour. Regular reviews and evaluations are required of writing committees. all relevant data to create fair, patient-centred, suggestions for clinical action.

3) Ammar KA, Jacobsen SJ et al.,(2007) Mar 27 as in this comparative study - To emphasize early identification and prevention, an American Heart Association/American College of Cardiology staging classification of HF has been created. In a community cohort,
Notably, stage A (risk factors) or stage B was identified in 56% of persons aged 45 or older (asymptomatic ventricular dysfunction)so we can conclude that the prevalence of the population at risk for developing overt HF is highlighted by HF staging.

4) Murphy J, Coster G et al.,(1997 Dec) as per review of clinical journal study; the patient compliance plays a major role in the therapeutic diagnosis of the disease. The negligence of the patient towards drug utilisation during discharge period may show severe consequences on patient morbidity, death and cost of usage for treatment.

5) Virani SS, Alonso A et al., (2021 Jan 27)as per the circular- study we can conclude that The Statistical Update also provides the most recent information on a number of significant clinical heart and circulatory disease conditions (such as stroke, congenital HD, rhythm problems, subclinical atherosclerosis, coronary HD [CHD], heart failure [HF], valvular HD, venous disease, and peripheral artery disease) and the outcomes (such as the standard of care, procedures, and financial costs). The Statistical Update yearly reports have been referenced in the literature more than 20.000 times since 2007.

6) Rudolph W et al.,(1990 Jun1) as per analytical study -conclude that it refers to ventricular dysfunction due to an abnormality of the heart which is associated with typical hemodynamic, renal, and hormonal reactions, characterizes the clinical syndrome heart failure.

7) Schwinger RH et al.,(2021 Feb 11) As per the practical guidelines study -it concludes that it goes with determining the appropriate therapy approach uniquely for each patient, it is crucial to understanding the underlying pathophysiology of heart failure. The chance of heart failure must also be reduced by preventing cardiovascular risk factors.

8) van der Meer et al.,(2019 Jun 4). A Clinical guideline study concludes that - The American College of Cardiology/American Heart Association's 2013 (with revisions in 2016 and 2017) and the European Society of Cardiology's 2016 guidelines offer useful, evidence- based clinical recommendations for the diagnosis and management of both acute and chronic heart failure (HF).

9) Thummak S,Uppor W,Wannarit Let al.,(2023) as per concept analysis provides a valuable perspective on patient compliance that guides the nursing practice in providing better interventions to promote better compliance among patients.

10) Boron-Walter F, Boulpaep-E et al., (2005) findings of this meta-analysis suggest a strong positive association between hypertension and systolic and diastolic blood pressure and the risk of heart failure. For more information we have to analyse the structural and functional parameters of heart.

11) Shigeyama J, Yasumura Y et al.,the analytical study shows that treatment with β- blockers inhibits the expression of collagen mRNA, which is linked to cardiac sympathetic nerve activity. It appears that cardiac TGF-β1 is the mediator of this inhibition. β-blockers affect this collagen metabolism helps to explain some of their therapeutic benefits.

12) Magnussen C, Niiranen et al., (2019 Mar) the study concludes that compared to men, women were less likely to develop HF. There were differences between the sexes in terms of heart rate, systolic blood pressure, CRP, and NT-proBNP, with women having a lower risk of heart failure.

13) Emmons-Bell S, Johnson C, Roth G. Prevalence et.,al (2022 Jan 18). analytical studies had very different heart failure prevalence, incidence, and survival rates, which is indicative of different study designs. Heart failure is still a disease that affects a large percentage of older adults and has a high one-year mortality rate. Everything that has been published about the burden of heart failure is compiled in this review. In the future, estimates of the global disease burden from heart failure will be produced through the use of geospatial statistical models. An enhanced comprehension of heart failure in the general population would be beneficial in directing research, allocating resources and policies, and informing broader initiatives to lessen the prevalence of non-communicable diseases, given its role as a common terminal pathway for a wide range of conditions.

14) J Am Coll Cardiol HF et.,al (2022 Feb,10) the meta-analysis support treatment of patients with HFrEF with a combination of ARNi, BB, MRA, and SGLT2i. The number of life-years gained with four classes are mentioned as best four pillars of heart failures.

V. METHODOLOGY

A. Material And Methods

1) Type of study:

This is a prospective observational study.

2) Study Site:

This study conducted in the in-patient and out-patient department of Cardialogy, Krishna Institute of Medical Sciences(KIMS) Hospital, Secunderabad.

3) Study Duration:

This study was carried out over six months period.

4) Sample Size:

100 Participants were enrolled in this study.

B. STUDY CRITERIA:

1) Inclusion Criteria:

- Adults >18years.
- Diabetic and Non-Diabetic Patients.
- Both Male and Female.
- Outpatient and Inpatient Data
- Patient giving their consent.
- All CVD patients, greater than 18 years old.
- Receiving multiple classes of drugs.
- Treated as inpatients for at least 72 hours at the cardiology ward or cardiac intensive card units.

2) Exclusion Criteria:

- Patient not giving consent.
- Pregnant and lactating women.
- Pediatrics and young adults<18 years.

3) Source of Data:

All the relevant and necessary data will be collected from patient records, lab reports, and prescription from inpatient department.

- Communicating with healthcare professionals.

C. Ethical Consideration:

Institutional human ethics committee approval consent will be taken. Informed written consent will be obtained from all the study participants, and only those participants willing to sign informed consent will be included in the study and the confidentiality of the study participants will be maintained.

D. Institutional Review Board:

This protocol and the corresponding informed consent form (ICF) used to obtain the informed consent of study patients were reviewed and approved by the ethics committee of the scientific review board. The protocol and the ICF for the study were reviewed and approved by the Ethics Committee.

E. Statistical Analysis:

The studies goals were met by displaying categorical data using frequencies and percentages, continuous variables were presented as standard deviation.

Chi-Square test/Fisher exact test was used to test the differences between proportions as appropriate.

P-Value <0.05 was considered as the statistically significant.

F. Proposed plan of work: -

- Preparation of protocol and data copy.

- Getting permission from the ethics committee.
- All those patients who meet the study criteria will be included in the study after obtaining informed consent.
- During the initial visit, collection baseline information of the patients with study criteria and the collected data will be documented.
- Patient's demographics data, other comorbidities conditions, the medication details, Duration of the therapy, the cover-up of the patients, and all other required data will be collected from various data sources.
- Medication details, mainly about the dosage and its frequency along with drug interactions are documented for the analysis of the patient condition.
- The obtained data will be analysed using suitable statistical methods

G. Conflict of Interest: Nill

VI. RESULTS AND DISCUSSIONS

In this study, a total of 100 subjects were taken into consideration and accordingly their prescriptions pertaining to heart failure medications were thoroughly studied/examined and their adherence to the medication was noted.

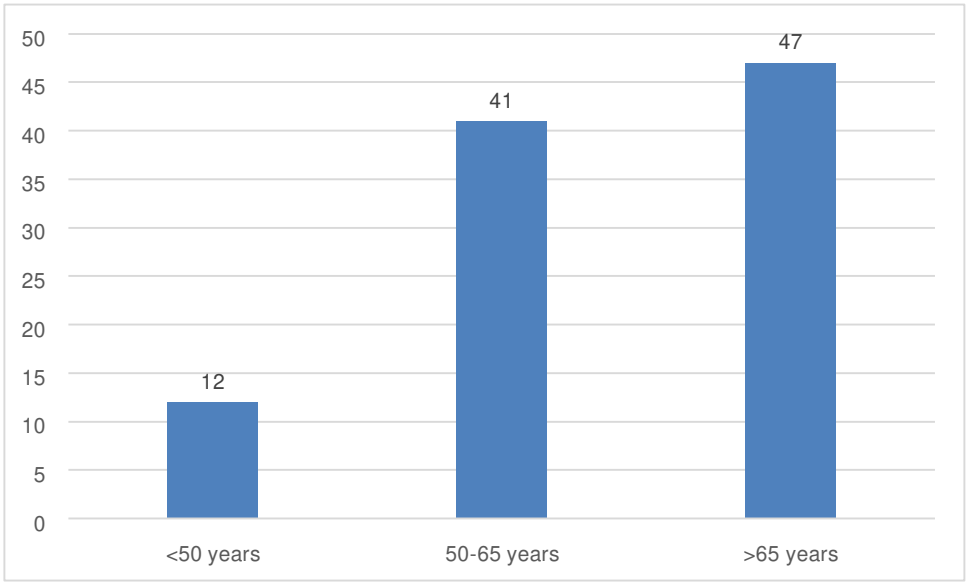
Analysis of prescription data revealed the frequent use of certain four classes of drug. These insights contribute to enhancing heart failure management strategies, emphasizing tailored interventions for improved patient outcomes. The following are the no worthy findings revealed as per the study.

1) Age wise distribution of the subjects:

Table no. 1- Frequency and percentage of subjects according to age.

Age group	Frequency(n)	Percentage(%)
<50 years	12	12
50-65 years	41	41
>65 years	47	47
Total	100	100
Mean	63.89 (11.32)	

Figure no: 3 Bar diagram representing frequency and percentage of subjects according to age.



DISCUSSION:-

In this study, ages ranged from 18 to above 65 years.

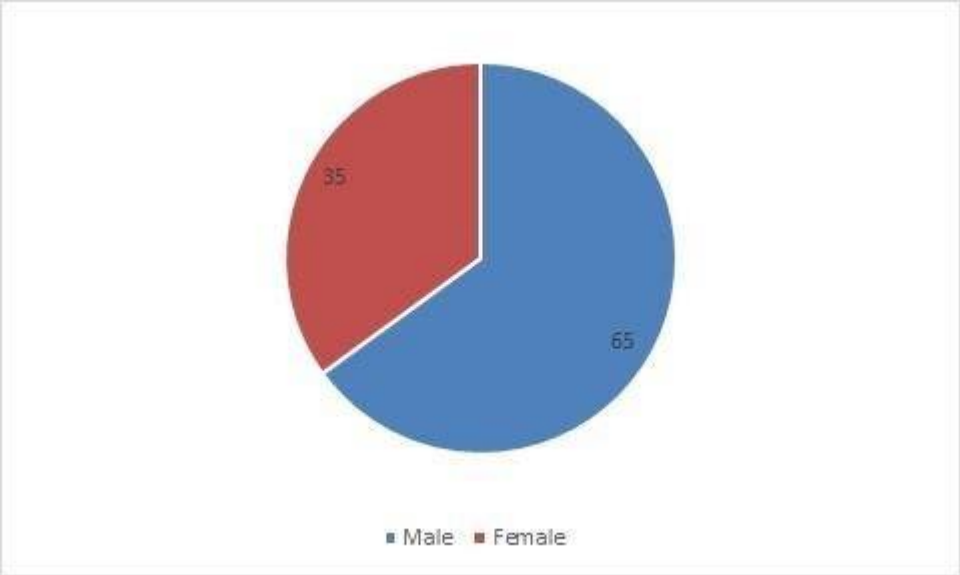
In our study, age more than 65 years are more likely risk of heart failure and less likely for below 50 years . The majority of the patients with heart failure lies in the age group interval of above 65 years was 47% (47 of 47) and 50-65 years was 41% (41 of 41), below 50 years was 12% (12 of 12) respectively.

2) Gender wise distribution of the subjects:

Table no: 2 – Frequency and percentage of subjects according to the gender.

Gender	Frequency(n)	Percentage (%)
Male	65	65
Female	35	35
Total	100	100

Figure no: 4 – Pie diagram representing frequency and percentage of subjects according to gender.



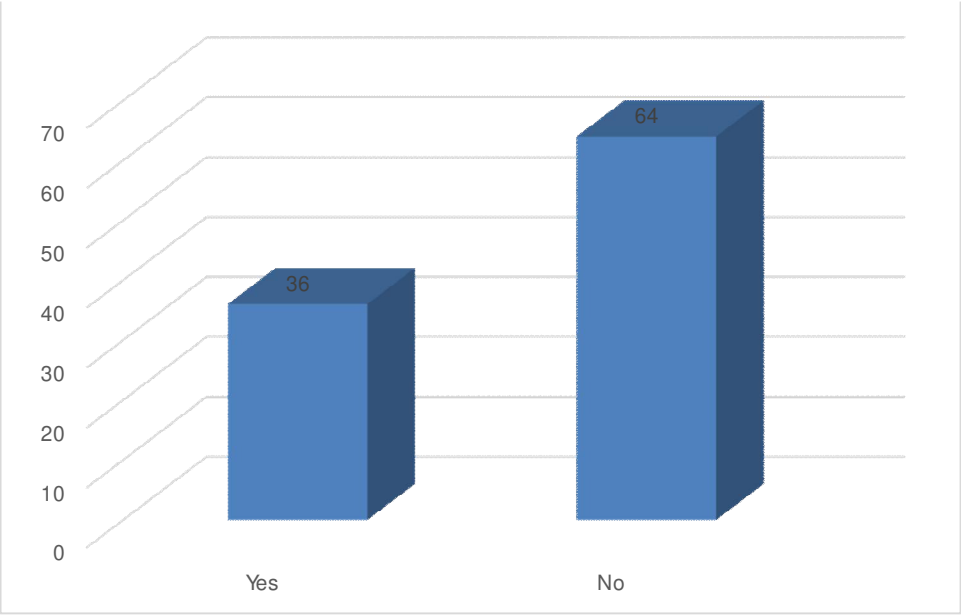
DISCUSSION:-

In our study, the distribution subjects according to gender wise causing heart failure after was (65% & 35%) respectively. This study shows males are predominance.

Table no – 3 Frequency and percentage distribution of heart failure subjects according to the Diabetes.

Diabetes	Frequency(n)	Percentage (%)
Yes	36	36
No	64	64
Total	100	100

Figure no: 5 - Bar diagram represents frequency and percentage distribution of heart failure subjects according to the Diabetes.



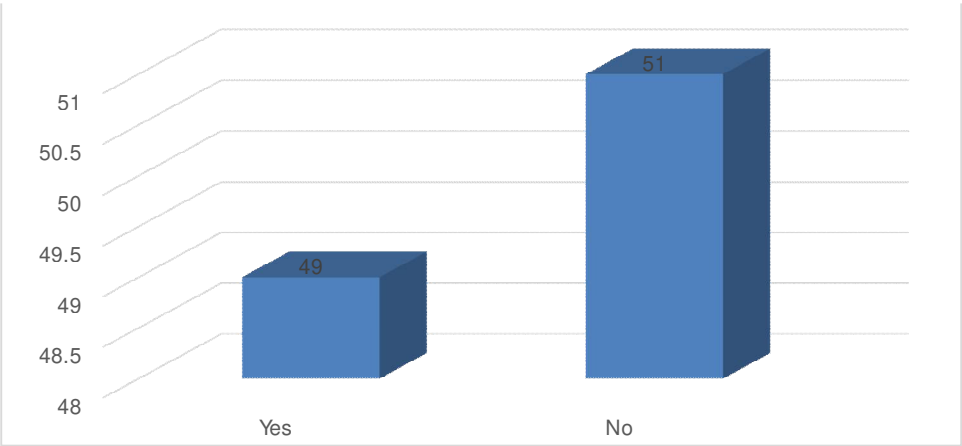
DISCUSSION:-

In our study, the distribution subjects according to diabetes wise causing heart failure was 36%(36 of 36) respectively, and the subjects who don't have diabetes are 64% (64 of 64) respectively.

Table No: 4 Frequency and percentage distribution of heart failure subjects according to Hypertension

Hypertension	Frequency	Percentage
Yes	49	49
No	51	51
Total	100	100

Figure no: 6 - Bar diagram represents frequency and percentage distribution of heart failure subjects according to the Hypertension.



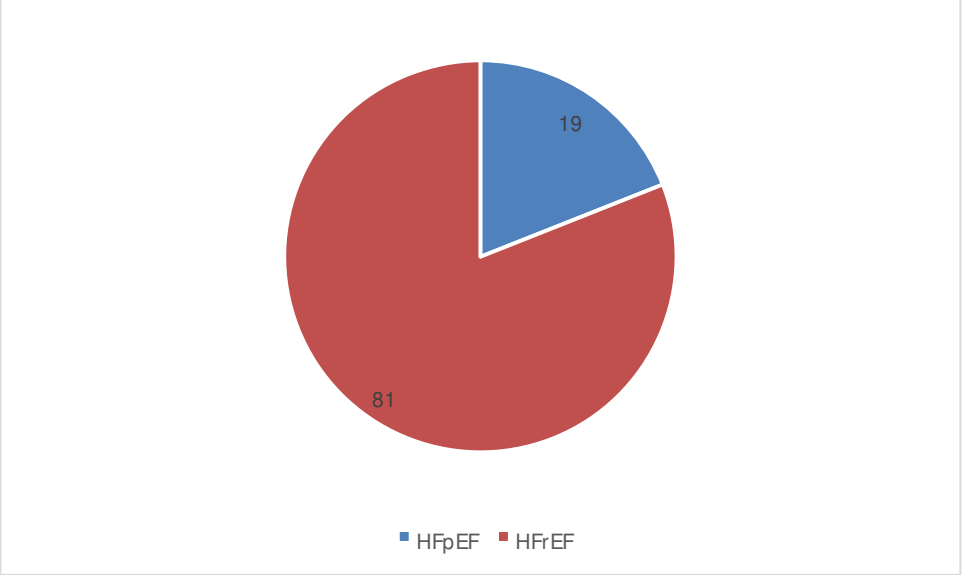
DISCUSSION:-

In our study, the distribution subjects according to hypertension wise causing heart failure was 49% (49 of 49) respectively, and the subjects who don't have diabetes are 51% (51 of 51) respectively.

3) Frequency and percentage distribution of heart failure subjects according to Ejection Fraction

EF	Frequency	Percentage
HFpEF	19	19
HFrEF	81	81
Total	100	100

Figure no: 7 - Pie diagram represents frequency and percentage distribution of heart failure subjects according to the Ejection Fraction.



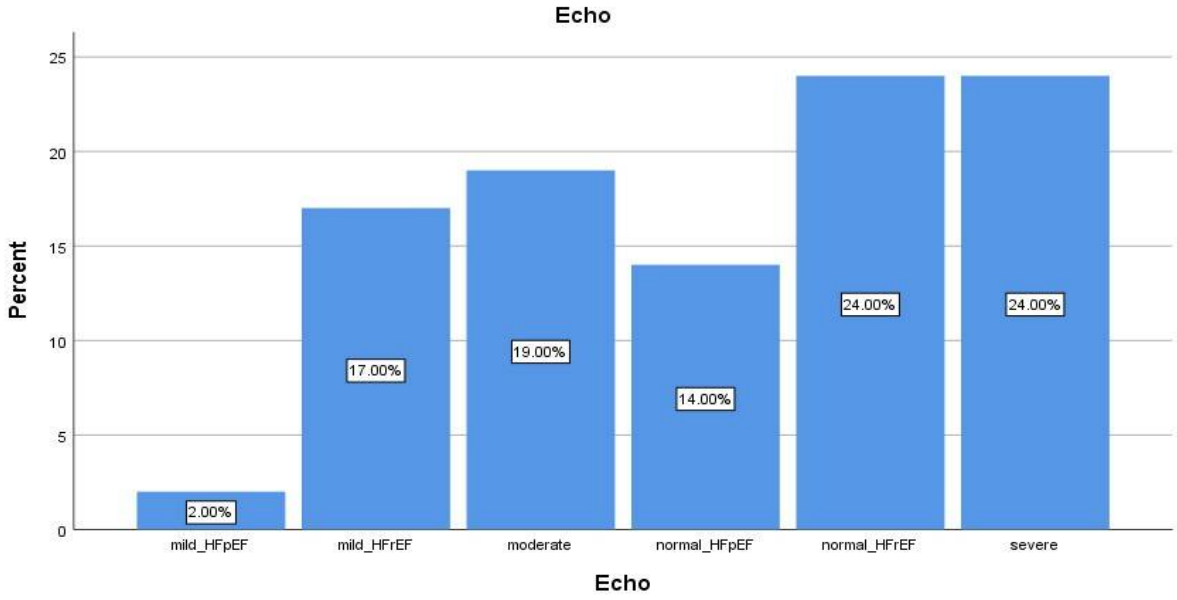
DISCUSSION: -

In our study, the distribution subjects according to ejection fraction wise causing heart failure was 16%(16 of 16) of HFpEF respectively, and the subjects according of HFrEF are 84% (84 of 84) respectively.

4) Frequency and percentage distribution of heart failure subjects according to 2D Echo severity

2D Echo severity	Frequency	Percentage
Mild LV Dysfunction HFpEF	2	2
Mild LV Dysfunction	17	17
Moderate LV Dysfunction	19	19
Normal HFpEF	14	14
Normal HFrEF	24	24
Severe LV Dysfunction	24	24
Total	100	100

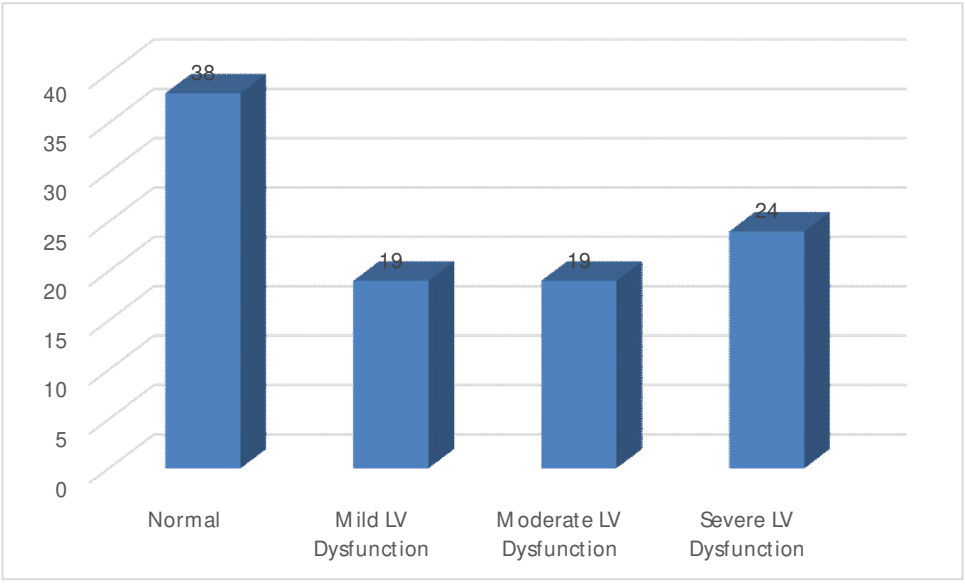
Figure no: 8 - Bar diagram represents frequency and percentage distribution of heart failure subjects according to the 2D Echo severity.



5) Frequency and percentage distribution of heart failure subjects according to 2D Echo severity

2D Echo severity	Frequency	Percentage
Normal	38	38
Mild LV Dysfunction	19	19
Moderate LV Dysfunction	19	19
Severe LV Dysfunction	24	24
Total	100	100

Figure no: 9 - Bar diagram represents frequency and percentage distribution of heart failure subjects according to the 2D Echo severity.



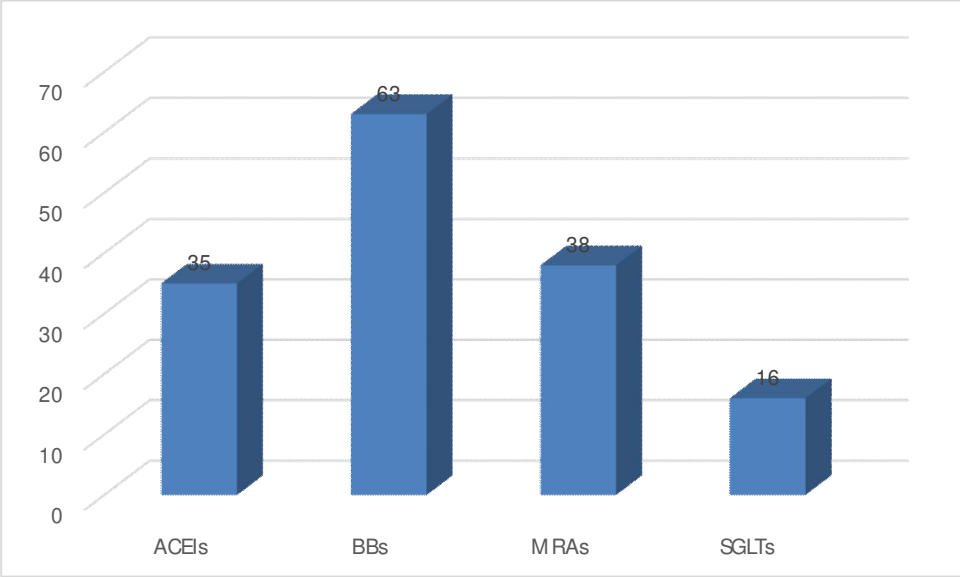
DISCUSSION:-

In our study, the distribution subjects according to 2D Echo severity wise causing heart failure was 14%(14 of 14) of normal HFpEF ,and the subjects according to normal HFrEF was 24% (24 of 24), the subjects with mild LV dysfunction of HFrEF are 17% (17 of 17) ,and the subjects with mild LV dysfunction of HFpEF are 2% (2 of 2), and the subjects with moderate LV dysfunction was 19% (19 of 19), the subjects with severe LV dysfunction was 24% (24 of 24) respectively.

6) Frequency and percentage distribution of heart failure subjects according to classes of drugs.

Type of drug	Frequency	Percentage
ACEIs	35	35
BBs	63	63
MRAs	38	38
SGLTs	16	16

Figure no: 10 - Bar diagram represents frequency and percentage distribution of heart failure subjects according to the classes of drugs



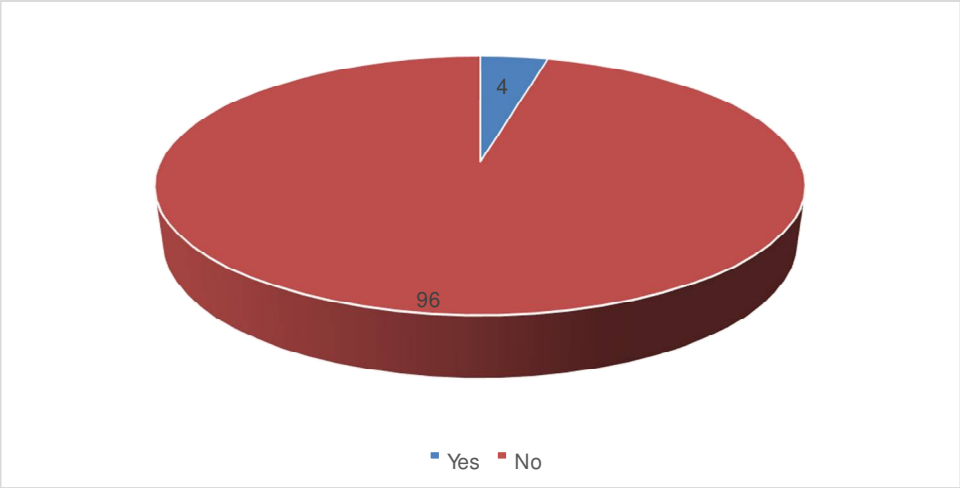
DISCUSSION:-

In our study , the distribution of total number of subjects were 100 and for each class the percentages were calculated according to individual percentage class of drugs among those subjects ,out of them Beta-Blockers took by the heart failure subjects was 63% (63 of 100), MRA's took by the heart failure subjects was 38% (38 of 100), ACE's took by the heart failure subjects was 35% (35 of 100) and SGLT-I took by the heart failure subjects was 16% (16 of 100) respectively.

7) Frequency and percentage distribution of heart failure subjects according to discontinuation of drugs.

Discontinuation	Frequency	Percentage
Yes	4	4
No	96	96
Total	100	100

Figure no: 11 - Pie diagram represents frequency and percentage distribution of heart failure subjects according to the discontinuation of drugs.



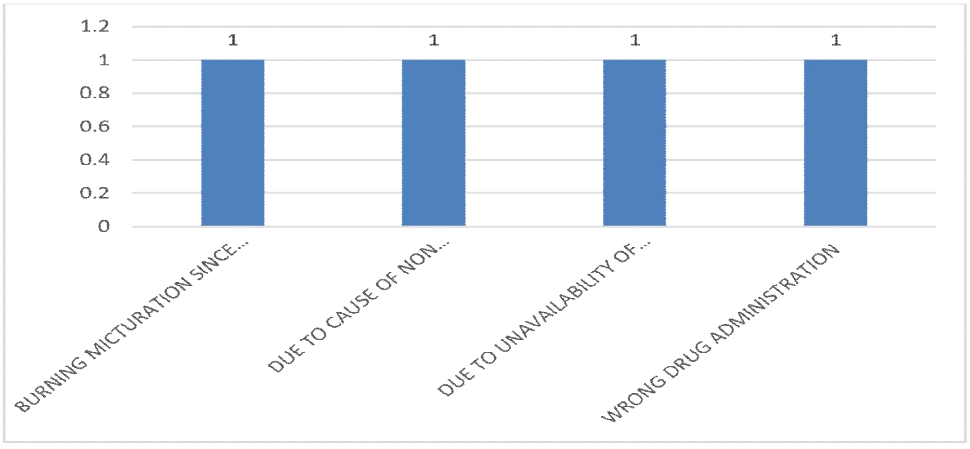
DISCUSSION: -

In our study, the distribution of subjects according to discontinuation frequency out of 100 subjects ,the subjects who discontinued the medication was 4% (4 of 100) and continued the medication was 96% (96 of 100) respectively.

8) Frequency and percentage distribution of heart failure subjects according to reasons for discontinuation of drugs.

Reasons for discontinuation	Frequency
BURNING MICTURATION SINCE 10 DAYS CUE WAS DONE AND PUS CELL WERE OBSERVED THAT IS 20CELLS FOR HF	1
DUE TO CAUSE OF NON TOLARANCE[PYELONEPHRITIS]	1
DUE TO UNAVAILABILITY OF PARTICULATE DRUG	1
WRONG DRUG ADMINISTRATION	1

Figure no: 12 - Bar diagram represents frequency and percentage distribution of heart failure subjects according to the reasons for discontinuation of drugs.



DISCUSSION: -

In our study, distribution of subjects according to reason for discontinuation out of 100 subjects was 4% (4 of 100) were discontinued due to the above reasons mentioned in the table.

9) Frequency and percentage distribution of heart failure subjects according to reasons for discontinuation of drugs.

Reasons for discontinuation	Type of drug
BURNING MICTURATION SINCE 10 DAYS CUE WAS DONE AND PUS CELL WERE OBSERVED THAT IS 20CELLS FOR HF	ACEI and SGLT
DUE TO CAUSE OF NON TOLARANCE[PYELONEPHRITIS]	SGLT
DUE TO UNAVAILABILITY OF PARTICULATE DRUG	SGLT
WRONG DRUG ADMINISTRATION	SGLT and MRA

DISCUSSION: -

In our study, total subjects included 100, out of those ,the distribution of subjects according to reason for discontinuation type of drug as mentioned in above table,who ever have taken SGLT-I's show major contraindications and was 4% (4 of 100) and other class of drugs show less contraindications each ,and was 1% (1 of 100) respectively.

10) Distribution of lab parameters based upon standard deviation.

Descriptive Statistics				
Lab parameters	Minimum	Maximum	Mean	Std. Deviation
SERUM CREATININE	0.24	6.70	1.42	0.78
E-GFR	1.07	88.74	51.09	24.90
CK-MB	0.62	150.20	8.85	23.83
TROP-T	0.01	1.91	0.25	0.49

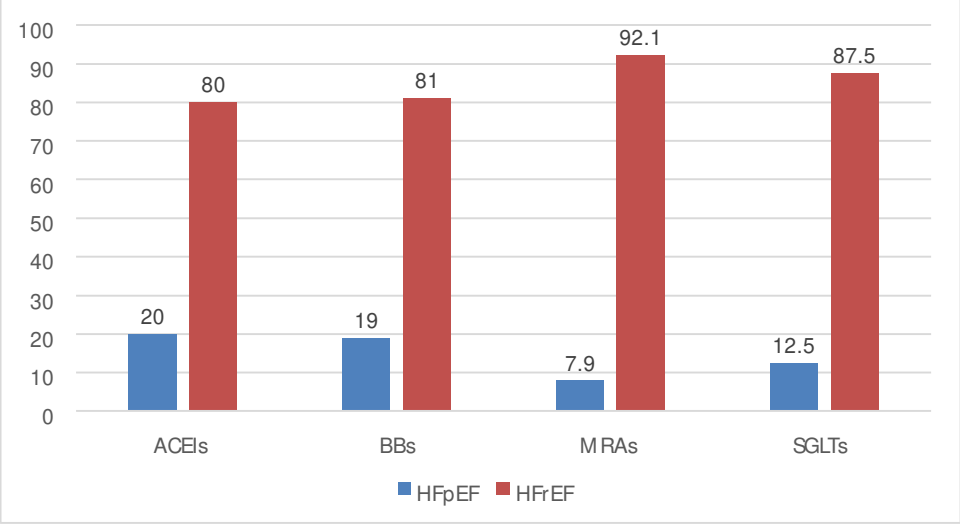
DISCUSSION:-

In our study , investigation parameter showed serum creatine minimum level as of 0.24 and maximum showed 6.70 and mean is 1.42 (Sd- 0.78).E-GFR showed minimum level as 1.07, maximum level as 88.74 and mean is 51.09 (Sd-24.90),CK-MB showed minimum level as 0.62, maximum level as 150.20 and mean is 8.85(Sd-23.83), TROP-T showed minimum level as 0.01, maximum level as 1.91, mean is 0.25 (Sd-0.49) respectively

11) Frequency and percentage distribution of classes of drugs based upon HFpEF and HFrEF.

Type of drug	HFpEF	HFrEF
ACEIs (n=35)	7 (20)	28 (80)
BBs (n=63)	12 (19)	51 (81)
MRAs (n=38)	3 (7.9)	35 (92.1)
SGLTs (n=16)	2 (12.5)	14 (87.5)

Figure no: 13 - Bar diagram represents frequency and percentage distribution of classes of drugs based upon HFpEF and HFrEF.



DISCUSSION:-

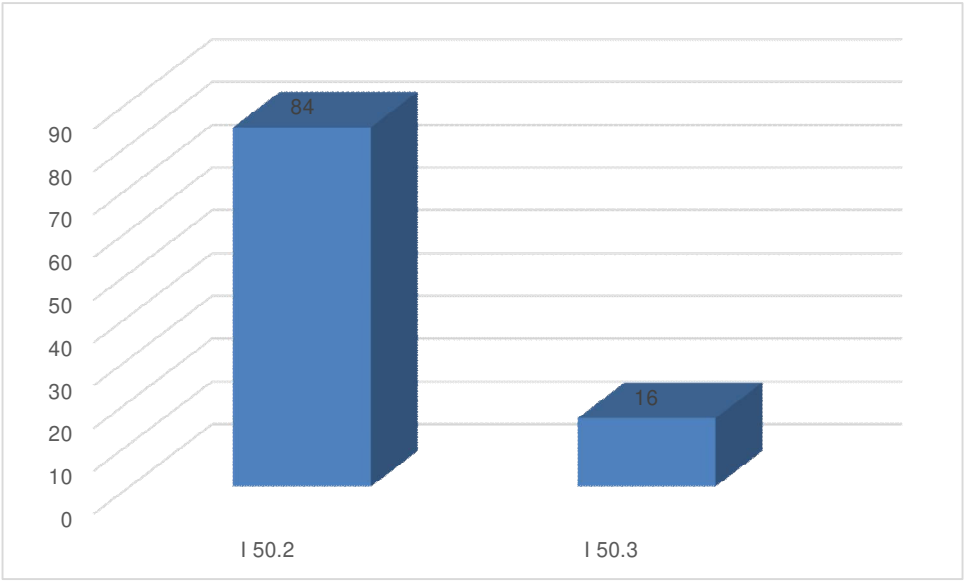
In our study,distribution of subjects according to type of disease condition HFpEF and HFrEF

And type of drug class for ACEI's normal value is (n=35) according to disease condition HFpEF is 7 (20) and HFrEF is 28 (80) , and for Beta- Blockers normal value is (n=63) according to disease condition HFpEF is 12 (19) and HFrEF is 51 (81),and for MRA's normal value is (n=38) according to disease condition HFpEF is 3 (7.9) and HFrEF is 35 (92.1) and the last class of drugs is SGLT-I's normal value is (n=16) according to disease condition HFpEF is 2 (12.5) and HFrEF is 14 (87.5) respectively.

12) Frequency and percentage distribution of ICD coding based upon type of EF

ICD coding	Frequency	Percentage
I 50.2	84	84
I 50.3	16	16
Total	100	100

Figure no: 14 - Bar diagram represents frequency and percentage distribution of ICD coding based upon type of EF



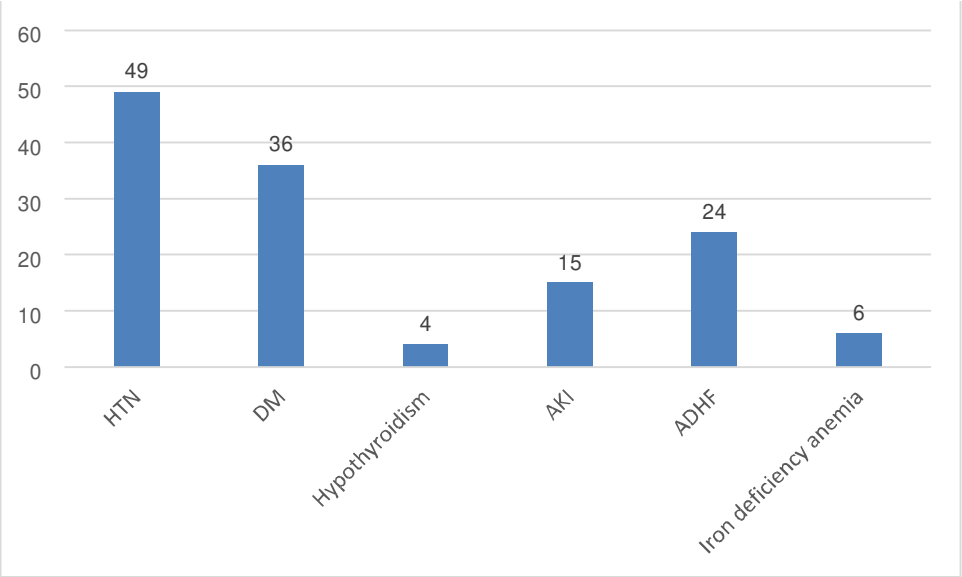
DISCUSSION: -

In our study , 100 subjects are taken into consideration and the ICD codings are included according to the disease condition that are HFpEF (I 50.2) and HFrEF (I 50.3) and their are 84% (84 of 100) HFpEF (I 50.2) and 16% (16 of 100) HFrEF (I 50.3) respectively.

13) Frequency and percentage distribution of subjects with heart failure based upon diagnosis.

Diagnosis	Frequency	Percentage
HTN	49	49
DM	36	36
Hypothyroidism	4	4
AKI	15	15
ADHF	24	24
Iron deficiency anemia	6	6

Figure no: 15 - Bar diagram represents frequency and percentage distribution of subjects with heart failure based upon diagnosis.



DISCUSSION:-

In our study , 100 subjects are taken into consideration and the distribution of subjects according to diagnosis for HTN 49% (49 of 100) and for the DM 36% (36 of 100) for HYPOTHYROIDISM 4% (4 of 100) for AKI 15% (15 of 100) and for the ADHF it is 24% (24 of 100) and for the iron deficiency anemia it us 6% (6 of 100) respectively.

14) Distribution of frequency of subjects with heart failure based upon reasons for not prescribing with best 4 classes of drugs.

Not prescribed reasons	Frequency
Due to good ejection fraction patient is treated with Anticoagulants, Antiplatelets classes.	1
Due to presence of high cholesterol levels in the patient was prescribed with statins and antiplatelets class of drugs.	1

Due to bacterial infection in the heart wall patient was treated with antibiotics, anti-platelets, statins etc.	1
Due to CAD blockage statins and anti platelets, anti-coagulants, diuretics were prescribed.	1
Due to good EF conditions patient was treated with other class of drugs like statins, antiplatelets.	1
Due to good ejection fraction patient was treated with anti-hypertensive class of drugs.	1
due to high blood pressure the patient was treated with calcium channel blockers and nitrates, multivitamins.	1
Due to high hypertension, the patient was treated with anti-hypertensive drugs, statins, NSAIDs, anti-coagulants.	1
Due to high lipo-profile of patient pt was treated with statins and antiplatelets, anti-coagulant drug classes .	2
Due to hypotension patient was treated with beta-histamine, iron supplements fluids.	1
Due to iron deficiency in the blood the pt was treated with ferric supplements and diuretics to increase the blood volume.	1
Due to ischemic conditions in the patient was treated with statins and nitrates, anti-platelets.	1
Due to mild chest pain patient was treated with NSAIDS, nitrates and other supportive medications.	1
Due to plaque formation in the coronary artery the patient was prescribed with statins, nitrates, antibiotics for infection.	1
Due to presence of high potassium in the patient was prescribed with loop diuretics and antiplatelets class of drugs.	1

Figure no: 16 - Bar diagram represents distribution of frequency of subjects with heart failure based upon reasons for not prescribing with best 4 classes of drugs.

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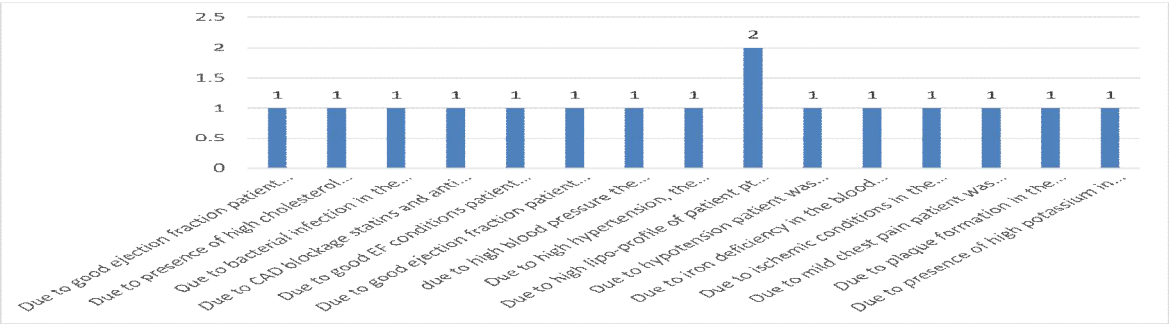

DISCUSSION:

In our study, 16 subjects were not prescribed by these four pillars of drug classes according to their disease condition other class of drugs were prescribed rather than these four classes of drugs like statins, anti-platelets, anti-coagulants etc., because these subjects have less comorbidities than the subjects in the heart failure. The above conditions are mentioned according to their disease condition. Above Fig 16. Bar diagram includes the reasons for their conditions.

VII. DISCUSSION

Our research states that Heart Failure and patient compliance plays a major critical role to bring good therapeutic outcome in the patients, which avoids the complication linked to extended hospitals stays and increased financial burden. The severe congestive heart failure signs and symptoms are minimized up to survival fittest of the patient by using medication. The prevalence shows that the patients with HFpEF compared with HFrEF are more likely older have a higher prevalence of hypertension, obesity and anemia suffer more often from comorbidities such as chronic kidney disease, chronic pulmonary disease, valvular heart disease and cancer.

HFmrEF has been recognized as a potentially distinct entity sharing features with both HFpEF such as hypertension, milder HF symptoms, lower levels of natriuretic peptides. The evidence suggests that in terms of pathophysiology, clinical characteristics, and response to therapy, HFmrEF resembles on average, more HFrEF than HFpEF

Among the 100 individuals who underwent heart failure treatment, a total of 100 subjects went through heart failure treatment, while 81% of subjects had HFrEF and 19% subjects had HFpEF . The age range spanned from above 18 adults greater than 65 years, with a predominant representation of males 65% (n =65).

Heart failure has occurred in all the 100 subjects, out of 100, 81 subjects were prone to heart failure with reduced ejection fraction (81%) and 19 were prone to heart failure with preserved ejection fraction (19%). In our study, male gender, and older age group above 65 years formed most of the cases who developed heart failure, however we didn't find any significant relationship between the occurrence of heart failure and age, and sex. According to the literature, there is no evidence for the effect of age on the development of heart Condition. However, certain studies found that heart failure occurs due to hypertension and diabetes mellitus Heart failure is more frequent in older patients, whereas other studies have reported it to be more common in the younger now a days. Heart failure was more common in patients who underwent hypertension and diabetes mellitus and also due to being obese and personal habits like alcohol consumption and smoking there is an increased risk of heart failure to subjects who are exposed to medications or toxic substances like alcohol, cocaine etc. , as well as subjects who are undergoing cancer treatment such as radiation and chemotherapeutic agents are responsible for the heart failure. (81% followed by HFrEF which was seen in 81 of cases. In our study all patients underwent treatment with four pillar of drugs like(Beta-
Blockers,MRA's ,ACEI's and SGLT-I's), while heart failure had so many other drug classes but mostly prescribed drugs classes are this four pillar of classes.

Our study shows the reasons for continuation of 4 pillars of drug classes and discontinuation reasons as well. The authors found that the risk of heart failure was higher in cases of toxic substances consumption and due to non-compliance of subjects is also an important reason to be considered. Furthermore, In this study, in those with heart failure, patients with HFrEF formed the majority of cases who developed more Comorbidities and most of them are treated with four pillars of drug classes and this drugs are prescribed to minimize the severity of heart failure and the subjects with HFpEF are less experienced with the severity of heart failure when compared to HFrEF subjects.

In our study we have found that there is significant resolution and improvement in serum creatinine levels, E-GFR and biomarkers like CK-MB and TROP-T by taking the Beta-Blocker, MRA's,
ACEI's and SGLT inhibitors.

Our study has several limitations. First, it was based upon physician consultation and afterwards including this there should be results according to the subjects continuation and discontinuation of medication and their reasons, and also the subjects with heart failure condition who are not at all prescribed this 4 pillars of drug classes, study with a large sample size. Second, it was prospective in nature.

VIII. CONCLUSION

By summarizing the statistical data of our study we have observed that the total number of patients who were prescribed on four pillars of drug classes (ACI's , MRA's , Beta- Blockers , SGLT-I's) out of this drug classes ,this class of drug class , i.e. Beta - blockers class of drugs have major differences in effectiveness in preventing heart failure. The tolerability of all the drugs is different, Beta- Blockers are preferred more than other class of drugs among the study population. The next preferred drug class is MRA's and then ACEI's in this ARNI also included and at last SGLT-I's are preferred according to the disease condition, The subjects of having a heart failure is more in patients with HFrEF than that of patients with HFpEF.

Based on reviewing literatures and reference articles, the studies that have been carried out on Heart failure have shown that HFrEF are majorly effective as Beta-Blockers, MRA's , ACEI's and SGLT-
I's has an advantage of reduced complications like SOB in improving blood pressure and other heart related effects and has a fixed dosage regimen due to these advantages this four pillars of drug classes can be used in place of other drug classes.

And out of 100 subjects 4% were discontinued the drugs due to burning micturition since 10 days CUE was done and pus cells were observed that is 20 cells for HF by taking ACEI's and SGLT-I's ,in 1% of subjects and due to cause of non tolerance [PYELONEPHRITIS] due to unavailability of particular drugs this is seen in 1% of subjects and due to wrong drug administration that is SGLT-I's due to this 1% of subjects have comorbidities , so this are the reasons to that 4% subjects out of 100% have discontinued the medication, As there are limited number of samples the results may not be yet generalized.

Further studies can be carried out on large scale population for more accurate evaluation.

IX. ACKNOWLEDGEMENT

It is by the blessings of the God almighty that we are able to complete our investigational studies successfully and present this work for which we are eternally indebted. We take a privilege to acknowledge all those who helped us in the completion of this work.

We accolade our highest respect to our parents for their moral support and dedicated efforts to educate to this level .

It is our privilege to express our heartfelt gratitude to our institutional guide, DR. REDDY NAZEMOON M.PHARM, Ph.D., professor, Bharat Institute Of Technology (Pharmacy), for her valuable guidance, co-operation, affectionate, encouragement and moral support throughout our project.

We take the privilege to express our heartfelt gratitude to our hospital guide, DR. DAYASAGAR RAO.V, DM [CARDIOLOGY], FRCP[CANADA], FRCP[EDENBURGH], Sr. Consultant Interventional Cardiologist, Department of Cardiology, Krishna Institute of Medical Sciences for his valuable guidance, cooperation, affectionate, encouragement and moral support throughout our project.

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ANNEXURE - II PATIENT PROFORMA

PATIENT DETAILS:

PATIENT NAME:	UMR NO:
AGE:	GENDER:
HT:	WT:
BMI:	BSA:
MOBILE NO:	DATE:

VITAL SIGNS:

BP: HR: RR: PR:

CHIEF COMPLAINTS:

PRESENT HISTORY:

PAST HISTORY:

☐ HTN
☐ DM
☐ THYROID
☐CVD
☐ CARDIAC SURGERY
☐ LUNG DISEASE
☐ KIDNEY DISEASE

ANY OTHER, SPECIFY:

DIAGNOSIS:

COURSE IN THE HOSPITAL:

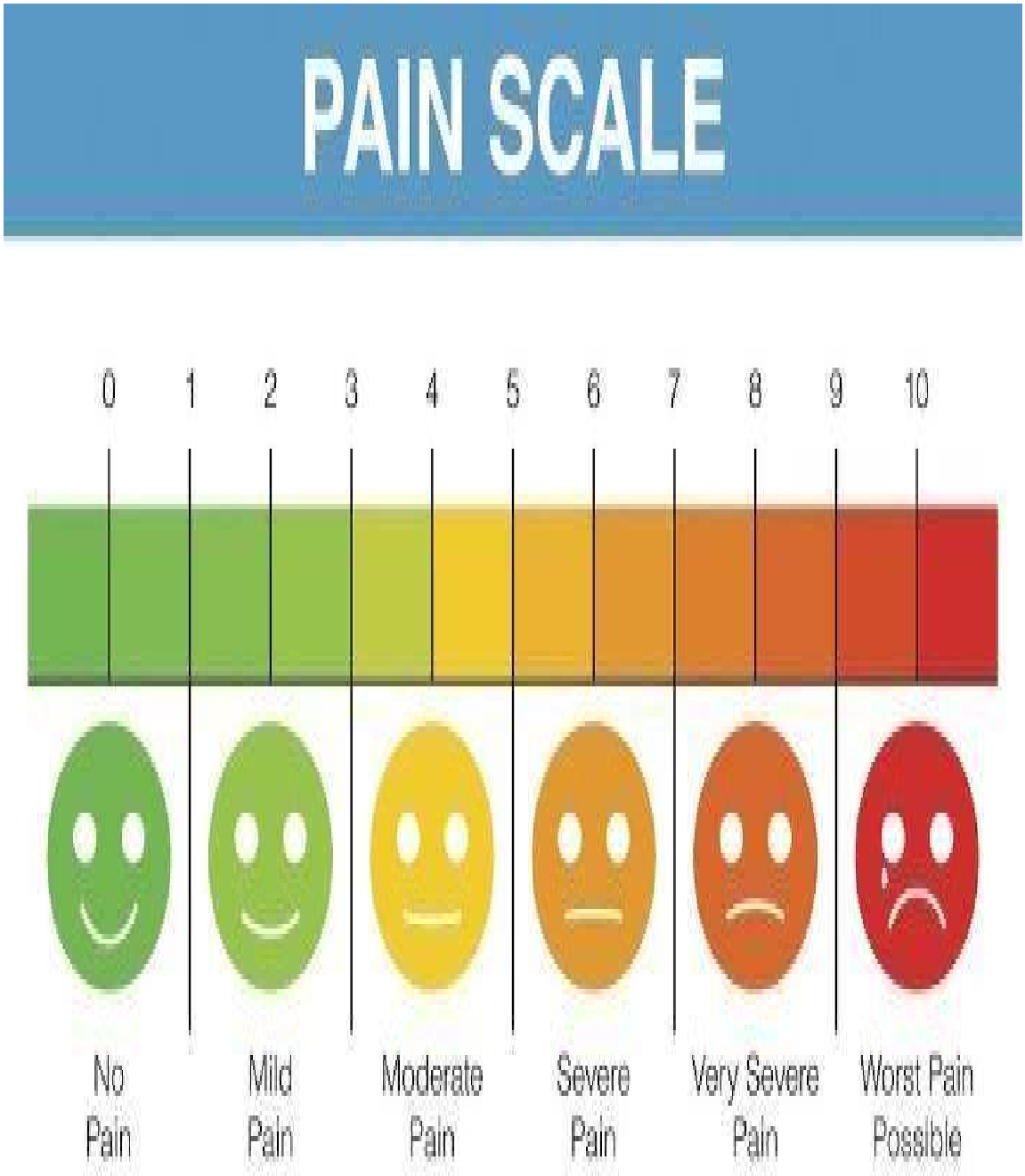
CONDITION AT THE END OF DISCHARGE:

PARAMETERS	DAY-1	DAY-2	DAY-3	DAY-4
Hb				
RBC				
WBC				
PLATELETS				
PT				
UREA				
CREATININE				
SERUM CREATININE				
SODIUM				
POTASSIUM				
CHLORIDES				
BILIRUBIN				
ESTIMATED GFR				
CRP				
SGOT				
SGPT				
CPK-MB				
TROP-T				

LABORATORY INVESTIGATIONS: DIAGNOSTIC TESTS:
ECG_____

2D-ECHO_____
CHEST X-RAY _____

PAIN SCALE (1 TO 10):



MEDICATION CHART

						Duration In Days						
S.No	DRUG	DOSE	ROA	FREQ	TIME	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7

DRUG TABLE			
CLASSES OF DRUGS			
ACE	MRA	B-BLOCKER	SGLT
DOSAGE ADEQUACY			
ACE :	PRESCRIBED VALUE	NRML VALUE	
MRA			
B - BLOCKER			
SGLT			
MISSING DRUG CLASS & REASONS			
ACE			
MRA			
B - BLOCKER			
SGLT			
NON-COMPLIANCE REASONS			
NOT PRESCRIBED			
NO TOLERANCE			
COST-CONSTRAINTS			

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ANNEXURE - III
INFORMED CONSENT FORM

Subject identification number for the study:
Title of the project: "PRESCRIBING DRUGS OF HEART FAILURE AND PATIENT COMPLIANCE"

Name of the principal investigator:
P. SAMPATH REDDY
P. SOWMYA SHETTY
P.N.S.K. SHARMA

I have received the information sheet on the above study and have read and/ or understood the written information. I have been given the chance to discuss the study and ask questions. I consent to take part in the study and I am aware that my participation is voluntary. I understand that I may withdraw at any time without affecting my future care. I understand that the information collected about me from my participation in this research and sections of any of my medical notes may be looked at by responsible persons (ethics committee members/ regulatory authorities). I give access to these individuals to have access to my records. I understand I will receive a copy of the patient information sheet and the informed consent form.
Name of the participant: Signature of the participant:
Contact no:
Date:
Place:

సమాచార సమ్మ తి పత్రం

అధ్యయనం కోసం విషయ గురతం ్ పు సంఖ్య:
ప్రా జెక్ట్ యొక్క శీరిక్ ్: "గుండె వఫ ్ై ల్యం మర్యు రోగ్కి మందుల్ ంు సూచంచడం కరరియ్ ్ాల్ీ విభాగంల్ ్ో దరఖ్ ్ాసతు"
పాధాన పర్యోధ్క్ ంుడి పేరు:
పి.సంపత్ రడె ిపి. సౌమయ శట్ ్ె ్ట పి.ఎన్.ఎస్.కె. శరమ

నేను ప ్ై అధ్యయన సమాచార పత్ ్ా న్ని అందుక్ ంునాిను మరయ్ ంు వరా సిన సమాచారరన్ని చదివరను/ల్ ేదా అరథం చేసుక్ ంునాిను. అధ్యయనం గుర్ంచ చర్చంచడాన్నకి మరయ్ ంు పశా ిల్ ంు అడిగే అవకరశం నాక్ ంు ఇవవబడింది. ననే ంు అధ్యయనంల్ ్ో ప్రల్ ్ొగనడాన్నకి అంగీక్రస్ తునాిను మర్యు నా భాగసరవమయం సవచఛందమన ్ై దన్న నాక్ ంు త్ ్ె ంుసు. నా భవిషయత్తత సంరక్షణను పభా ావిత్ం చేయక్ ంుండా ననే ంు ఎపుుడనై ా ఉపసంహర్ంచుకోవచచన్న నేను అరథం చేసుక్ ంునాిను. ఈ పర్యోధ్నల్ ్ో నా భాగసరవమాయన్ని మర్యు నా మడ ిక్ల్ నోట్స లో్ న్న ఏదనై ా విభాగరల్ నుండి నా గుర్ంచ సేక్రంచన సమాచారరన్ని బాధ్యత్తల్ వయక్తుల్ ంు (నత ్ై క్ క్మిట్ీ సభుయల్ ంు/ న్నయంత్తా అధకి రరుల్ ంు) వీక్ించవచచన్న ననే ంు అరథం చసే ంుక్ ంునాిను. నా ర్కరర్ి ల్ను యాకెసస్ చేయడాన్నకి నేను ఈ వయక్తుల్ క్ ంు యాకెసస్ న్న ఇసతునాిను. ననే ంు రోగ్ సమాచార షట్ీ మరయ్ ంు సమాచార సమమతి పత్ ా ం యొక్క కరపీన్న సీవక్రసతరనన్న అరథం చేసుక్ ంునాిను.

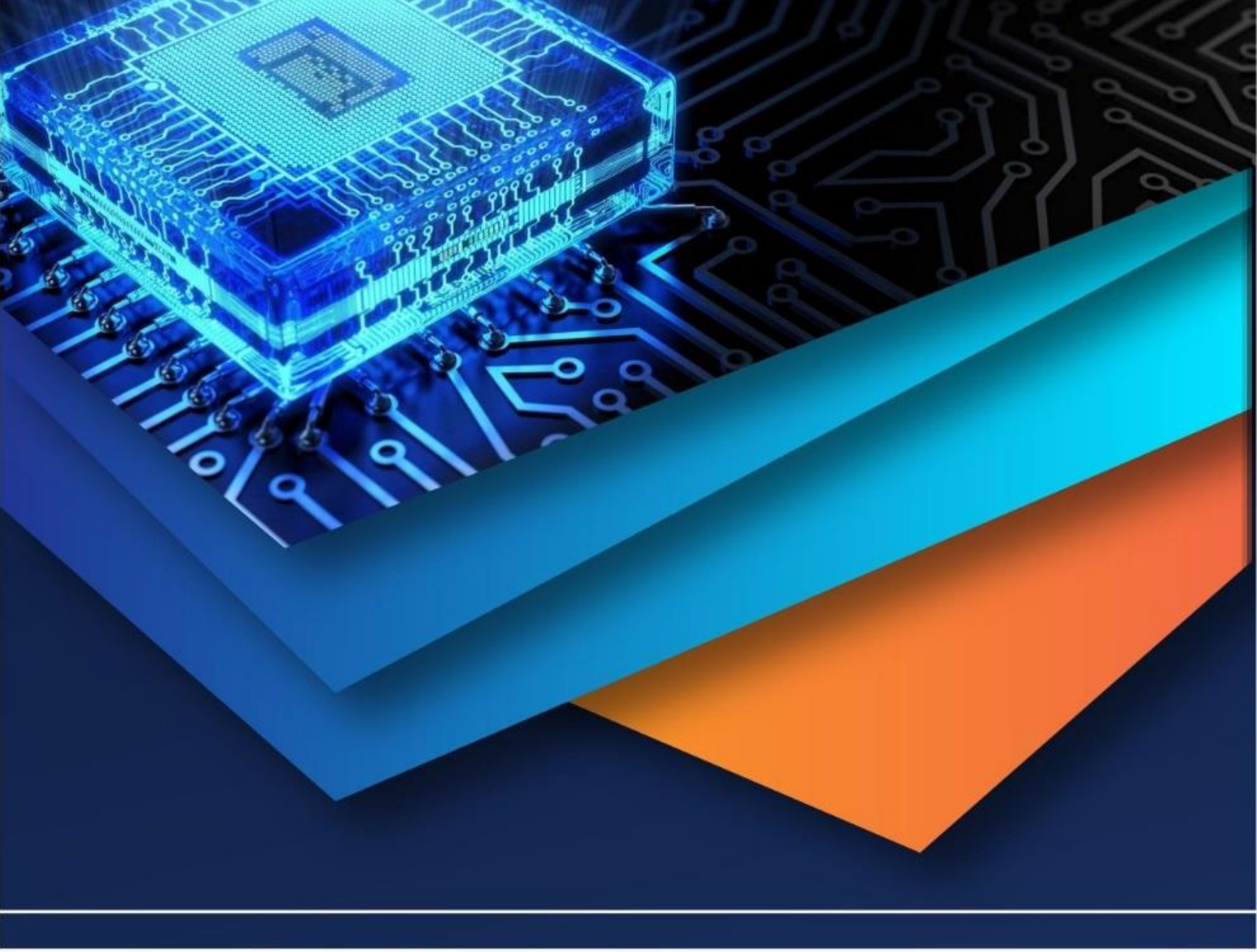
ప్రల్ ్ొగనవే రర్ పేరు: ప్రల్ ్ొగనవే రర్ సంత్క్ం:
సంపదా ిం్ప సంఖ్య: త్ ేదీ: సథల్ం: **सूचित सहमचत प्रपत्र**

अध्ययन के लिए लिषय पहचान संख्या:

परियोजना का शीषषक: "कालडषयोि ॉजी लिभाग में हृदय लिफिता की दिाएं लिखना औि िोगी अनुपाि न"

प्रमुख अन्वेषक का नाम:
पी. संपत ि◌ेड्डी पी.सौम्या शेट्टी
पी.एन.एस.के.शमाष
मुझे उपिोक्त अध्ययन पि सूचना पत्र प्राप्त हुआ है औि मैंने लिखखत जानकाि◌ी पढ़ ि◌ी है औि/या समझ ि◌ी है। मुझे अध्ययन पि चचाष किने औि प्रश्न पूछने का मौका लदया गया है। मैं अध्ययन में भाग ि◌ेने के लिए सहमलत देता हं औि मुझे पता है लक मेि◌ी भागीदाि◌ी स्वैखिक है। मैं समझता हं लक मैं अपनी भलिष्य की देखभाि◌ को प्रभालित लकए लिना लकसी भी समय ि◌ापस ि◌े सकता हं। मैं समझता हं लक इस शोध में मेि◌ी भागीदाि◌ी से मेि◌े ि◌ाि◌े में एकत्र की गई जानकाि◌ी औि मेि◌े लकसी भी मेलडकि नोट्स के अनुभागों को लजम्मेदाि◌ व्यखक्तयों (नैलतकता सलमलत के सदस्◌ो◌ं / लनयामक अलधकारियों) द्वाि◌ा देखा जा सकता है। मैं इन व्यखक्तयों को अपने रिकॉडष तक पहुंच प्रदान किता हं। मैं समझता हं लक मुझे ि◌ोगी सूचना पत्रक औि सूलचत सहमलत प्रपत्र की एक प्रलत प्राप्त होगी।

प्रलतभागी का नाम: प्रलतभागी के हस्ताक्षि: संपकष नं◌ि◌:
ताि◌ीख: जगह:



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