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A REVIEW ON ETIOLOGY, SYMPTOMS, STAGES AND TREATMENT OF CONGESTIVE HEART FAILURE

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ABSTRACT

Heart failure is a pathological medical disorder where there is an abnormality of heart function, which results in an inability to pump blood to the rest of the body resulting in poor perfusion of the organs. Congestive heart failure is a progressive disease generally seen in elderly, which if not properly managed leads to death. Heart failure is a serious condition with significant morbidity and mortality the continuous increases is local congestive heart failure cases and the economic burden associated with it has led to the over helming demand for bowel therapies quite a few drugs for heart failure have shown some promise in preclinical as well as early phase clinical trails but most of them were unsuccessful. In addition to the pharmacological and non-pharmacological intervention also plays an essential role in management of heart failure to assess the true effectiveness of these attractive compounds.

KEY WORDS:-

Congestive heart failure, fatigue, ACE Inhibitors, Beta-Blockers, cardiac resynchronization therapy, Biventricular pacemaker.

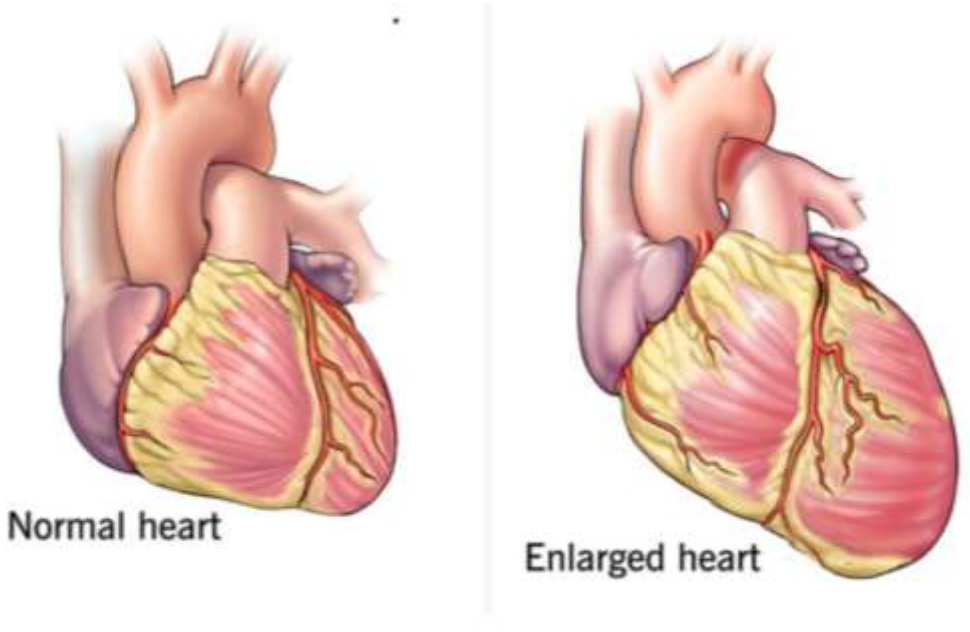
INTRODUCTION

Congestive heart failure, or heart failure, is a long-term condition in which your heart can't pump blood well enough to meet your body's needs. Your heart is still working. But because it can't handle the amount of blood it should, blood builds up in other parts of your body. Most of the time, it collects in your lungs, legs and feet. CHF results when damage to the heart means it no longer works as it should to pump blood around the body.

If the heart becomes weakened and cannot supply the cells with sufficient blood, it can lead to fatigue, breathlessness, and swelling due to fluid retention.

Usually there is no cure for CHF, but with the right treatment, people can often stay active and have a good quality of life.

Here is the difference between the normal heart and failure heart



ETIOLOGY:-

Several factors and underlying conditions can contribute to the development of CHF, including

1.Coronary Artery Disease (CAD): This is the most common cause of CHF. CAD occurs when the arteries supplying blood to the heart muscle become narrowed or blocked, leading to reduced blood flow and oxygen to the heart.

2.High Blood Pressure (Hypertension): Chronic high blood pressure forces the heart to work harder to pump blood, which can lead to the thickening of the heart muscle and eventually weaken it.

3.Heart Attack (Myocardial Infarction): A heart attack damages the heart muscle, reducing its ability to pump blood effectively.

4.Cardiomyopathy: This is a disease of the heart muscle itself, which can be caused by genetic factors, infections, alcohol abuse, and certain drugs. It leads to weakened heart muscles that can't pump blood efficiently.

5.Heart Valve Problems: Malfunctioning heart valves (due to congenital defects, infections, or other conditions) can lead to heart failure by forcing the heart to work harder to pump blood.

6.Arrhythmias: Abnormal heart rhythms can cause the heart to beat too fast, too slow, or irregularly, impairing its ability to pump blood effectively.

7.Diabetes: Diabetes increases the risk of high blood pressure and coronary artery disease, both of which can lead to heart failure.

8.Chronic Kidney Disease: Poor kidney function can lead to fluid overload and high blood pressure, both of which can contribute to heart failure.

9.Congenital Heart Defects: Structural heart defects present from birth can strain the heart and lead to heart failure over time.

10.Obesity: Excess weight increases the workload on the heart and can contribute to conditions like high blood pressure and diabetes, which are risk factors for CHF.

11.Sleep Apnoea: This condition, where breathing repeatedly stops and starts during sleep, can lead to high blood pressure and heart enlargement, both risk factors for heart failure.

12.Alcohol and Drug Abuse: Excessive alcohol consumption and the use of certain drugs can weaken the heart muscle and lead to heart failure.

SYMPTOMS :-

common symptoms include

- Shortness of Breath (Dyspnoea): This can occur during activity or while lying down. It is often due to fluid buildup in the lungs.
- Fatigue and Weakness: Reduced blood flow and oxygen to the body's tissues can cause persistent tiredness and muscle weakness.
- Swelling (Oedema): Fluid retention in the legs, ankles, feet, and sometimes the abdomen can cause swelling and weight gain.
- Rapid or Irregular Heartbeat (Palpitations): The heart may beat faster or irregularly as it tries to pump more blood.
- Persistent Cough or Wheezing: This is often accompanied by white or pink blood-tinged mucus due to fluid accumulation in the lungs.
- Increased Need to Urinate at Night (Nocturia): Lying down can make it easier for the kidneys to remove excess fluid from the bloodstream, leading to increased urination at night.
- Difficulty Concentrating or Decreased Alertness: Reduced blood flow to the brain can cause cognitive issues and confusion.
- Chest Pain: Although less common, chest pain can occur if heart failure is caused by a heart attack.
- Lack of Appetite and Nausea: Digestive issues and decreased blood flow to the digestive organs can lead to a reduced appetite and nausea.
- Sudden Weight Gain: Rapid weight gain from fluid retention is a sign that heart failure may be worsening.
- Swelling of the Abdomen (Ascites): Fluid accumulation in the abdominal cavity can cause bloating and discomfort.
- Nocturia
- Haemoptysis
- Abdominal pain
- Anorexia
- Pulmonary rales
- Pulmonary oedema
- S3 gallop
- Pleural effusion
- Cheyne-Stokes respiration
- Tachycardia

Sometimes, you may have mild symptoms of congestive heart failure or none at all. This doesn't mean you don't have heart failure anymore. Symptoms of heart failure can range from mild to severe and may come and go.

Unfortunately, congestive heart failure usually gets worse over time. As it worsens, you may have more or different signs or symptoms.

STAGES:-

Stage A: High Risk of Developing Heart Failure

Patients are at high risk for heart failure but do not have structural heart disease or symptoms of heart failure.

Stage A (pre-heart failure) means you're at a high risk of developing heart failure because you have a family history of congestive heart failure or you have one or more of these medical conditions:

- ✓ Hypertension.
- ✓ Diabetes.
- ✓ Coronary artery disease.
- ✓ Metabolic syndrome.
- ✓ History of alcohol use disorder.
- ✓ History of rheumatic fever.
- ✓ Family history of cardiomyopathy.
- ✓ History of taking drugs that can damage your heart muscle, such as some cancer drugs.

Stage B: Asymptomatic Heart Failure

Patients have structural heart disease but have never shown symptoms of heart failure. Stage B (pre-heart failure) means your left ventricle isn't working well and/or is structurally abnormal but you've never had symptoms of heart failure.

Stage C: Symptomatic Heart Failure

Patients have known structural heart disease and have current or previous symptoms of heart failure.

Examples: Individuals experiencing symptoms like shortness of breath, fatigue, or reduced exercise tolerance.

Stage D: Refractory End-Stage Heart Failure

Patients have advanced heart disease and marked symptoms of heart failure at rest despite maximal medical therapy.

Examples: Individuals with frequent hospitalizations for heart failure, severe symptoms at rest, or requiring specialized interventions.

TREATMENT:-

GOALS OF TREATMENT

The goals of therapy in the management of chronic heart failure were to improve the patient's quality of life, reduce symptoms, reduce hospitalizations, slow progression of the disease process,and prolong survival.

A patient's symptoms can change frequently Although these goals are still important, identification of risk factors for heart failure development and recognition of its progressive nature have led to increased emphasis on preventing the development of this disorder. over a short period of time owing to changes in medications, diet, intercurrent illnesses, etc.

NONPHARMACOLOGICAL THERAPY

- Recent studies demonstrate that cardiac resynchronization therapy (CRT) offers a promising approach to selected patients with chronic heart failure.
- Implantation of a specialized biventricular pacemaker to restore synchronous activation of the ventricles can improve ventricular contraction and hemodynamic
- Exercise training for stable Hf patients increased Exercise capacity decreased hospitalization rate, increased quality of life, decreased symptoms

- weight loss in obese patients
- dietary Na restriction
- Smoking-avoid smoking (adverse effects on coronary disease, adverse haemodynamic effects)
- Vaccination-patients should consider influenza and pneumococcal vaccinations
- Alcohol-advise moderate alcohol consumption (abstinence in alcohol related cardiomyopathy)
- Fluid and free water restriction especially if hyponatremia
- Minimize medications known to have deleterious effects on hear failure (negative inotropes, NSAIDs, OTC stimulants)
- Oxygen
- Fluid removal (dialysis, thoracentesis, paracentesis)

Pharmacological Treatment

TREATMENT OF STAGE A HEART FAILURE

Patients in stage A do not have structural heart disease or symptoms but are at high risk for developing heart failure because of the presence of risk factors. The emphasis here is on identification and modification of these risk factors to prevent the development of structural heart dis-ease and subsequent heart failure Effective control of blood pressure reduces the risk of developing heart failure by approximately 50%.35 Control of hyper glycemia reduces the risk of end-organ damage and may decrease the risk of heart failure. Appropriate management of coronary artery disease and its associated risk factors is also important, including treatment of hyper lipidemia according to published guidelines and smoking cessation. Although treatment must be individualized, ACE inhibitors should be strongly considered for antihypertensive therapy in patients with multiple vascular risk factors.

TREATMENT OF STAGE B HEART FAILURE

Patients in stage B have structural heart disease but do not have heart failure symptoms. This group includes patients with left ventricular hypertrophy or fibrosis, previous MI, valvular disease, or left ventricular systolic dysfunction. These individuals are at risk for developing heart failure, and treatment is targeted at minimizing additional injury and preventing or slowing the remodeling process. In addition To the treatment measures outlined in stage A, ACE inhibitors and β-blockers are important components of therapy. Patients with a previous MI should receive both ACE inhibitors and β-blockers regard-less of the ejection fraction (EF).

TREATMENT OF STAGE C HEART FAILURE

Most patients in stage C should be treated routinely with four medications: an ACE inhibitor, a diuretic, a β-blocker, and digoxin (see “Standard First-Line Therapies” below) The benefits of these medications on slowing heart failure progression, reducing morbidity and mortality, and improving symtoms are clearly established. Aldosterone receptor antagonists, ARBs,and hydralazine-isosorbide dinitrate are also useful in selected patients. Other general measures also are important, including moderate sodium restriction, daily weight measurement, immunization against influenza and pneumococcus, modest physical activity, and avoidance of medications that can exacerbate heart failure. Recent evidencesuggests that careful follow-up and patient education that reinforces dietary and medication compliance can prevent clinical deterioration

TREATMENT OF STAGE D HEART FAILURE

This includes patients who undergo recurrent hospitalizations or cannot be discharged from the hospital without special interventions. These individuals have the most advanced form of heart failure and should be considered for specialized therapies, including mechanical circulatory support, continuous positive inotropic therapy, cardiac transplantation, or hospital care.

ACE INHIBITORS

ACE inhibitors are the cornerstone of pharmacotherapy of patients with heart failure. By blocking the conversion of angiotensin I to angiotensin II by ACE, the production of angiotensin II and, in turn, aldosterone is decreased but not completely eliminated

ACE inhibitor therapy appears to play an important role in preventing angiotensin II–mediated progressive worsening of myocardial function. The endogenous vasodilator bradykinin, which is inactivated by ACE, is also increased by ACE inhibitors, along with the release of vasodilatory prostaglandins and histamine

The acute response to ACE inhibitor therapy is greater in patients with high levels of plasma renin activity. However, long-term hemodynamic and clinical responses to ACE inhibition cannot be predicted from the plasma renin activity or from response to the initial dose of ACE inhibitor.

Since many heart failure patients have concomitant disorders (e.g., diabetes, hypertension, or previous MI) that also may be affected favorably by ACE inhibitors, renal dysfunction should not be a contraindication to ACE inhibitor use in patients with left ventricular dysfunction. However, these patients should be monitored carefully for the development of acute renal failure and/or hyperkalemia, with special attention to risk factors associated with this complication of ACE inhibitor therapy

β-Blockers

Initiation of β-blocker therapy at normal doses in patients with heart failure has the potential to lead to symptomatic worsening or decompensation owing to the drug's negative inotropic effect.

Patients should receive a β-blocker even if their symptoms are well controlled with diuretic and ACE inhibitor therapy becausethey remain at risk for progression of disease.

An important aspect to the safe use of β-blockers in heart failure is initiation of therapy at a low dose, with slow upward dose titration. Current guidelines recommend initiation of therapy once patients have been relatively stable for several weeks. However, a recent study indicated that initiation of carvedilol therapy before discharge in patients hospitalized for decompensated heart failure increased the number of patients treated with β-blockers compared with usual care and did not increase the risk of serious adverse effects.68 Typically, starting doses have been 1/10 to 1/20 the final dose, with doses doubling no more frequently than every 2 weeks until the target dose is reached. the starting dose for bisoprolol is 1.25 mg/day. However, the smallest commercially available tablet of bisoprolol is a scored 5-mg tablet. Since the starting dosage of bisoprolol is not readily available, this drug is the least commonly used of the three agents, and it is not approved by the Food and Drug Administration (FDA) for use in heart failure.

Diuretics

The compensatory mechanisms in heart failure stimulate excessive sodium and water retention, often leading to signs and symptoms of systemic and pulmonary congestion. Consequently, diuretic therapy is recommended for all patients with clinical evidence of fluid retention. Among the drugs used for management of heart failure, the diuretics are most rapid in producing symptomatic benefits. The majority of patients with heart failure will require chronic diuretic therapy to control their fluid status, and as such, diuretics represent a cornerstone of heart failure therapy. However, because they do not alter disease progression (or prolong survival), they are not considered mandatory therapy. Thus patients who do not have fluid retention would not require diuretic therapy

The primary goal of diuretic therapy is to reduce symptoms associated with fluid retention and pulmonary congestion, improve quality of life, and reduce hospitalizations from heart failure. They accomplish this by decreasing edema and pulmonary congestion through reduction of preload.A reduction in preload improves symptoms but has little effect on the patient's stroke volume or cardiac output until the steep portion of the curve is reached. However, diuretic therapy must be used judiciously because overdiuresis can lead to a reduction in cardiac output and symptoms of dehydration. Once diuretic therapy is initiated, dosage adjustments are based on symptomatic improvement and daily body weight. Change in body weight is a

sensitive marker of fluid retention or loss, and it is recommended that patients monitor their status by taking daily morning body weights.

Thiazide diuretics such as hydrochlorothiazide block sodium and chloride reabsorption in the distal convoluted tubule (approximately 5% to 8% of filtered sodium). The thiazides therefore are relatively weak diuretics and infrequently are used alone in heart failure. However, as is reviewed in detail in the section "Treatment: Advanced/Decompensated Heart Failure" under "Diuretic Resistance," thiazides or the thiazide-like diuretic metolazone can be used in combination with loop diuretics to promote a very effective diuresis.

Loop diuretics are the most widely used diuretics in heart failure. They act in the thick ascending limb of the loop of Henle, where 20% to 25% of filtered sodium normally is reabsorbed. Because loop diuretics are highly bound to plasma proteins, they are not highly filtered at the glomerulus. They reach the tubular lumen by active transport via the organic acid transport pathway. Competitors for this pathway (probenecid or organic by-products of uremia) can inhibit delivery of loop diuretics to their site of action and decrease effectiveness. Loop diuretics also induce a prostaglandin-mediated increase in renal blood flow, which contributes to their natriuretic effect. Coadministration of NSAIDs and COX-2 inhibitors blocks this prostaglandin-mediated effect and can diminish diuretic efficacy. Unlike thiazides, loop diuretics maintain their effectiveness in the presence of impaired renal function, although higher doses are necessary to obtain adequate delivery of the drug to the site of action.

DIGOXIN

The efficacy of digoxin in patients with heart failure and supraventricular tachyarrhythmias such as atrial fibrillation is well established and widely accepted.

In patients with heart failure and supraventricular tachyarrhythmias such as atrial fibrillation, it should be considered early in therapy to help control ventricular response rate. For patients in normal sinus rhythm, although digoxin does not improve survival, its effects on symptom reduction and quality-of-life improvement are evident in patients with mild to severe heart failure. Therefore, it should be used together with other standard heart failure therapies, including diuretics, ACE inhibitors, and β-blockers, in patients with symptomatic heart failure. Clinicians may want to consider adding digoxin after instituting β-blocker therapy so that the potential bradycardic effect of digoxin does not preclude the use of a β-blocker.

CONCLUSION:-

Congestive heart failure is the lifelong diagnosis managed with lifestyle changes and medications to prevent acute congestive episodes. Commonly underlined conditions include coronary atherosclerosis, valvular disease, cardiomyopathy inflammatory or degenerative muscle disease and arterial hypertension the optimum utilisation of the available drugs, general measures and surgical procedures and appropriate to the condition improves the outcomes of these patients.In this article we have highlighted about the congestive heart failure and its causes and their symptoms and preventive and treatment measures to control the CHF .

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