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A Review on Congestive Heart Failure

Mr. Kale A. S[1], Ms. Amle V. S[2], Ms. Kathoke G. G[3], Mr. Bedre A. B[4]
Shree Bhairavnath Nisarg Mandal College of Pharmacy, Hatta, India

Abstract: *Congestive Heart Failure (CHF) is a clinical syndrome that occurs when the heart is unable to pump blood effectively to meet the body's metabolic demands, leading to the accumulation of fluid in the lungs, liver, and peripheral tissues. It is commonly the result of underlying cardiovascular conditions such as coronary artery disease, hypertension, valvular heart diseases, and cardiomyopathies. The pathophysiology of CHF involves impaired myocardial contractility, increased preload, and afterload, which disrupt normal hemodynamic and tissue perfusion. Symptoms include dyspnea, fatigue, orthopnoea, edema, and ascites. Diagnosis relies on clinical presentation, imaging studies (echocardiography), and laboratory tests, including natriuretic peptide levels. Management involves pharmacological therapy, such as ACE inhibitors, beta-blockers, diuretics, and aldosterone antagonists, as well as lifestyle modifications. In severe cases, mechanical circulatory support or heart transplantation may be considered. Despite advances in treatment, CHF remains a major cause of morbidity, mortality, and hospitalization, particularly among older populations. Ongoing research is focused on improving early diagnosis, understanding the molecular mechanisms underlying CHF, and developing novel therapeutic approaches..*

Keywords: Congestive heart failure, Types, Etiology, Treatment, Classification.

I. INTRODUCTION

Congestive heart failure (CHF) is a complex clinical syndrome that can result from any functional or structural cardiac disorder that impairs the ventricle's ability to fill with or eject blood. Since there is no definitive diagnostic test for heart failure, it remains a clinical diagnosis that is largely based on a careful history and physical examination and supported by ancillary tests such as chest radiograph, electrocardiogram, and echocardiography. Heart failure is a common disease, affecting approximately 5 million people in the United States, and it occurs predominately in the elderly, with almost 80% of cases occurring in patients over the age of 65.1.

The magnitude of the problem cannot be precisely assessed, because reliable population-based data on the prevalence, incidence, and prognosis are lacking. Nevertheless, several studies have found that CHF is associated with a 2-year mortality rate of approximately 45–50%, which approaches that of many malignancies.2 Moreover, from a societal perspective, caring for patients with CHF accounts for 2–3% of the federal health-care budget. The estimated direct and indirect cost of CHF in the United States in 2005 was $27.9 billion.

There are 2 mechanisms of reduced cardiac output and heart failure: systolic dysfunction and diastolic dysfunction. The most common causes of systolic dysfunction (defined by a left-ventricular ejection fraction of 50%) are ischemic heart disease, idiopathic dilated cardiomyopathy, hypertension, and valvular heart disease. Diastolic dysfunction (defined as dysfunction of left-ventricular filling with preserved systolic function) may occur in up to 40–50% of patients with heart failure, it is more prevalent in women, and it increases in frequency with each decade of life.

Diastolic dysfunction can occur in many of the same conditions that lead to systolic dysfunction. The most common causes are hypertension, ischemic heart disease, hypertrophic cardiomyopathy, and restrictive cardiomyopathy. Many patients who have symptoms suggestive of heart failure (shortness of breath, peripheral edema, paroxysmal nocturnal dyspnea) but also have preserved left ventricular function may not have diastolic dysfunction; instead, their symptoms are caused by other etiologies, such as lung disease, obesity, or occult coronary ischemia.3 This article will review the pathophysiology, diagnosis, and treatment of CHF, with specific discussion of the pulmonary manifestations and their treatment, including noninvasive positive-pressure ventilation (NPPV) strategies.

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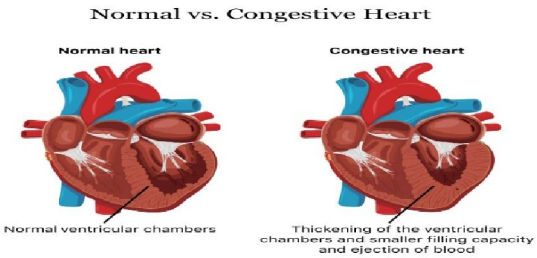

II. TYPES OF CONGESTIVE HEART FAILURE

Types of congestive heart failure include:

- Left-sided heart failure.
- Right-sided heart failure.
- High-output heart failure.

Left Sided Heart Failure

Left-sided heart failure occurs when the heart loses its ability to pump blood. This prevents organs from receiving enough oxygen. The condition can lead to complications that include right-sided heart failure and organ damage.

This condition occurs when the left side of the heart no longer functions appropriately. There are two types:

Systolic heart failure: The bottom pumping chamber of your heart called the left ventricle is too weak to pump blood out to your body. It's also known as heart failure with reduced ejection fraction.

Diastolic heart failure: The left ventricle is stiff and can't relax appropriately, making it difficult to fill with blood. This condition is also known as heart failure with preserved ejection fraction.

Right Sided Heart Failure

Right-sided heart failure is one type of heart failure. Right-sided heart failure is also called right ventricular (RV) heart failure or right heart failure.

The right side of your heart pumps "used" blood from your body back to your lungs, where it refills with oxygen. Right-sided heart failure means your heart's right ventricle is too weak to pump enough blood to the lungs. As a result:

Blood builds up in your veins, vessels that carry blood from the body back to the heart.

This buildup increases pressure in your veins.

The pressure pushes fluid out of your veins and into other tissue.

Fluid builds up in your legs, abdomen or other areas of your body, causing swelling.

High-Output Heart Failure

High-output heart failure is a condition in which your heart is initially working normally (either with reduced or preserved ejection fraction) but can't keep up with your body's increasing need for more blood. Your heart ultimately becomes weak and can no longer pump blood effectively throughout your body.

High cardiac output sets high-output heart failure apart from the other types of heart failure. With most types of heart failure, cardiac output is normal or lower than normal. People with high-output heart failure have a cardiac output of 8 liters (about 2 gallons) of blood per minute. A normal cardiac output is 5 to 6 liters (1.3 to 1.6 gallons) of blood per minute.

III. ETIOLOGY

There are many etiologies of CHF, and coronary artery disease (CAD) causing ischemic heart disease is the most common cause. Every attempt should be made to identify causative factors to help guide treatment strategies. The etiologies can be broadly classified as intrinsic heart disease and pathologies that are infiltrative, congenital, valvular, myocarditis-related, high-output failure, and secondary to systemic disease. These classifications have significant

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overlap. The 4 most common etiologies responsible for about two-thirds of CHF cases are ischemic heart disease, chronic obstructive pulmonary disease (COPD), hypertensive heart disease, and rheumatic heart disease. Higher-income countries have higher rates of ischemic heart disease and COPD; lower-income countries have higher rates of hypertensive heart disease, cardiomyopathy, rheumatic heart disease, and myocarditis.

Ischemic heart disease is by far the most common cause of CHF worldwide. Ischemia leads to a lack of blood flow to heart muscles, reducing the EF. Incidence is increasing in developing countries as they adopt a more Western diet and lifestyle, and improved medical care decreases the infectious burden in these countries (myocarditis is often infection-related.)

Valvular heart disease is another common intrinsic heart condition that can cause CHF. Rheumatic heart disease is the most common cause of valvular heart disease in children and young adults worldwide. It is caused by an immune response to group A Streptococcus and primarily causes mitral and aortic stenosis. The most common overall cause of valvular disease is age-related degeneration, and the aortic valve is the most commonly affected valve. Women are more likely to experience mitral valve rheumatic heart disease or mitral valve prolapse, while men are more likely to suffer from aortic valve diseases such as regurgitation or stenosis. Endocarditis is also more common in men.

Hypertension causes CHF even in the absence of CAD or ischemic heart disease. High blood pressure causes mechanical stress by increased afterload and neurohormonal changes that increase ventricular mass. HTN is also strongly associated with other comorbidities for CHF development, and aggressively treating hypertension is shown to lower the incidence of CHF.

Cardiomyopathy is a heterogeneous group of diseases characterized by enlarged ventricles with impaired function not related to secondary causes such as ischemic heart disease, valvular heart disease, hypertension, or congenital heart disease. The most common types of cardiomyopathies are hypertrophic, dilated, restrictive, arrhythmogenic right ventricular, and left ventricular noncompaction. In addition to CHF, cardiomyopathy can present as arrhythmia or sudden cardiac death, further compelling the identification of underlying disorders. Many of these conditions have a genetic basis, and a detailed family history of sudden cardiac death, especially in first-degree relatives older than 35 years, should be taken. There are over 50 identified genes contributing to the development of dilated cardiomyopathy alone. Genetic determinants have variable phenotypic expression, and many nongenetic factors also affect the clinical symptoms. Some of these factors include diabetes, toxic exposure, or pregnancy. Fabry disease is a rare glycogen storage disease that can cause CHF symptoms through a hypertrophic cardiomyopathy pattern.

Inflammatory cardiomyopathy is defined by myocarditis along with ventricular remodeling and cardiac dysfunction. The most common cause is viral infection. Other etiologies are bacterial, fungal, or protozoal infections; toxic substances or drugs; and immune-mediated diseases. Chagas disease is caused by *Trypanosoma cruzi,* which is endemic in Latin America and commonly causes myocarditis, cardiomyopathy, and CHF. Other viral causes of myocarditis and inflammatory cardiomyopathy include adenoviruses, enteroviruses, herpes virus 6, Epstein-Barr virus, and cytomegalovirus. Viruses can also activate autoimmune myocarditis, including HIV, hepatitis C virus, influenzas A and B, and coronaviruses (including COVID-19). When associated with CHF, these conditions tend to have a poor prognosis.

Infiltrative cardiomyopathies cause a restrictive cardiomyopathy pattern (simar to the genetically determined restrictive cardiomyopathy variant), which is notable for normal ventricular systolic function, but with diastolic dysfunction and restrictive filling dynamics of the LV and RV. This is often associated with a high E/A ratio showing increased early filling and delayed late filling.

Cardiac amyloidosis results from misfolded protein deposits in the heart; this leads to cardiomyocyte separation, cellular toxicity, and tissue stiffness. Patients are preload dependent and are prone to symptomatic hypotension. Currently, tamifidis is the only medication known to prevent cardiac amyloidosis. It prevents, but does not reverse, amyloid deposition. Its high cost is also a limiting factor.

Sarcoidosis is an acquired cardiomyopathy that presents with conduction defects and arrhythmias due to granuloma formation. The most common cardiac manifestation is CHF. Caution must be used when treating with beta-blockers due to the associated conduction abnormalities.

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Cardiac hemochromatosis is present in 15% to 20% of patients with hereditary hemochromatosis. This condition initially presents with a restrictive pattern but develops into biventricular systolic dysfunction. Patients with restrictive cardiomyopathy physiology can develop hypotension when treated with traditional CHF medications due to preload dependence, so caution should be used to avoid systemic hypoperfusion.

Takotsubo or stress-induced cardiomyopathy (colloquially broken-heart syndrome) is an underrecognized cause of CHF, which causes transient left-ventricular wall abnormalities that are not localized to a specific vascular territory. It has several proposed pathophysiologic mechanisms, including coronary vasospasm, microcirculatory dysfunction, and increased activation of the sympathetic nervous system. This condition is treated with medications typical for CHF with the addition of antithrombotic medications in certain clinical situations with wall motion abnormalities. Recognized cases increased significantly during the COVID-19 epidemic.

Peripartum cardiomyopathy is a significant cause of maternal mortality. During pregnancy, cardiac output is increased by 20% to 30% due to increased heart rate and stroke volume. It presents with CHF due to LV systolic dysfunction during late pregnancy, postpartum, or up to several months after delivery. There is likely an underlying genetic component, and it is more common in women with advanced maternal age, Black race, and multifetal pregnancies. If wall motion abnormalities are present, anticoagulation is essential due to the hypercoagulable state caused by pregnancy. Recovery is variable by global region and inversely correlates with lowered EF.

Obesity is a leading cause of CHF in patients younger than 40 years, according to the "Candesartan in Heart Failure Assessment of Reduction in Mortality and Morbidity" (the CHARM study). The "obesity paradox" described elsewhere has significant study flaws and is derived from older data. It is thought that up to 10% of CHF cases are attributable to obesity alone. Patients with obesity are more likely to have HFpEF, possibly secondary to adipose-produced cytokines such as IL-1b, IL-8, and TNFα. Adipose tissue also degrades natriuretic peptides.

Tachycardia and arrhythmia can induce a low-output CHF state. There is usually dilation of all cardiac chambers, and there is preservation or thinning of biventricular wall thickness. Electrophysiologic changes, including prologued duration and decreased amplitude of action potentials in the myocytes, accompany this. All of these factors induce the typical neurohormonal response causing CHF. With rate control, these changes are often reversible due to myocardial hibernation.

Thyrotoxicosis is a rare cause of HF despite initiating a hyperdynamic circulatory state. This may be partially due to activation of the renin-angiotensin-aldosterone axis, causing sodium and water retention, as well as upregulation of erythropoietin-stimulating agent, both of which will cause increased blood volume. Sustained tachycardia with or without atrial fibrillation can also cause CHF.

High-output cardiac failure can be associated with thiamine deficiency, which is a rare condition found primarily among patients who are elderly, homeless, or have alcohol abuse disorder. Thiamine deficiency causes decreased ATP production with an accumulation of adenosine, which causes systemic vasodilation. This leads to lowered systemic vascular resistance and increased cardiac output. This evolves to weakened myocardium and decreased EF. Diuretic use can also cause urinary thiamine loss, further compounding the situation. Other common causes of high-output cardiac failure are obesity, liver disease, and arteriovenous shunts. The causative physiologic changes are decreased afterload (ie, systemic vascular resistance) and increased metabolism. These can often present with preserved EF, pulmonary congestion, increased filling pressures, and elevated natriuretic peptides.

Epidemiology

The global magnitude of the disease cannot be accurately assessed given the significant differences in geographical distribution, assessment methods, lack of imaging modalities, and non-adherence to the uniform staging and diagnosis of the disease. Approximately 1.2 million hospitalizations were due to CHF in 2017, with an increase in the percentage of patients with HFpEF compared to HFrEF.

By some reports, the incidence rate has plateaued; however, the prevalence increases as more patients receive therapy. This has not translated to improved quality of life or a decrease in the number of hospitalizations for patients with CHF. According to the Global Health Data Exchange registry, the current worldwide prevalence of CHF is 64.34 million cases. This translates to 9.91 million years lost due to disability (YLDs) and 346.17 billion US dollars in healthcare expenditure.

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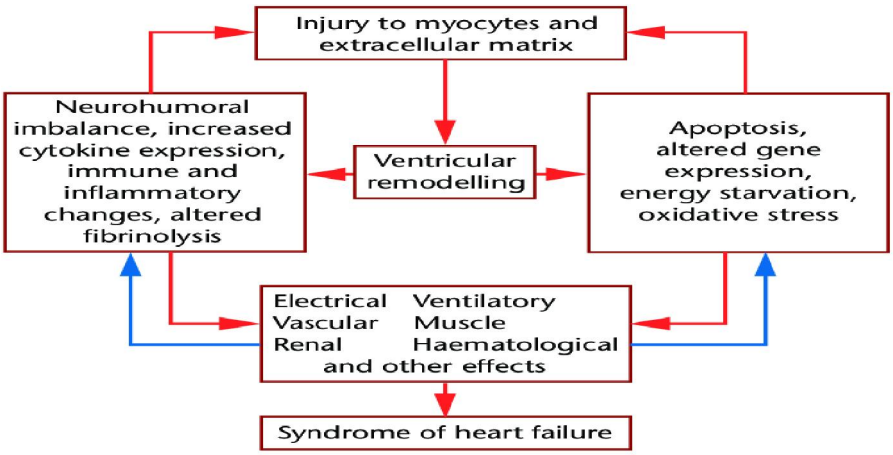
Age is a major determinant of HF. Regardless of the cause or the definition used to classify patients with HF, the prevalence of HF increases steeply with age. The Framingham Heart Study showed CHF prevalence to be 8 per 1000 males aged 50 to 59 years, with an increase to 66 per 1000 males aged 80 to 89. The incidence of HF in men doubles with each 10-year age increase after the age of 65, whereas in women, for the same age cohort, the incidence triples. Men have higher rates of heart disease and CHF than women worldwide.

The global registry also notes a predilection for a race with a 25% higher prevalence of HF in Black patients than in White patients. HF is still the primary cause of hospitalization in the elderly population and accounts for 8.5% of cardiovascular-related deaths in the United States.

International statistics regarding the epidemiology of HF are similar. The incidence increases dramatically with age, metabolic risk factors, and a sedentary lifestyle. Ischemic cardiomyopathy and hypertension are significant causes of HF in developing countries. A notable difference based on a review of small cohort studies from these nations is a higher prevalence of isolated right HF. The theoretical cause of this is thought to be due to the higher prevalence of tuberculous, pericardial, and lung diseases. There is a lack of robust data to verify these claims.

Pathophysiology



HF is a progressive disease. Any acute insult to cardiac structure or acute alteration secondary to genetic mutation, cardiac tissue infiltration, ischemia, valvular heart disease, myocarditis, or acute myocardial injury may initiate the compensatory mechanism, which, once exhausted, results in maladaptation.

In the initial stages of CHF, several compensatory mechanisms attempt to maintain cardiac output and meet the systemic demands. The chronic activation of the sympathetic nervous system results in reduced beta-receptor responsiveness and adrenaline stores. This results in changes in myocyte regeneration, myocardial hypertrophy, and myocardial hypercontractility. The increased sympathetic drive also results in the activation of the renin-angiotensin-aldosterone system (RAAS) system, systemic vasoconstriction, and sodium retention.

A decrease in cardiac output and increased sympathetic drive stimulate the RAAS, leading to increased salt and water retention, along with increased vasoconstriction. This further fuels the maladaptive mechanisms in the heart and causes progressive HF. In addition, the RAAS system releases angiotensin II, which has been shown to increase myocardial cellular hypertrophy and interstitial fibrosis, contributing to myocardial remodeling.

A decrease in cardiac output stimulates the neuroendocrine system with a release of epinephrine, norepinephrine, endothelin-1 (ET-1), and vasopressin. These mediators cause vasoconstriction, leading to increased afterload. There is an increase in cyclic adenosine monophosphate (cAMP), which causes an increase in cytosolic calcium in the myocytes. This increases myocardial contractility and further prevents myocardial relaxation. Increased afterload and myocardial contractility with impaired myocardial relaxation increase myocardial oxygen demand. This paradoxical

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need for increased cardiac output to meet myocardial demand eventually leads to myocardial cell death and apoptosis. As apoptosis continues, a decrease in cardiac output with increased demand leads to a perpetuating cycle of increased neurohumoral stimulation and maladaptive hemodynamic and myocardial responses. The loss of myocytes decreases EF (cardiac contractility), which leads to incomplete LV emptying. Increased LV volume and pressure cause pulmonary congestion.

Renal hypoperfusion causes the release of antidiuretic hormone (ADH), further potentiating sodium and water retention. Increased central venous and intraabdominal pressure causes reduced renal blood flow, further decreasing GFR.

Decompensated CHF is characterized by peripheral vasoconstriction and increased preload delivery to the overburdened heart. The natriuretic peptides BNP and ANP are secreted but are ineffective in counteracting the excess sodium and water retention.

Neprilysin is an enzyme that breaks down several hormones, including BNP, ANP, and bradykinin; it targets several novel therapeutics. It is always used with an angiotensin receptor blocker because it increases angiotensin II levels, and when administered with an ACE inhibitor, it causes significant angioedema.

Causes of CHF are split about equally between HFrEF and HFpEF but require different treatment plans. In HFpEF, there is a decrease in myocardial relaxation and an increase in the stiffness of the ventricle due to an increase in ventricular afterload. This perpetuates a similar maladaptive hemodynamic compensation and leads to progressive HF. Patients with HFpEF tend to be older, female, and hypertensive. Atrial fibrillation and anemia are also more likely co-occurring conditions. There is some evidence that the prognosis is worse than those with HFrEF. It is possible that appropriate targets have not been identified for optimal therapeutic interventions.

History and Physical Examination

History

The diagnosis and classification of HF are primarily based on the presence and severity of symptoms and physical exam findings. It is imperative to obtain a detailed history of symptoms, underlying medical conditions, and functional capacity to treat the patient adequately.

Acute CHF presents primarily with signs of congestion and may also present with organ hypoperfusion or cardiogenic shock. The most commonly reported symptom is shortness of breath. This must be further classified as exertional, positional (orthopnea), and whether acute or chronic. Other commonly reported symptoms of CHF include chest pain, anorexia, and exertional fatigue. Anorexia is due to hepatic congestion, bowel edema, and reduced blood flow to splanchnic circulation. Some patients may present with a recumbent cough due to orthopnea. Patients may also experience abdominal discomfort due to hepatic congestion or ascites. Patients with arrhythmias can present with palpitations, presyncope, or syncope.

Another symptom that increases morbidity is edema, especially of the lower extremities. This can limit mobility and balance; total body water and weight increases of > 20 lbs are not uncommon.

While patients with acute HF present with overt respiratory distress, orthopnea, and paroxysmal nocturnal dyspnea, patients with chronic heart failure tend to curtail their physical activity; hence, symptoms may be obscured. It is essential to identify triggers of acute decompensation such as recent infection, noncompliance with cardiac medications, use of NSAIDs, or increased salt intake.

Physical Examination

The examination findings vary with the stage and acuity of the disease. Patients may have isolated symptoms of left-sided HF, right-sided HF, or combined.

General physical examination: The general appearance of patients with severe CHF or those with acutely decompensated HF includes anxiety, diaphoresis, tachycardia, and tachypnea. Patients with chronic decompensated HF can appear cachexic. On chest examination, the classical finding of pulmonary rales translates to heart failure of moderate-to-severe intensity. Wheezing may be present in acute decompensated heart failure. As the severity of pulmonary congestion increases, frothy and blood-tinged sputum may be seen. It is important to note that the absence of

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rales does not exclude pulmonary congestion. Jugular venous distention is another classical finding that must be assessed in all patients with HF. In patients with elevated left-sided filling pressures, hepatojugular reflux (sustained increase in JVP of >4 cm after applying pressure over the liver with the patient lying at a 45° angle) is often seen.

Patients with Stage D HF may show signs of poor perfusion, such as hypotension, reduced capillary refill, cold extremities, poor mentation, and reduced urine output. There may be pulsus alternans (an alternating weak and strong pulse), suggestive of severe ventricular dysfunction. The pulse can be irregular in the presence of atrial fibrillation or ectopic beats. Some degree of peripheral edema is present with most HF. Weight gain is another method for assessing volume retention, and precise daily weights can be a useful monitoring tool.

Precordial findings in patients with HF include an S3 gallop, or displaced apex beat (dilated heart). There may be murmurs of associated valvular lesions such as the pansystolic murmur of mitral regurgitation or tricuspid regurgitation, systolic ejection murmur of aortic stenosis, or early diastolic murmur of aortic regurgitation. Patients with pulmonary hypertension may have palpable or loud P2 or parasternal heave. Patients with congenital heart disease may also have associated clubbing, cyanosis, and splitting of the second heart sound.

An S3 gallop is the most significant and early finding associated with HF. Patients with hypertensive heart disease may have an S4 or loud A2. Patients with HF with preserved EF may have an S4 gallop related to ventricular noncompliance.

The commonly used Framingham Diagnostic Criteria for Heart Failure require the presence of 2 major criteria or 1 major and 2 minor criteria to make the diagnosis. This clinical diagnostic tool is highly sensitive for the diagnosis of HF but has a relatively low specificity. The Framingham Diagnostic criteria are as follows:

Major Criteria

- Acute pulmonary edema
- Cardiomegaly
- Hepatojugular reflex
- Neck vein distention
- Paroxysmal nocturnal dyspnea or orthopnea
- Pulmonary rales
- Third heart sound (S3 Gallop)

Minor Criteria

- Ankle edema
- Dyspnea on exertion
- Hepatomegaly
- Nocturnal cough
- Pleural effusion
- Tachycardia (heart rate greater than 120 beats per minute)

Evaluation

A comprehensive assessment is required when evaluating a patient with HF. This includes a complete blood picture, iron profile, renal profile, and liver profile. After the basic metabolic and blood panel, patients require further investigations, depending on the etiology and clinical stage.

A **CBC** may suggest anemia or leukocytosis suggestive of an infection triggering CHF.

A **complete renal profile** is necessary for all patients with HF. It indicates the degree of renal injury associated with HF and guides medication choice. It is essential to know baseline renal function before the patient is started on medications, including renin-angiotensin-aldosterone (RAAS) inhibitors, sodium-glucose transporter-2 (SGLT-2) inhibitors, or diuretics. Serum sodium level has prognostic value as a predictor of mortality in patients with chronic HF. "The Outcomes of a Prospective Trial of Intravenous Milrinone for Exacerbations of Chronic Heart Failure" (OPTIME-

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CHF) trial demonstrated a significantly increased risk of in-hospital mortality as well as 30-day mortality in patients with HF who presented with hyponatremia.

A **liver profile** is usually performed. Hepatic congestion secondary to HF may result in elevated gamma-glutamyl transferase levels, aspartate aminotransferase (AST), and alanine aminotransferase (ALT).

Urine studies can be useful in diagnosis. If amyloidosis is suspected, urine and serum electrophoresis and monoclonal light chain assays should be performed. If clinical suspicion is high despite negative testing for light chains, bone scintigraphy can be performed.

Serum B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-ProBNP) levels can aid in differentiating cardiac from noncardiac causes of dyspnea in patients with ambiguous presentations. BNP is an independent predictor of increased left ventricular end-diastolic pressure, and it is used for assessing mortality risk in patients with HF. BNP levels correlate with NYHA classification, and the utility is primarily used as a marker to assess treatment efficacy. NT-ProBNP is the chemically inert N-terminal fragment of BNP and has a longer half-life. The ratio of NT-ProBNP/BNP varies depending on underlying comorbidities and may be a useful tool in the future. In patients with a clear clinical presentation of HF, natriuretic peptides should not be used to drive treatment plans. It is important to remember that BNP and NT-ProBNP levels can be elevated in patients with renal dysfunction, atrial fibrillation, and older patients. Conversely, BNP levels can be falsely low in patients with obesity, hypothyroidism, and advanced HF (due to myocardial fibrosis).

Troponin-I or T suggests ongoing myocardial injury when persistently elevated and predicts adverse outcomes and mortality.

An **electrocardiogram** may show evidence of prior infarction, chamber enlargement, intraventricular conduction delay, or arrhythmia. It may also give clues to specific etiologies. A low voltage and pseudo infarction pattern of ECG is seen in cardiac amyloidosis. An epsilon wave is seen in ARVC. ECG also suggests the presence of ventricular desynchrony, with a QRS duration of more than 120 msec, predicting the patient's response to device therapy for HF.

Chest radiographs are used to assess the degree of pulmonary congestion and cardiac contour (to determine the presence of cardiomegaly). Findings indicative of CHF on chest radiographs include enlarged cardiac silhouette, edema at the lung bases, and vascular congestion. In florid HF, Kerley B lines may be seen on chest radiographs. The absence of these findings in patients with a suggestive clinical presentation does not rule out CHF.

Echocardiography is the initial choice of modality in patients with suspected HF and is an easily available bedside tool. Echocardiography quantifies right and left ventricular function, denotes structural abnormalities in cardiac chambers and valves, and helps visualize the presence of focal wall motion abnormalities. However, in patients with severe obesity, pregnancy, or mechanical ventilation, it may be challenging to obtain adequate acoustic windows. Transesophageal echocardiography (TEE) is an alternative for these patients. Adequate rate control in patients with tachyarrhythmias is necessary to obtain adequate echocardiographic images.

Cardiac catheterization is often required for diagnosing ischemic cardiomyopathy and can be useful for accurately evaluating intracardiac pressures such as left ventricular end-diastolic pressure or pulmonary artery pressures.

Computed tomography may be used for the assessment of coronary artery disease in a young patient with ventricular dysfunction (older patients are likely to have baseline calcifications). It may also be used in patients with congenital heart diseases causing HF. Cardiac CT may help with the detection of tumors causing HF. CT may also be used for the evaluation of stent patency and graft evaluation.

SPECT-Myocardial Perfusion Imaging helps define the presence of ischemia in patients with newly diagnosed left ventricular dysfunction and not undergoing coronary angiography. It is particularly useful for assessing CAD in patients with no history of ischemia but elevated troponin. ECG-gated myocardial perfusion imaging is used to evaluate LV EF, regional wall motion, and regional wall thickening. EF measurement with this study may be affected in patients with an irregular heart rate, low count density, and extracardiac radiotracer uptake. ECG-gated images are also useful in recognizing artifactual defects seen on SPECT imaging, such as breast tissue and diaphragmatic attenuation.

Cardiac magnetic resonance imaging has evolved as an essential tool when a discrepancy exists between the clinical stage of the disease and echocardiographic findings. It helps with the precise evaluation of volume, chamber sizes, and ventricular function. It also assesses the stage of valvular heart disease in detail. Cardiac MRI also helps with the

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evaluation of complex congenital heart diseases. The tool can also be used for noninvasive assessment of conditions such as myocarditis, dilated cardiomyopathy, infiltrative cardiomyopathy, or arrhythmogenic right ventricular dysplasia.

Radionuclide multiple-gated acquisition (MUGA) scan is a reliable imaging technique for evaluating EF and is used in patients when there is a disparity of EF measurements from other studies.

Noninvasive stress imaging includes stress echocardiography, stress cardiac MRI, and SPECT imaging. These studies can be used to assess the benefit of coronary revascularization in patients with ischemic cardiomyopathy.

Genetic testing is indicated for identifying genetic variants causing cardiomyopathies, such as Titin, laminin A or C, myosin heavy chain, and cardiac troponin-T mutations.

Treatment / Management

The goal of therapy for chronic CHF is to improve symptoms and quality of life, decrease hospitalizations, and improve cardiac mortality. The goal of pharmacologic therapy is to control symptoms and to initiate and escalate drugs that reduce mortality and morbidity in HF.

Management for the respective stages of HF is outlined by the American College of Cardiology and the American Heart Association.

For Stage A (At-Risk for HF)

In patients with hypertension, guideline-directed medical therapy (GDMT) should be used for the management of hypertension.

In patients with type 2 diabetes, SGLT-2 inhibitors are indicated to reduce HF hospitalizations.

Lifestyle modifications such as healthy eating, physical activity, maintaining a normal weight, and avoidance of smoking are indicated.

The use of prognostication scores is recommended in patients with HF to estimate the risk of future HF events. Examples include the Framingham Heart Failure Risk Score (1999), Health ABC Heart Failure Score (2008), ARIC Risk Score (2012), and PCP-HF score (2019).

There should be optimal management of cardiovascular diseases in patients known to have coronary artery disease.

Patients at risk for HF due to exposure to cardiotoxic medications (eg, chemotherapy) should be managed with a multidisciplinary approach.

Natriuretic peptide screening and periodic evaluation are recommended.

For Stage B (Pre-HF)

Management of Stage B is focused on preventing clinical HF and reducing mortality and adverse cardiovascular events.

For patients with LV EF ≤40%, ACEi should be used to prevent clinical HF and for mortality reduction.

For patients with LV EF ≤ 40% and evidence of prior or recent acute coronary syndrome or myocardial infarction, the use of a statin and beta-blocker is recommended for reduction of mortality, CHF, and reducing adverse cardiovascular events.

For patients with LV EF ≤ 30% and receiving optimal medical therapy, with NYHA-class I and an expectation of meaningful survival of more than 1 year, a primary prevention ICD is recommended.

Beta-blockers are recommended for patients with LV EF ≤ 40%, irrespective of the etiology, to prevent symptomatic HF.

For patients with LV EF ≤ 50%, the use of thiazolidinediones and non-dihydropyridine calcium channel blockers increases the risk of adverse outcomes and HF hospitalizations, so should be avoided.

Valve repair, replacement, or interventions have associated guidelines for asymptomatic valvular heart disease.

Patients with congenital heart disease also have associated guidelines.

For Stage C (HF)

Multidisciplinary management is indicated for improving self-care and mortality of patients with HF.

Patient education and social support are required for optimal management.

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Vaccination against respiratory illnesses is effective in reducing mortality.

It is reasonable to screen patients for frailty, depression, low literacy, low social support, and resource and transport logistics during healthcare encounters.

A low-sodium diet is recommended.

Exercise training is effective in improving functional class and quality of life.

For patients with congestion, diuretics improve symptoms and reduce HF progression.

A thiazide diuretic (such as metolazone) should be added only to patients who do not respond well to a moderate or high dose of loop diuretics.

For patients with HFrEF, an ARNi is recommended to reduce mortality and morbidity. ARNi should not be given to patients who are intolerant of ACEi, and an ARB should be substituted. For patients not able to take an ARNi due to economic factors, the use of an ACEi or ARB is indicated. ARNi should not be used within 36 hours of the last dose of ACEi. For patients tolerating ACEi/ARB well, switching to ARNi is recommended, with a high economic value. As with ACEi, ARNi should not be given to patients with a history of angioedema.

For patients with HFrEF, the use of the beta-blockers carvedilol, bisoprolol, or sustained-release metoprolol is effective in reducing mortality and hospitalization.

For patients with HFrEF, NYHA class II-IV, an eGFR of more than 30 mL/min/1.73 m2 and a serum potassium of less than 5.0 mEq/L, the use of MRA is recommended. For patients with a serum potassium of more than 5.0 mEq/L, the use of MRA is harmful.

For patients with HFrEF, the use of SGLT-2 inhibitors is recommended to reduce mortality and HF hospitalization, irrespective of the diabetes status.

For African American patients with HFrEF and NYHA class III-IV, who are already receiving optimal medical therapy (OMT), the addition of a combination of hydralazine and nitrate is recommended to reduce morbidity and mortality. This is of high economic value.

For patients with HFrEF and intolerant to RAASi or in whom RAASi is contraindicated due to renal insufficiency, the use of a combination of hydralazine and nitrate might be effective.

It is recommended to titrate medications aggressively to achieve desired outcomes. This can be done as frequently as 1-2 weeks as tolerated.

Ivabradine can be useful in patients on OMT with and heart rate of more than 70 bpm, providing mortality benefits, and reducing HF hospitalization.

Digoxin may be considered in symptomatic patients with sinus rhythm despite adequate goal-directed therapy to reduce the all-cause rate of hospitalizations, but its role is limited.

In patients with HFrEF and recent HF, an oral soluble guanylate cyclase stimulator (Vericiguat) might be useful in reducing mortality and HF hospitalization. Vericiguat is a soluble guanylate cyclase stimulator that stimulates the intracellular receptor for endogenous NO, which is a potent vasodilator. It also improves cardiac contractility.

Device therapy:

An implantable cardioverter-defibrillator (ICD) is indicated for primary prevention of sudden cardiac death in patients with HF who have an LVEF of less than or equal to 35% and an NYHA functional class of II to III while on goal-directed medical therapy. It is also indicated if a patient has NYHA functional class I and an EF of less than or equal to 30% on adequate medical therapy.

Cardiac resynchronization therapy (CRT) with biventricular pacing is recommended in patients with HFrEF and an NYHA functional class of II to III or ambulatory class IV with an LVEF less than or equal to 35%, QRS duration $\geq$ 150 msec, and sinus rhythm with left bundle branch block (LBBB) morphology. It can also be considered in non-LBBB morphology and QRS $\geq$ 150 msec.

Revascularization is indicated in selected patients with coronary artery disease and HFrEF while on GDMT.

Valvular heart disease interventions such as transcatheter edge-to-edge mitral valve repair or mitral valve surgery might be beneficial for patients with HF and on GDMT.

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For Stage D (Advanced HF)

Referral to an HF specialist is indicated.

It is reasonable to utilize inotropic support and device therapy in patients awaiting mechanical cardiac support or transplant. Inotropic support alone can be used in patients not eligible for a transplant or mechanical cardiac support.

Mechanical cardiac support such as a durable left ventricle assist device (LVAD) or ECMO can be beneficial as a bridge to transplant.

For highly selected patients, cardiac transplant is indicated to improve survival and quality of life.

Goals of care should be decided by shared decision-making. This includes considering comorbid conditions, frailty, and socio-economic support. Palliative care should be offered as indicated after shared decision-making.

Congestive heart failure (CHF) is a condition in which the heart is unable to pump blood effectively, leading to fluid accumulation in the lungs and other body tissues. Various medications are available to manage CHF, which can help alleviate symptoms and improve the patient's quality of life. This article will explore the classification of drugs used for CHF, their pharmacological actions, uses, adverse effects, contraindications, and drug interactions.

IV. CLASSIFICATION OF CONGESTIVE HEART FAILURE DRUGS

1.1. Class I: Inotropes

Examples include digoxin and milrinone.

Inotropes function by increasing the force of heart muscle contractions, thus improving cardiac output. Here's a detailed look at the mechanisms of action for two inotropes, digoxin and milrinone:

Digoxin: Digoxin is a cardiac glycoside that primarily acts by inhibiting the sodium-potassium ATPase pump. This inhibition increases intracellular sodium levels, which then leads to an increase in intracellular calcium via the sodium-calcium exchanger. The increased calcium levels boost the force of myocardial contractions, thereby improving cardiac output. Additionally, digoxin has a vagotonic effect, which slows down atrioventricular (AV) node conduction and can help control heart rate in certain arrhythmias.

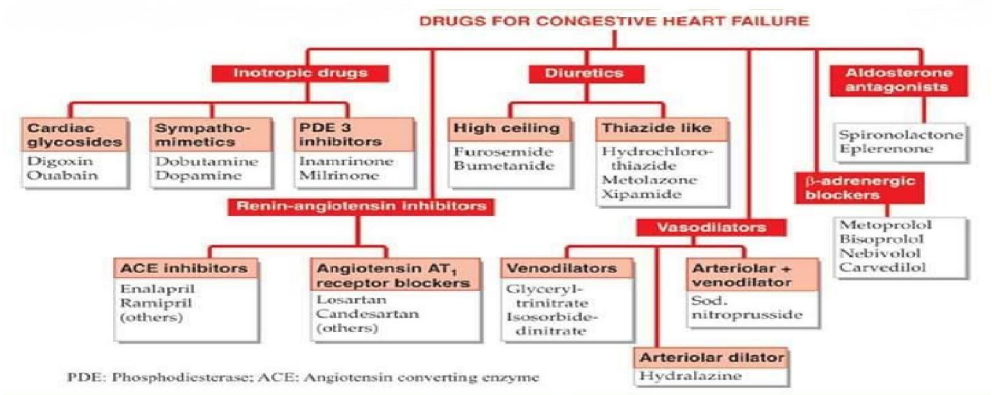
Milrinone: Milrinone is a phosphodiesterase-3 (PDE-3) inhibitor. By inhibiting PDE-3, milrinone increases cyclic adenosine monophosphate (cAMP) levels in cardiac myocytes and vascular smooth muscle cells. The elevated cAMP levels cause increased calcium influx into the sarcoplasmic reticulum, thereby enhancing myocardial contractility. Milrinone also leads to vasodilation, as the increased cAMP levels in vascular smooth muscle cells cause relaxation.

1.2. Class II: Diuretics

Examples include furosemide, bumetanide, and hydrochlorothiazide.

Diuretics function by increasing urine production, which in turn helps remove excess fluid from the body. This reduces enema and pulmonary congestion. Here's a detailed look at the mechanisms of action for three diuretics, furosemide, bumetanide, and hydrochlorothiazide:

Furosemide and **Bumetanide**: These loop diuretics primarily act by inhibiting the sodium-potassium-chloride



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cotransporter 2 (NKCC2) in the thick ascending limb of the loop of Henle in the nephron. This inhibition prevents the reabsorption of sodium, potassium, and chloride ions, leading to increased water excretion and reduced fluid retention.

Hydrochlorothiazide: Hydrochlorothiazide is a thiazide diuretic that primarily acts by inhibiting the sodium-chloride cotransporter (NCC) in the distal convoluted tubule of the nephron. This inhibition prevents the reabsorption of sodium and chloride ions, leading to increased water excretion and reduced fluid retention.

1.3. Class III: Vasodilators

Examples include hydralazine, nitrates, and angiotensin-converting enzyme (ACE) inhibitors.

Vasodilators relax blood vessels, thereby lowering blood pressure and reducing the heart's workload. Here's a detailed look at the mechanisms of action for three vasodilators, hydralazine, nitrates, and ACE inhibitors:

Hydralazine: Hydralazine is a direct-acting vasodilator that primarily acts by relaxing vascular smooth muscle cells through interference with calcium influx and mobilization. This relaxation leads to a decrease in peripheral resistance and lower blood pressure.

Nitrates: Nitrates, such as nitroglycerin, function by releasing nitric oxide (NO) in the vascular smooth muscle cells. NO activates guanylate cyclase, leading to increased cyclic guanosine monophosphate (cGMP) levels. Elevated cGMP levels cause the vascular smooth muscle cells to relax, resulting in vasodilation and a decrease in preload and afterload.

ACE inhibitors: Angiotensin-converting enzyme (ACE) inhibitors, like lisinopril, block the conversion of angiotensin I to angiotensin II. This prevents the vasoconstrictive and aldosterone-secreting effects of angiotensin II, leading to reduced vasoconstriction, decreased blood pressure, and decreased sodium and water retention.

1.4. Class IV: Beta-Blockers

Examples include metoprolol, carvedilol, and bisoprolol.

Beta-blockers block the effects of catecholamines (epinephrine and norepinephrine) on beta-adrenergic receptors, slowing down the heart rate and decreasing blood pressure. This reduces the heart's oxygen demand. Here's a detailed look at the mechanisms of action for three beta-blockers, metoprolol, carvedilol, and bisoprolol:

Metoprolol and **Bisoprolol**: These are selective beta-1 adrenergic receptor blockers, meaning they predominantly block beta-1 receptors found in the heart. This blockade decreases heart rate, contractility, and blood pressure, ultimately reducing myocardial oxygen consumption and workload.

Carvedilol: Carvedilol is a non-selective beta-blocker, blocking both beta-1 and beta-2 adrenergic receptors and alpha-1 adrenergic receptors. By blocking beta-1 receptors, carvedilol decreases heart rate and contractility, and blocking alpha-1 receptors causes vasodilation. This combination of actions results in a reduction of blood pressure, myocardial oxygen consumption, and workload.

1.5. Class V: Miscellaneous

Examples include spironolactone, an aldosterone antagonist, and ivabradine, a heart rate-modulating agent.

This class includes medications that don't fit into the previous categories but are still used to manage CHF. Here's a detailed look at the mechanisms of action for two such medications, spironolactone and ivabradine:

Spironolactone: Spironolactone is an aldosterone antagonist that competes with aldosterone for binding to mineralocorticoid receptors in the distal nephron. By blocking the action of aldosterone, spironolactone prevents sodium and water reabsorption while promoting potassium retention. This reduces fluid retention, edema, and pulmonary congestion and lowers blood pressure.

Ivabradine: Ivabradine is a heart rate-modulating agent that selectively inhibits the funny current (If) in the sinoatrial (SA) node of the heart. This inhibition slows down the spontaneous depolarization rate of the SA node, resulting in a reduction of heart rate without affecting contractility or conduction. This reduced heart rate allows for improved filling of the ventricles and increased cardiac output, ultimately reducing the workload of the heart in CHF patients.

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2.Examples of Individual Drugs

2.1. Digoxin

An inotropic drug derived from the foxglove plant, digoxin helps to increase the force of heart muscle contractions, improving cardiac output.

2.2. Furosemide

Furosemide is a potent diuretic that promotes the excretion of excess fluid from the body, reducing edema and pulmonary congestion.

2.3. Hydralazine

Hydralazine is a vasodilator that works by relaxing the smooth muscle in blood vessels, which in turn reduces blood pressure and the workload on the heart.

2.4. Metoprolol

Metoprolol is a beta-blocker that slows the heart rate and decreases blood pressure, reducing the heart's oxygen demand and helping to manage CHF symptoms.

2.5. Spironolactone

Spironolactone is an aldosterone antagonist that helps manage fluid balance and blood pressure, providing additional benefits in the treatment of CHF.

3. Pharmacological Actions

Each class of CHF drugs has unique pharmacological actions:

Inotropes: Increase the force of heart muscle contractions.

Diuretics: Promote the excretion of excess fluid from the body.

Vasodilators: Relax blood vessels, reducing blood pressure and the workload on the heart.

Beta-blockers: Slow the heart rate and decrease blood pressure.

Miscellaneous: Provide additional benefits through various mechanisms, such as inhibiting aldosterone or modulating heart rate.

4. Uses

These drugs are used to manage symptoms and improve the quality of life for individuals living with congestive heart failure (CHF). They can help:

Increase cardiac output

Reduce edema and pulmonary congestion

Lower blood pressure

Slow heart rate

Minimize the risk of further heart damage

5. Adverse Effects

As with any medication, CHF drugs can have adverse effects. Some common side effects include:

Inotropes: Nausea, vomiting, irregular heartbeat

Diuretics: Dehydration, electrolyte imbalances, kidney dysfunction

Vasodilators: Dizziness, headache, flushing

Beta-blockers: Fatigue, dizziness, bradycardia (slow heart rate)

6. Contraindications

Certain individuals should not take These drugs or should do so under close medical supervision. Contraindications can include:

Inotropes: Ventricular fibrillation, hypersensitivity to the drug

Diuretics: Severe kidney or liver dysfunction, anuria (inability to produce urine)

Vasodilators: Hypotension (low blood pressure), severe kidney or liver dysfunction

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Beta-blockers: Asthma, bradycardia, heart block

7. Drug Interactions

These drugs can interact with other medications, potentially leading to adverse effects or diminished effectiveness. Examples of potential drug interactions include:

Inotropes: Calcium channel blockers, beta-blockers, diuretics

Diuretics: ACE inhibitors, nonsteroidal anti-inflammatory drugs (NSAIDs), lithium

Vasodilators: Other antihypertensive medications, nitrates

Beta-blockers: Calcium channel blockers, insulin, and other antihypertensive medications

V. CONCLUSION

Congestive heart failure (CHF) is a chronic condition where the heart is unable to pump blood efficiently, leading to a buildup of fluid in the lungs, liver, and other parts of the body. It can result from various underlying conditions such as coronary artery disease, high blood pressure, or heart valve problems. CHF is a progressive disease that requires careful management to improve quality of life and reduce symptoms.

Treatment typically involves lifestyle changes, medications (such as diuretics, ACE inhibitors, or beta-blockers), and in some cases, surgical interventions like heart valve repair or implanting devices such as pacemakers or defibrillators. Early diagnosis and effective treatment are crucial for managing symptoms and preventing complications.

While CHF is not curable, with proper management, individuals can lead relatively normal lives. However, ongoing monitoring and adjustments in treatment are essential to prevent the condition from worsening. It's important for patients to adhere to prescribed therapies and maintain a heart-healthy lifestyle to improve outcomes and quality of life.

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