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Review Article

A REVIEW ON CAUSES SYMPTOMS PATHOPHYSIOLOGY DIAGNOSIS AND THERAPY OF CONGESTIVE HEART FAILURE

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Abstract: Congestive heart failure (CHF) occurs when the heart isn't able to pump blood normally.. The left ventricle no longer functions as an adequate pump to maintain normal cardiac output (normal ejection fractions). As a result, there is not enough blood flow to provide the body's organs with oxygen and nutrients. CHF is usually the result of other health problems. Heart failure can cause a number of symptoms including shortness of breath, leg swelling, and exercise intolerance. The appearance of symptoms of CHF can be delayed for years as the heart tries to compensate when it is not pumping efficiently. The pathophysiology of heart failure is complex, and there is no single lesion. Any form of heart disease can lead to heart failure. The condition is diagnosed with echocardiography and blood tests. Treatment commonly consists of lifestyle measures such as smoking cessation, light exercise including breathing protocols, decreased salt intake and other dietary changes, and medications. This article briefly reviews about various causes, symptoms, pathophysiology and treatment of congestive heart failure.

Key words: Congestive heart failure, systolic dysfunction, Diastolic dysfunction

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.[1]

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 fillingwith preserved systolic function) may occur in up to40–50% 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 leftventricular function may not have diastolic dysfunction; instead, their symptoms are caused by other etiologies, such as lung disease, obesity, or occult coronary ischemia.[3]

Causes of Congestive Heart Failure

CHF is usually the result of other health problems:

- Coronary artery disease, a condition that causes narrowing of the arteries that supply the heart with blood, can damage and weaken areas of the heart
- Persistent high blood pressure forces the heart to pump against higher pressure, which causes it to weaken over time -

people who have uncontrolled high blood pressure are more likely to develop CHF than those who don't

- Heart attack damages the heart muscle - people who have had heart attacks are at 5 times the average risk of developing CHF
- Diabetes also increases CHF risk
- Arrhythmias (abnormal heart rhythms) can cause the heart to pump inefficiently
- Heart valve disease may have been caused by abnormalities that have been present since birth or have developed over time
- Heart valve damage may have been caused by rheumatic disease or infection
- Viral infection of the heart muscle can seriously weaken the heart
- An enlarged wall between the heart chambers (a genetic condition) may be a cause
- Certain kidney conditions that increase blood pressure and fluid buildup can increase the risk of CHF by placing more stress on the heart

In addition, all the risk factors that normally increase the chances of heart disease, such as smoking and obesity, also increase your risk of congestive heart failure.

Symptoms and Complications of Congestive Heart Failure

The heart compensates in three ways:

- dilating (enlarging) to form a bigger pump
- adding new muscle tissue to pump harder
- beating at a faster rate

As the heart compensates, several things happen that can result in symptoms. The heart cannot pump well enough to pump the blood through the body and back to the heart again. Blood then backs up into the legs and the lungs, causing fluid buildup. This causes visible swelling of the ankles and legs and shortness of breath.

The most common symptoms of CHF include:

- breathing difficulties during the night or when lying down
- coughing and wheezing
- fatigue and weakness
- shortness of breath
- swollen ankles

Other symptoms of CHF include:

- abdominal pain, bloating, or loss of appetite
- accumulation of fluid in the abdomen
- bluish skin around the mouth
- constipation
- pale skin and cold hands or feet
- urination at night

Pathophysiology of CHF:

Heart failure is caused by any condition which reduces the efficiency of the myocardium, or heart muscle, through damage or overloading. As such, it can be caused by as- diverse an array of conditions as myocardial infarction (in which the heart muscle is starved of oxygen and dies), hypertension (which increases the force of contraction needed to pump blood) and amyloidosis (in which protein is deposited in the heart muscle, causing it to stiffen). Over time these increases in workload will produce changes to the heart itself:

- Reduced force of contraction, due to overloading of the ventricle. In a healthy heart, increased filling of the ventricle results in increased force of contraction (by the Frank–Starling law of the heart) and thus a rise in cardiac output. In heart failure this mechanism fails, as the ventricle is loaded with blood to the point where heart muscle contraction becomes less efficient. This is due to reduced ability to cross-link actin and myosin filaments in over-stretched heart muscle.[4]
- A reduced stroke volume, as a result of a failure of systole, diastole or both. Increased end systolic volume is usually caused by reduced contractility. Decreased end diastolic volume results from impaired ventricular filling – as occurs when the compliance of the ventricle falls (i.e. when the walls stiffen).
- Reduced spare capacity. As the heart works harder to meet normal metabolic demands, the amount cardiac output can increase in times of increased oxygen demand (e.g. exercise) is reduced. This contributes to the exercise intolerance commonly seen in heart failure. This translates to the loss of one's cardiac reserve, or the ability of the heart to work harder during strenuous physical activity. Since the heart has to work harder to meet the normal metabolic demands, it is incapable of meeting the metabolic demands of the body during exercise.
- Increased heart rate, stimulated by increased sympathetic activity [5]in order to maintain cardiac output. Initially, this helps compensate for heart failure by maintaining blood pressure and perfusion, but places further strain on the myocardium, increasing coronary perfusion requirements, which can lead to worsening of ischemic heart disease. Sympathetic activity may also cause potentially fatal arrhythmias.
- Hypertrophy (an increase in physical size) of the myocardium,(figure;1) caused by the terminally differentiated heart muscle

fibres increasing in size in an attempt to improve contractility. This may contribute to the increased stiffness and decreased ability to relax during diastole.

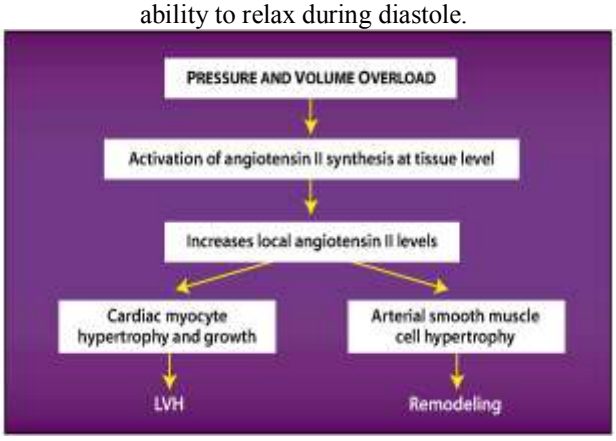

Figure 1: Response to pressure and volume loading of the left ventricle LVH: Left ventricular hypertrophy

- Enlargement of the ventricles, contributing to the enlargement and spherical shape of the failing heart. The increase in ventricular volume also causes a reduction in stroke volume due to mechanical and contractile inefficiency.[6]
- The general effect is one of reduced cardiac output and increased strain on the heart. This increases the risk of cardiac arrest (specifically due to ventricular dysrhythmias), and reduces blood supply to the rest of the body. In chronic disease the reduced cardiac output causes a number of changes in the rest of the body, some of which are physiological compensations, some of which are part of the disease process:
- Arterial blood pressure falls. This destimulates baroreceptors in the carotid sinus and aortic arch which link to the nucleus tractus solitarius. This center in the brain increases sympathetic activity, releasing catecholamines into the blood stream. Binding to alpha-1 receptors results in systemic arterial vasoconstriction. This helps restore blood pressure but also increases the total peripheral resistance, increasing the workload of the heart. Binding to beta-1 receptors in the myocardium increases the heart rate and make contractions more forceful, in an attempt to increase cardiac output. This also, however, increases the amount of work the heart has to perform.
- Increased sympathetic stimulation also causes the hypothalamus to secrete vasopressin (also known as antidiuretic hormone or ADH), which causes fluid retention at the kidneys. This increases the blood volume and blood pressure.
- Reduced perfusion (blood flow) to the kidneys stimulates the release of renin – an enzyme which catalyses the production of the potent vasopressor angiotensin. Angiotensin and its metabolites cause further vasoconstriction, and stimulate increased secretion of the steroid aldosterone from the adrenal glands. This promotes salt and fluid retention at the kidneys.
- The chronically high levels of circulating neuroendocrine hormones such as catecholamines, renin, angiotensin, and aldosterone affects the myocardium directly, causing structural remodelling of the heart over the long term. Many of these remodelling effects seem to be mediated by transforming growth factor beta (TGF-beta), which is a common downstream target of the signal transduction cascade. initiated by catecholamines [7]and angiotensin II[8], and also by epidermal growth factor (EGF), which is a target of the signaling pathway activated by aldosterone[9]
- Reduced perfusion of skeletal muscle causes atrophy of the muscle fibres. This can result in weakness, increased fatigueability and decreased peak strength - all contributing to exercise intolerance.[10]
- The increased peripheral resistance and greater blood volume place further strain on the heart and accelerates the process of damage to the myocardium. Vasoconstriction and fluid retention produce an increased hydrostatic pressure in the capillaries. This shifts the balance of forces in favour of interstitial fluid formation as the increased pressure forces additional fluid out of the blood, into the tissue. This results in edema (fluid build-up) in the tissues. In right-sided heart failure this commonly starts in the ankles where venous pressure is high due to the effects of gravity (although if the patient is bed-ridden, fluid accumulation may begin in the sacral region.) It may also occur in the abdominal cavity, where the fluid build-up is called ascites. In left-sided heart failure edema can occur in the lungs - this is called cardiogenic pulmonary edema. This reduces spare capacity for ventilation, causes stiffening of the lungs and reduces the efficiency of gas exchange by increasing the distance between the air and the blood. The consequences of this are dyspnea (shortness of breath), orthopnea and paroxysmal nocturnal dyspnea.

The symptoms of heart failure are largely determined by which side of the heart fails. The left side pumps blood into the systemic circulation, whilst the right side pumps blood into the pulmonary circulation. Whilst left-sided heart failure will reduce cardiac output to the systemic circulation, the initial symptoms often manifest due to effects on the pulmonary circulation. In systolic dysfunction, the ejection fraction is decreased, leaving an abnormally elevated volume of blood in the left ventricle. In diastolic dysfunction, end-diastolic ventricular pressure will be high. This increase in volume or pressure backs up to the left atrium and then to the pulmonary veins. Increased volume or pressure in the pulmonary veins impairs the normal drainage of the alveoli and favors the flow of fluid from the capillaries to the lung parenchyma, causing pulmonary edema. This

impairs gas exchange. Thus, left-sided heart failure often presents with respiratory symptoms: shortness of breath, orthopnea and paroxysmal nocturnal dyspnea.

In severe cardiomyopathy, the effects of decreased cardiac output and poor perfusion become more apparent, and patients will manifest with cold and clammy extremities, cyanosis, claudication, generalized weakness, dizziness, and syncope.

The resultant hypoxia caused by pulmonary edema causes vasoconstriction in the pulmonary circulation, which results in pulmonary hypertension. Since the right ventricle generates far lower pressures than the left ventricle (approximately 20 mmHg versus around 120 mmHg, respectively, in the healthy individual) but nonetheless generates cardiac output exactly equal to the left ventricle, this means that a small increase in pulmonary vascular resistance causes a large increase in amount of work the right ventricle must perform. However, the main mechanism by which left-sided heart failure causes right-sided heart failure is actually not well understood. Some theories invoke mechanisms that are mediated by neurohormonal activation[11]. Mechanical effects may also contribute. As the left ventricle distends, the intraventricular septum bows into the right ventricle, decreasing the capacity of the right ventricle.

Systolic dysfunction

Heart failure caused by systolic dysfunction is more readily recognized. It can be simplistically described as failure of the pump function of the heart. It is characterized by a decreased ejection fraction (less than 45%). The strength of ventricular contraction is attenuated and inadequate for creating an adequate stroke volume, resulting in inadequate cardiac output. In general, this is caused by dysfunction or destruction of cardiac myocytes or their molecular components. In congenital diseases such as Duchenne muscular dystrophy, the molecular structure of individual myocytes is affected. Myocytes and their components can be damaged by inflammation (such as in myocarditis) or by infiltration (such as in amyloidosis). Toxins and pharmacological agents (such as ethanol, cocaine, doxorubicin, and amphetamines) cause intracellular damage and oxidative stress. The most common mechanism of damage is ischemia causing infarction and scar formation. After myocardial infarction, dead myocytes are replaced by scar tissue, deleteriously affecting the function of the myocardium. On echocardiogram, this is manifest by abnormal or absent wall motion.

Because the ventricle is inadequately emptied, ventricular end-diastolic pressure and volumes increase. This is transmitted to the atrium. On the left side of the heart, the increased pressure is transmitted to the pulmonary vasculature, and the resultant hydrostatic pressure favors extravassation of fluid into the lung parenchyma, causing pulmonary edema. On the right side of the heart, the increased pressure is transmitted to the systemic venous circulation and systemic capillary beds, favoring extravassation of fluid into the tissues of target organs and extremities, resulting in dependent peripheral edema.

Diastolic dysfunction

Heart failure caused by diastolic dysfunction is generally described as the failure of the ventricle to adequately relax and typically denotes a stiffer ventricular wall. This causes inadequate filling of the ventricle, and therefore results in an inadequate stroke volume. The failure of ventricular relaxation also results in elevated end-diastolic pressures, and the end result is identical to the case of systolic dysfunction (pulmonary edema in left heart failure, peripheral edema in right heart failure.)

Diastolic dysfunction can be caused by processes similar to those that cause systolic dysfunction, particularly causes that affect cardiac remodeling.

Diastolic dysfunction may not manifest itself except in physiologic extremes if systolic function is preserved. The patient may be completely asymptomatic at rest. However, they are exquisitely sensitive to increases in heart rate, and sudden bouts of tachycardia (which can be caused simply by physiological responses to exertion, fever, or dehydration, or by pathological tachyarrhythmias such as atrial fibrillation with rapid ventricular response) may result in flash pulmonary edema. Adequate rate control (usually with a pharmacological agent that slows down AV conduction such as a calcium channel blocker or a beta-blocker) is therefore key to preventing decompensation.

Left ventricular diastolic function can be determined through echocardiography by measurement of various parameters such as the E/A ratio (early-to-atrial left ventricular filling ratio), the E (early left ventricular filling) deceleration time, and the isovolumic relaxation time.

Diagnosis

No system of diagnostic criteria has been agreed as the gold standard for heart failure. Commonly used systems are the "Framingham criteria" (derived from the Framingham Heart Study), the "Boston criteria",the "Duke criteria", and (in the setting of acute myocardial infarction) the "Killip class".[15]

Imaging

Echocardiography is commonly used to support a clinical diagnosis of heart failure. This modality uses ultrasound to determine the stroke volume (SV, the amount of blood in the heart that exits the ventricles with each beat), the end-diastolic volume (EDV, the total amount of blood at the end of diastole), and the SV in proportion to the EDV, a value known as the ejection fraction (EF). In pediatrics, the shortening fraction is the preferred measure of systolic function. Normally, the EF should be between 50% and 70%; in systolic heart failure, it drops below 40%. Echocardiography can also

identify valvular heart disease and assess the state of the pericardium (the connective tissue sac surrounding the heart). Echocardiography may also aid in deciding what treatments will help the patient, such as medication, insertion of an implantable cardioverter-defibrillator or cardiac resynchronization therapy. Echocardiography can also help determine if acute myocardial ischemia is the precipitating cause, and may manifest as regional wall motion abnormalities on echo.

Chest X-rays are frequently used to aid in the diagnosis of CHF. In the compensated patient, this may show cardiomegaly (visible enlargement of the heart), quantified as the cardiothoracic ratio (proportion of the heart size to the chest). In left ventricular failure, there may be evidence of vascular redistribution ("upper lobe blood diversion" or "cephalization"), Kerley lines, cuffing of the areas around the bronchi, and interstitial edema.

Electrophysiology

An electrocardiogram (ECG/EKG) may be used to identify arrhythmias, ischemic heart disease, right and left ventricular hypertrophy, and presence of conduction delay or abnormalities (e.g. left bundle branch block). Although these findings are not specific to the diagnosis of heart failure a normal ECG virtually excludes left ventricular systolic dysfunction.[16]

Blood tests

Blood tests routinely performed include electrolytes (sodium, potassium), measures of renal function, liver function tests, thyroid function tests, a complete blood count, and often C-reactive protein if infection is suspected. An elevated B-type natriuretic peptide (BNP) is a specific test indicative of heart failure. Additionally, BNP can be used to differentiate between causes of dyspnea due to heart failure from other causes of dyspnea. If myocardial infarction is suspected, various cardiac markers may be used.

According to a meta-analysis comparing BNP and N-terminal pro-BNP (NTproBNP) in the diagnosis of heart failure, BNP is a better indicator for heart failure and left ventricular systolic dysfunction. In groups of symptomatic patients, a diagnostic odds ratio of 27 for BNP compares with a sensitivity of 85% and specificity of 84% in detecting heart failure.[17]

Angiography

Heart failure may be the result of coronary artery disease, and its prognosis depends in part on the ability of the coronary arteries to supply blood to the myocardium (heart muscle). As a result, coronary catheterization may be used to identify possibilities for revascularisation through percutaneous coronary intervention or bypass surgery.

Therapy for Congestive Heart Failure

Understanding the pathophysiology of heart failure allows one to achieve the goals of treatment, which are to relieve symptoms, avoid hospital admissions, and prolong life. Treatment for CHF consists of a combination of pharmacologic and nonpharmacologic therapies. Much literature and research has been published on medical management of CHF. The basic theories include termination of the renin-angiotensin system to prevent the long-term complications of the cascade. Treatment often focuses on a combination of after load-reduction with angiotensin-converting- enzyme (ACE) inhibitors, reduction of catecholamine with β-blockers, and preload-reduction with diuretics. Each modality presents difficulties for certain characteristic patient types, and patients with diastolic dysfunction often develop dizziness or hypotension with over diuresis[18]. Although clinical trials have shown significantly lower mortality with multiple interventions, the overall death rate from heart failure continues to rise,[19] which reflects the avoidance of premature mortality from predisposing conditions (myocardial infarction and hypertension) that are only palliated, not cured, by advances in therapy.

Congestive heart failure can be treated with the following medications:

The Cardiac Glycosides (Cardenolides) - the Digitalis Preparations are most useful in treating CHF

Drug Members :

- Digitoxin (Crystodigin)
- Digoxin (Lanoxin)
- Deslanoside (Cedilanid-D)

Natural plant analogs of today's modern glycoside preparations have been used for at least 3,000 years. Cardiac glycosides were used for heart conditions by the Egyptians, Romans and the early Europeans. The cardiac glycosides are commonly found in plants such as Milkweed, Lilly of the Valley, the Oleander plant, and in the Foxglove plant. This is a very good reason to preserve the earth's rain forests because of the very real chance they hold plants that will one day cure diseases.

Mechanisms of Action:

The main action of the Cardiac Glycosides is to increase the force of cardiac contraction. They do this in the following ways:

1. A rise in the concentration of intracellular sodium. An enzyme called Na+- K+ ATPase cleaves ATP to ADP and Pi. The energy released from the hydrolysis of ATP drives the Na+-K+ pump which normally pumps Na+ out of the cell and K+ into the cell. If, however, this pump is disabled by the inhibition of this enzyme, the net effect is the malfunction of the pump and an increase of sodium inside the cell with a loss of intracellular potassium to the extracellular space. The influx of Na+ is partly due to the passive re-entry of sodium inside the cell while the efflux of K+ is passive to the outside of the cell.

2. A rise in the concentration of intracellular calcium. In the heart, there is a second pump called the $Na+$-Ca_2+ pump. This pump normally takes 1 intracellular Ca_2+ ion out of the myocyte in exchange for 4 extracellular $Na+$ ions brought into the myocyte. This pump is turned on by a diffusion gradient difference in extracellular to intracellular sodium when the extracellular sodium concentration is higher than the intracellular sodium concentration. When the [Na+] outside drops because the [Na+]Inside rises, then the pump becomes deranged and stops pumping Ca_2+ out of the cell. When the $Na+$ - $K+$ pump is disabled, there is a rise of [Na+] inside the cell as well as a rise in the [Ca_2+] inside. The [Ca_2+] inside will rise because of passive diffusion back into the cell coupled with the fact that the cell is not pumping any Ca_2+ ions out.

Additional Mechanisms of Action:

1. The glycosides enhance vagal tone over the heart which:

- slows the heart rate
- slows the AV node conduction velocity
- increases the AV nodal refractory period

The net effect of the glycosides on the heart is as follows:

a. heart rate is slowed
b. contraction is greater due to increased filling volumes - Starling's Law
c. ejection fraction is improved
d. increased ejection velocity

Adverse side effects of the glycosides:

Fatigue, delirium, anorexia, headaches, hallucinations, visual disturbances, atrioventricular blocks, and EKG changes, nausea, vomiting, diarrhea, anorexia, blurred vision, color vision disturbances, and toxic psychosis.

EKG changes seen with administration of the glycosides:

- Prolonged P-R Interval
- Inverted T Wave
- S-T Segment Depression
- Shortened Q-T Interval

Medical Uses:

1. Used to treat congested heart failure
2. Used to treat/suppress supraventricular tachydysrhythmias such as:

a. atrial flutter
b. atrial fibrillation
c. paroxysmal supraventricular tachycardia (PSVT)

II. Angiotensin Converting Enzyme Inhibitors (ACE Inhibitors)

Drug Members:

- Captopril (Capoten)
- Enalapril (Vasotec)
- Lisinopril (Prinivil)

Mechanism of the ACE Inhibitors - Blood Pressure Regulation - The Renin-Angiotensin Axis

Renin is released into the blood from the kidneys when blood pressure is low. Renin changes angiotensinogen in the blood to Angiotensin I which then, in the presence of angiotensin converting enzyme, is changed into Angiotensin II. Angiotensin II is a potent vasoconstrictor. An increased peripheral resistance (higher blood pressure due to vasoconstriction) creates a lot of afterload on the left ventrical. Untreated and sustained hypertension will eventually create so much work for the left ventricle that it will fail - eg. - congestive heart failure. Angiotensin-Converting Enzyme Inhibitors (ACE Inhibitors) simply prevent or block the conversion of Angiotensin I to Angiotensin II. These medications do this by inhibiting the enzymatic activity of Converting Enzyme - the enzyme that converts Angiotensin I to Angiotensin II. Once Converting Enzyme is inhibited, the systemic blood pressure drops and with the lower blood pressure there is an improvement in cardiac function - i.e. - lower blood pressure, lower myocardial oxygen demand, reduced preload, decreased afterload and improved cardiac function.

ACE inhibitors reduce the production of angiotensin II and exert a biologic effect that improves symptoms, reduces hospitalizations, and prolongs survival[20]. ACE inhibitors are recommended for all patients with heart failure with reduced systolic function.

Adverse Side Effects: GI distress, dizziness, skin rashes, hypotension

Medical Uses:

To treat Hypertension and Congestive Heart Failure β-blockers (e.g., bisoprolol, carvedilol, metoprolol) protect the heart from the harmful effects of norepinephrine and epinephrine. β-blockers, when started in low doses and slowly increased, further reduce mortality when added to an ACE inhibitor. Intolerance to β -blockers is uncommon and is usually due to bradycardia or dizziness. β-blockers may worsen pulmonary function in patients with obstructive lung disease, but most patients tolerate cardio-selective β-blockers when carefully monitored.Consequently, the combination of an ACE inhibitor and a β -blocker is now the cornerstone of treatment for heart failure[21].

Diuretics (e.g., furosemide, hydrochlorothiazide) are essential in the relief of dyspnea and signs of sodium and water retention (peripheral edema or pleural effusion). They are best used in the minimum dose needed to maintain the "dry weight" in patients with symptomatic heart failure.

Aldosterone receptor blockers (e.g., eplerenone, spironolactone) work by blocking the effects of aldosterone, which can make CHF worse, and by

helping the body eliminate excess salt and water. They may help to reduce the risk of death in certain people with heart failure who have had a heart attack.

Angiotensin receptor blockers (ARBs; e.g., candesartan, valsartan) may be useful in place of ACE inhibitors when they cannot be used or sometimes in addition to ACE inhibitors.

Hydralazine and nitrates (e.g., isosorbide dinitrate, nitorglycerin patch) may be useful in place of ACE inhibitors or ARBs when they cannot be used, or sometimes in addition to other therapies when symptoms are still present.

A nonpharmacologic method that has proven useful is the implantation of a biventricular pacing device to create ventricular synchronization, in the hope of improving cardiac output.[22] additionally; implantable cardioverter defibrillators reduce the risk of death in patients who have moderate-to-severe symptomatic heart failure and a reduced ejection fraction despite maximum medical therapy[23].

Conclusion:

Chronic heart failure is a complex cardiac condition that encompasses several etiologies and comorbidities. It arises in the differential diagnosis in all adult patients who present with dyspnea and/or respiratory failure. Definitive diagnosis is established by a careful history and physical examination and supportive laboratory data. A chest radiograph is useful in excluding a pulmonary etiology (eg, pneumonia); however, a spiral computed-tomography angiogram may be required if the diagnosis of pulmonary emboli is entertained. The availability of measuring serum brain natriuretic peptide and bedside echocardiography has aided in our diagnostic precision. Therapy is primarily directed toward normalizing the underlying physiologic changes with ACE inhibitors and slow titration of blockers.

Diuretics are useful in reducing pulmonary vascular congestion, which may reduce or resolve dyspnea. Excessive therapy often reduces cardiac output or causes symptomatic hypotension, which occurs most commonly in patients with diastolic dysfunction. Treatment of the underlying etiology (silent ischemia or poorly controlled hypertension) may halt or slow the progression of the disease. Treatment of comorbidities (eg, underlying pulmonary disease, cigarette abuse, or diabetes) is essential in optimizing patient outcome and improving quality of life.

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