Congestive Heart Failure for the Primary Care Physician

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ABSTRACT

Congestive heart failure is a clinical condition in which the heart cannot send enough blood to meet the body's metabolic needs due to pathological cardiac changes. In recent decades, a progressive increase in patients affected by this disease has been observed; this could be due to the increase in population age throughout the world, as it is a disease that mainly affects elderly individuals, a higher rate of general healing as a decrease in mortality results in a more significant number of patients affected by it, as well as more effective treatments for cardiac pathologies such as primary angioplasty in acute myocardial infarction, causing a more substantial number of patients with decreased cardiac function.

INTRODUCTION

Heart failure is the presence of signs and symptoms caused by functional and/or anatomical impairment of ventricular systole and/or blood ejection, clinical signs and symptoms in which the heart cannot satisfy the body's metabolic needs.[1] Approximately 2% of the US population has congestive heart failure, most commonly systolic heart disease with reduced ejection fraction; the data show a higher prevalence in African Americans and Hispanics, which could indicate an etiological trend in certain ethnic groups; the incidence increases proportionally with age, an affection of up to 10% is observed in individuals older than 60 years.[2. 3. 4]

Cardiac output, stroke volume multiplied by heart rate, is determined by three factors: preload, afterload, and ventricular contractility.[1]

Heart failure can be reduced ejection fraction (HFrEF, HF systolic) or preserved ejection fraction (HFpEF, HF diastolic). Heart failure with reduced ejection fraction is congestive heart failure with reduced volume, which reduces the ejection fraction; left ventricular ejection fraction is ≤35–40%, heart failure with preserved ejection fraction is congestive heart failure with reduced stroke volume, normal/reduced end-diastolic volume, and preserved ejection fraction (LVEF≥ 40–50%).[1, 2]

It can also be divided into right or left heart failure, depending on which ventricle has the dysfunction. Right heart failure is caused due to a dysfunction of the right ventricle that produces congestion in peripherin the vena cava and peripheral veins, which leads to an increase in venous hydrostatic pressure and makes peripheral edema, increased pressure jugular vein, ascites, and liver affection. Left heart failure is caused by left ventricular dysfunction that results in tissue hypoperfusion and increased pulmonary capillary pressure. It can exist in both natures, right or left, which is known as biventricular (or global) heart failure [1,2]

ETIOLOGY

The leading causes of heart failure are mainly cardiometabolic diseases, including coronary arteries disease, systemic arterial hypertension, diabetes mellitus, valvular heart disease, kidney disease, and infiltrative diseases (amyloidosis). However, patients usually have more risk factors contributing to the disease's development, such as obesity, smoking, COPD, drugs, and alcoholism.[4]

Specific causes of heart failure of the systolic dysfunction type (reduced EF) can be caused by dilated cardiomyopathy (generally due to chronic alcohol consumption, idiopathic or Chagas disease), cardiac arrhythmias, and myocarditis. Moreover, specific causes for heart failure with diastolic dysfunction (preserved EF) can be restrictive
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cardiomyopathy, hypertrophic cardiomyopathy, cardiac tamponade, or constrictive pericarditis.[4, 5]

CLINICAL FEATURES

Remembering that there are different modalities of heart failure, we can highlight general clinical characteristics such as nocturia, fatigue (see below NYHA classification), tachycardia, arrhythmias, auscultation of cardiac foci shows S3/S4 in gallop, as well as alternating pulse and the presence of cachexia in some patients. [6, 7]
The classic features of heart failure with systolic dysfunction are symptoms of pulmonary congestion such as breathing difficulty, orthopnea, pulmonary edema, paroxysmal nocturnal dyspnea (characterized by acute nocturnal attacks of cough and dyspnea, caused by reabsorption of peripheral edema during the night, resulting in increased venous return), as well as cardiac asthma with asthma-like symptoms, with dyspnea, wheezing and cough, this as a result of increased pressure in the bronchial arteries, which increase airway compression and bronchospasm. On physical examination, bilateral basilar rales may be found during field auscultation, laterally displaced apical heartbeat, and evidence of peripheral hypoperfusion (coldness, pallor, and decreased pulses).[6,7,8]

AHA Classification

Table 1. AHA classification. Congestive Heart Failure.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage A</td>
<td>High risk of developing heart failure (pre-existing high blood pressure, coronary artery disease, diabetes mellitus) and no structural cardiac changes or symptoms.</td>
</tr>
<tr>
<td>Stage B</td>
<td>There is a structural damage to the heart (heart attack scars, dilation, hypertrophy) and no signs or symptoms of heart failure.</td>
</tr>
<tr>
<td>Stage C</td>
<td>Structural damage to the heart and signs or symptoms of heart failure.</td>
</tr>
<tr>
<td>Stage D</td>
<td>End-stage heart failure.</td>
</tr>
</tbody>
</table>

NYHA Classification

Table 2. NYHA classification. Congestive Heart Failure.

<table>
<thead>
<tr>
<th>Class</th>
<th>Signs and symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>No physical activity limitations and no symptoms of heart failure.</td>
</tr>
<tr>
<td>Class II</td>
<td>Slight limitations of moderate physical activity and comfortable at rest.</td>
</tr>
<tr>
<td>Class III</td>
<td>Marked limitations in physical activity (symptoms during daily activities such as dressing and walking around rooms) and comfortable only at rest.</td>
</tr>
<tr>
<td>Class IV</td>
<td>Discomfort during any form of physical activity and symptoms at rest.</td>
</tr>
</tbody>
</table>

High-output heart failure or heart failure secondary to organic dysfunction that generate a high-output state, in which cardiac output rises to meet the oxygen-metabolic demands of the tissues. It is caused due to peripheral vasodilation or arteriovenous shunt, causing a decrease in systemic vascular resistance and, thus, an increase in heart rate and stroke volume, which increases cardiac output.[10]
The conditions that lead to an increase in cardiac demand and, therefore, a state of high output is varied and can range from physiological such as exercise, pregnancy, and even fever, to secondary to an underlying pathology, such as morbid obesity, cirrhosis in advanced stages, severe anemia, systemic arteriovenous fistulas, Paget's disease, hypothyroidism, vitamin B1 deficiency, sepsis, myeloma, glomerulonephritis, carcinoid heart disease and/or other types of cancer. [10]
Symptoms are almost entirely shared with low-output heart failures, such as dyspnea, tachypnea, tachycardia, peripheral edema, fatigue, low blood pressure, and high-output symptoms such as mid-systolic murmur, S3 gallop, jugular distention with audible buzz, pulsatile tinnitus and binding peripheral pulses. The diagnosis is clinical, although an X-ray
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or echocardiogram showing cardiomegaly suggests the disease. Treatment is symptom management and hemodynamic stabilization, as well as treatment of the underlying cause.[10]

**DIAGNOSIS**

Although many times the diagnosis can be made clinically, the most common differential diagnoses, such as COPD or pneumonia, can cause diagnostic confusion, so laboratory studies must be performed (CBC, blood chemistry with creatinine, sodium, glucose, liver chemistry, CRP, lipid profile, thyroid profile), cardiac biomarkers, electrocardiogram, chest x-ray, and echocardiogram, to establish a precise diagnosis, once the diagnosis of heart failure is confirmed, the underlying cause should be investigated (consider coronary angiography, imaging chest, and advanced cardiac imaging), and identification of modifiable risks (hypertension, coronary artery disease).[1, 12]

Once the laboratory studies have been requested, the findings are variable. Anemia or infection should be sought in the blood count based on hemoglobin levels or white formula levels; elevated creatinine would indicate renal changes, being important for the cardiorenal syndrome, hyponatremia is an indicator of poor prognosis, and fasting glucose as an indicator of risk factors such as diabetes. Altered liver chemistry could indicate hepatic venous congestion. Inflammatory markers such as elevated CRP indicate an infection or acute inflammation. The patient's lipid profile should be investigated and subsequently corrected as necessary. The thyroid profile is important due to the nature of its alterations.[1, 12, 13, 14]

Cardiac biomarkers should be requested in patients with suspected heart failure, brain natriuretic peptide (BNP) or brain natriuretic peptide N-terminal prohormone (NT-proBNP), it is indicated to request to establish the diagnosis of heart failure together with echocardiography, in addition to the evaluation of the prognosis, probability of diagnosis (Table 3), and the severity of the disease (taking the difference between their admission levels and their levels before discharge). [14, 15, 16, 17, 18]

Peptide biomarkers, the diagnostic probability for heart failure.18

<table>
<thead>
<tr>
<th>Peptide</th>
<th>Values</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>BNP</td>
<td>&lt;100pg/mL</td>
<td>Unlikely</td>
</tr>
<tr>
<td>NT-proBNP</td>
<td>&lt;300pg/mL</td>
<td>Unlikely</td>
</tr>
<tr>
<td></td>
<td>&gt;500pg/mL</td>
<td>Probable</td>
</tr>
<tr>
<td></td>
<td>&gt;1000pg/mL</td>
<td>Probable</td>
</tr>
</tbody>
</table>

Measurement of BNP (or NT-proBNP) is instrumental in patients with unclear diagnoses. When combined with a physical exam and imaging, BNPg, has a high value at the diagnostic. A low BNP (or NT-proBNP) makes diagnosing acute heart failure highly unlikely in a patient with acute dyspnea. [1, 15, 16, 18]

Cardiac troponin T or I can show high levels, which suggests ischemia. However, they can also be elevated in heart failure without acute myocardial ischemia, and their values are potentially useful for risk stratification.[1, 15, 16, 17, 18, 19, 20]

Transthoracic echocardiography is indicated in all patients with suspected new-onset heart failure. It should also be performed in patients undergoing treatment with changes in the clinical characteristics of their disease. And for evaluation for device therapy. [18, 19, 20]

The classic findings that can be observed range from the characteristics of ventricular dysfunction when evaluated by LVEF (Table 4). [21]

<table>
<thead>
<tr>
<th>Classification by Ejection Fraction. [21]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Failure</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>HFpFE</td>
</tr>
<tr>
<td>HFrFE</td>
</tr>
</tbody>
</table>

As well as pericardial and pleural effusion, evidence of complications such as cardiac asynchrony, functional mitral regurgitation, or left atrial enlargement, and underlying causes such as local coronary artery wall motion abnormalities left ventricular hypertrophy due to systemic hypertension, or abnormalities of flow through the heart valves.[21, 22, 23]

Chest X-ray is indicated in case of new-onset heart failure or due to high suspicion, changes in the cardiac silhouette can be observed, from a cardiothoracic width ratio >0.5, boot-shaped heart seen in PA and left Ventricular enlargement, as well as signs of pericardial effusion. Findings of pulmonary congestion are not uncommon to observe, and valvular or pericardial cardiac calcifications are often recurrent.[1]

Electrocardiogram abnormalities are common but nonspecific, ranging from left ventricular hypertrophy, left axis deviation, ST-T, or P wave abnormalities to prolonged QT interval or complete or left bundle branch block incomplete can be observed. Recurrent changes due to pre-existing cardiac disease are ischemic changes from

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myocardial infarction, arrhythmias, or pericardial effusion.[24, 25]
The diagnostic tests for heart failure have already been exposed. However, without limiting ourselves to the first instance tests, additional studies can be helpful when there is diagnostic uncertainty and as a diagnosis of underlying causes. There are advanced cardiac images, such as cardiac magnetic resonance imaging, which is the gold standard for evaluating ventricular volume, mass, and ejection fraction. Indications include diagnostic not being clear after echocardiography, investigating congenital heart disease in adults, or determining myocardial scar burden—alternatively, PET myocardial perfusion imaging as an alternative non-invasive study for suspected coronary artery disease. Right heart catheterization assesses proper heart function and pulmonary vascular resistance in patients considered for transplant mechanical circulatory advanced support. Blood pressure monitoring and electrocardiographic monitoring in patients with suspected systemic arterial hypertension or Holter monitoring in suspected paroxysmal atrial fibrillation or other arrhythmias.[1]

TREATMENT
The initial treatment for uncomplicated congestive heart failure begins with modifying the patient's lifestyle; these changes reduce the risk factors associated with the progression of heart failure and other cardiometabolic comorbidities such as diabetes mellitus and hypertension. These lifestyle changes will be specific to each case. However, in general terms, we can mention that aerobic exercise, smoking cessation, alcoholism, drug use, and weight loss are essential to reduce the progression of the disease. Should consider states of immunosuppression in patients with comorbid conditions, requiring immunization with pneumococcal and seasonal influenza vaccines, adapting to each case depending on national epidemiological recommendations. [1, 15, 21, 26]

Initial pharmacological treatment of heart failure.[8, 15]

It is important to make the patient understand the pathophysiological bases of their disease, which makes the treatment efficacy and, therefore, their quality of life substantially improve; salt restriction ranges from <3g/day to <1.5g/day. Depending on the stage where the patient is, foods rich in potassium should be avoided during the administration of aldosterone antagonists, and fluids should be limited to 1.5-2 L per day in patients with stage D, who present with edema and/or hyponatremia. The patient must control and recognize the symptoms from daily weight control; if there is an increase of >2kg in less than three days, the patient must return to the doctor to evaluate the use of diuretics. The patient must be able to recognize symptoms of worsening heart failure, such as increased dyspnea at lower exertion than before, and must recognize new symptoms suggestive of medication side effects. It is recommended to carry a copy of the medical records and avoid destinations with limited medical attention. [1, 26, 27]

Furthermore, in the case of significant atherosclerosis, revascularization should be considered. In the case of anemia, specific treatment for each type of anemia should be given to all patients with NYHA class II and III symptoms. [1, 27, 28, 29, 30]

Most antiarrhythmic agents should be avoided, as well as potassium channel blockers, except amiodipine (simultaneous use of calcium channel blockers with beta-blockers can cause complete heart block), thiazolidinediones, and anesthetics. Inhaled antidepressants should also be avoided, and due to the increased incidence of depression in patients of all age groups in recent years, careful selection of antidepressants should be made. [1, 15, 26, 29, 31]

Pharmacological treatment for heart failure is based on the stage of the disease in which the patient is present (Table 5), adding additional therapies to the treatment as symptoms worsen. The recommendations call for gradual initiation of all medications, starting with the lowest recommended dose and slowly increasing the dose, with each visit, up to the target dose. [1, 8, 15, 26, 29, 32, 33]

Table 5. ACEs; angiotensin-converting enzyme inhibitors. BRA; Angiotsensin receptor blockers. B-Blockers; Beta receptor blockers.

<table>
<thead>
<tr>
<th>Stage A</th>
<th>Treatment of cardiovascular and comorbid risk factors and medications for heart failure are not routinely indicated.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharmacological Class</td>
<td>examples</td>
</tr>
<tr>
<td>Stage B</td>
<td></td>
</tr>
<tr>
<td>ACEIs</td>
<td>Enalapril</td>
</tr>
<tr>
<td>Captopril</td>
<td>Renal function, Potassium 2 weeks after initiation or dose change</td>
</tr>
<tr>
<td>Lisinopril</td>
<td></td>
</tr>
<tr>
<td>BRA</td>
<td>Losartan</td>
</tr>
<tr>
<td>Valsartan</td>
<td>Renal function, Potassium 2 weeks after initiation or dose change</td>
</tr>
</tbody>
</table>

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Stage C

| aldosterone antagonist | Spironolactone | Eplerenone | All patients with HFrEF with NYHA II-IV symptoms and an LVEF <35%. Evaluate its use in patients with HFpEF. | Regular monitoring of potassium, the tendency to hyperkalemia. |
| diuretics | Handle Furosemide Bumetanide Thiazides Hydrochlorothiazide | For all patients with fluid retention and volume overload, thiazides, as a synergistic effect, do not use as ba as treatment. | Evaluate the dose according to weight, volume, and hemodynamic stability with periodic review of serum electrolytes. |

Isosorbide and Hydralazine

| isosorbide | Hydralazine | Patients who do not tolerate ACE inhibitors or ACEs used in Afro-descendants. | Separate dosage of both drugs and monitor volume depletion and hypotension. |

Stage D

Additional measures to the treatment above: For patients with Stage D Heart Failure, invasive intervention or a palliative care approach should be considered. Continuous intravenous inotropic support should be considered a bridge before heart transplantation or mechanical circulatory support.

Table 6. Drugs for the treatment of chronic heart failure with decreased Ejection Fraction. Initial dose and maximum dose. [8]

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Dose</th>
<th>Maximum dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>diuretics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>20-40 mg every 24 hours every 12 hours</td>
<td>400mg/day</td>
</tr>
<tr>
<td>Bumetanide</td>
<td>0.5-1.0 mg every 24 hours/12h</td>
<td>10mg/day</td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>25mg every 24h</td>
<td>100mg/day</td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 mg every 12 hours</td>
<td>50mg q8h</td>
</tr>
<tr>
<td>Captopril</td>
<td>6.25 mg every 8 hours</td>
<td>10-20mg every 12h</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>2.5-5.0 mg every 24 hours</td>
<td>20-40mg every 24h</td>
</tr>
<tr>
<td>Angiotensin receptor antagonists</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Losartan</td>
<td>25-50mg every 24h</td>
<td>150mg every 24h</td>
</tr>
<tr>
<td>Valsartan</td>
<td>40 mg every 12 hours</td>
<td>160mg q12h</td>
</tr>
<tr>
<td>B receptor antagonists</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>1.25mg every 24h</td>
<td>10mg every 24h</td>
</tr>
<tr>
<td>Metoprolol Succinate CR</td>
<td>12.5-25mg every 24h</td>
<td>200mg every 24h</td>
</tr>
<tr>
<td>Aldosterone antagonists</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spironolactone</td>
<td>12.5-25mg every 24h</td>
<td>25-50mg every 24h</td>
</tr>
<tr>
<td>Eplerenone</td>
<td>25mg every 24h</td>
<td>50mg every 24h</td>
</tr>
<tr>
<td>Additional Drugs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combination of hydralazine and isosorbide dinitrate</td>
<td>10-25mg/10mg q8h</td>
<td>75mg/40mg q8h</td>
</tr>
</tbody>
</table>

The use of Digoxin should be considered in patients with HFrEF with persistent symptoms and refractory to first-line treatment; renal function should be closely monitored. [1, 8, 15, 26, 29, 30, 31]

To consider the use of cyclic nucleotide-modulated hyperpolarization-activated channel blockers (ivabradine), all of the following must be present:

- NYHA class II-III
- HFrEF <35%
- Sinus rhythm with HR >70bpm at rest with the maximum tolerated dose of B-Blockers.

Patients who present arrhythmias such as ventricular tachycardia or fibrillation, in combination with heart failure, can cause symptomatic worsening and increase the risk of sudden cardiac death; these patients are candidates for
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Invasive interventions, devices with a pacemaker, and/or defibrillator functions. [34, 35]
The implantable cardioversion defibrillator works by administering an electrical shock, restoring sinus rhythm if an arrhythmia such as ventricular fibrillation or ventricular tachycardia is detected. [34, 35, 36, 37, 38]
The indications for its placement are for patients who previously presented sustained ventricular tachycardia or cardiac arrest secondary to ventricular fibrillation or tachycardia, as well as patients with HFrEF with expected survival > one year if they receive medical treatment for 3-6 months and still comply with any of the following criteria:

- Stage B with ischemic cardiomyopathy if LVEF is >30%
- Stage C with dilated cardiomyopathy or ischemic heart disease with LVEF >35% and NYHA class II-III symptoms [34, 35, 36, 37, 38]

In patients with end-stage heart failure, heart transplantation is the only cure. Unfortunately, most patients are not candidates. Patients accepted for transplantation generally require bridge measures, such as inotropic and/or mechanical circulatory support. Any patient with end-stage heart failure who is not a transplant candidate should be referred for palliative care. [1, 8, 37, 38]

COMPLICATIONS

 Decompensated heart failure is the worsening of heart failure symptoms. It is the most common cause of hospitalization for heart failure complications and one of the most common causes of hospitalization in older adults. Multiple causes can trigger pre-existing acute cardiac decompensation, but it can also occur in patients without a history of a heart condition. The diagnosis is based on typical clinical features as well as imaging findings. Management can be complicated because multiple comorbidities often accompany it. Most patients require treatment with diuretics, vasodilators, respiratory support, medications for underlying heart failure, and careful fluid management. [39, 40, 41]

Other common complications are:

- Cardiorenal syndrome
- Cardiac arrhythmias
- Cardiogenic shock
- Cerebrovascular accident (Usually due to thromboembolism)
- Cardiac cirrhosis (right CHF)
- Venous stasis

CARDIORENAL SYNDROME

It is a complex syndrome in which renal function is progressively diminished as a result of significant cardiac dysfunction; it occurs in 30% of patients with acute decompensated heart failure; its pathophysiology depends on which side of the heart the dysfunction is, in the case of systolic dysfunction, cardiac output is decreased, causing renal hypoperfusion, which would cause prerenal renal failure; in the case of diastolic dysfunction, it causes systemic venous and renal venous congestion, which decreases the gradient of transglomerular pressure, a decrease in glomerular filtration rate and consequently a decrease in renal function. The AHA has classified it into five types: Type I: Acute cardiorenal syndrome is when the heart failure leads to acute kidney injury.

- Type II: Chronic cardiorenal syndrome. Chronic heart failure leads to chronic kidney disease.
- Type III: Acute renocardiac syndrome. Acute kidney injury leads to acute heart failure.
- Type IV: Chronic renocardiac syndrome. Chronic kidney disease leads to chronic heart failure.
- Type V: Secondary SRC. Systemic disease leads to kidney and heart failure.

Suspicion of cardiorenal syndrome is classically made when a patient with heart failure presents with decreased glomerular filtration rate and increased creatinine that cannot be explained by underlying kidney disease. The treatment is to treat heart failure and nephroprotective measures. The prognosis depends on laboratory levels; in the case of creatinine >3mg/dl, it is associated with a poor prognosis.[1, 15, 39, 40, 41]

PROGNOSIS

The prognosis in patients with heart failure is poor unless the cause is corrected. However, it depends mainly on the patient, the type and severity of heart disease, medication regimens, and lifestyle changes. The of patients with preserved EF is better than those with decreased EF, and worse prognostic factors are elevated BNP, hyponatremia, systolic BP <120mmHg, diabetes, anemia, weight loss or low weight, S3, use implantable cardioverter-defibrillator and frequent hospitalizations for heart failure. [1, 42]

1-year survival according to NYHA stage:

- Class I: 95%
- Class II: 85%
- Class III: 85%
- Class IV: 35%

CONCLUSION

Heart Failure is an important public health problem, so the therapeutic approach must be multidisciplinary to impact all phases of the syndrome positively; that is why currently, the world trend is toward creating Heart Failure Clinics that are defined as specialized services for timely diagnosis and treatment of HF but also develop advanced research and educational programs to treat patients comprehensively.

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