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Management of Heart Failure: Part 1, Acute

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LEARNING OBJECTIVES
After completing this continuing education program, the pharmacist should be able to:

1. Identify categories of heart failure (HF).
2. Identify causes of HF.
3. Identify manifestations of HF.
4. Describe treatment options for acute HF.

ABSTRACT: As a large percentage of our population will soon be reaching 65 years of age, heart failure (HF) is becoming the new epidemic of our time. Organizations and associations have coordinated their resources to produce guidelines and treatment options to better assist health care professionals.

HF has been classified into several different categories: right-sided versus left-sided, forward versus backward, systolic versus diastolic, high-output versus low-output, and acute versus chronic. This continuing education series is divided into 2 sections, Part 1 and Part 2. In Part 1, management of "acute" HF will be addressed and Part 2 will be devoted to treatment of "chronic" systolic HF.

Most patients experiencing acute HF require hospital admission, especially if their symptoms are severe. The short-term goals of therapy include relief of symptoms, perfusion of organs, and stabilization of the patient. The treatment of acute HF depends on the presentation of the patient. In all cases, the underlying cause needs to be addressed and corrected, if possible. In general, if the pulmonary capillary wedge pressure (PCWP) is elevated, a diuretic may be the first-line agent of choice, followed by a vasodilator, if necessary. If the cardiac index (CI) is decreased, a positive inotropic agent may be the drug of choice. Other medications used in the treatment of acute HF will also be discussed.

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MANAGEMENT OF HEART FAILURE: PART 1, ACUTE

Introduction

As a large percentage of our population will soon be reaching 65 years of age, heart failure (HF) is becoming the new epidemic of our time. Organizations and associations have coordinated their resources to produce guidelines and treatment options to better assist health care professionals. In order to use treatments effectively, we first need to understand the pathophysiology of HF.

Diagnosis of HF usually occurs at the time of symptom onset. Prior to that time, the heart may have had a long pathologic period. HF may begin with a defective myocardium or fluid retention. Initially, the body may compensate by neurohormonal activation and, later in the process, patients may develop symptoms associated with cardio-vascular decompensation. Cardiac remodeling can also occur because of an increased pressure or volume overload.

Approximately 80%-90% of patients with HF suffer from symptoms attributable to an impairment of left ventricular function.[1] Medications used in treatment of HF may decrease systemic vascular resistance, decrease cardiac remodeling, or decrease fluid retention.

HF has been classified into several different categories: right-sided versus left-sided, forward versus backward, systolic versus diastolic, high-output versus low-output, and acute versus chronic (Table 1). Right-sided or left-sided HF is associated with the accumulation of fluid behind right or left ventricles. Manifestations include pulmonary edema for left-sided and congestive hepatomegaly for right-sided HF.[2] In forward HF, the heart is unable to expel the required amount of blood, while backward HF is the inability to fill or discharge properly.[2] Systolic HF is defined as the inability to expel the required amount of blood during ventricular contraction while diastolic HF is the inability to relax and allow proper ventricular filling.[2] High-output HF may be seen in patients with hyperthyroidism, anemia, pregnancy, arteriovenous fistulas, beriberi, and Paget's disease. Low-output HF may occur secondary to ischemic heart disease, hypertension, dilated cardiomyopathy, and valvular and pericardial disease.[2] Acute HF is the sudden development of HF while chronic HF is a progressive development of structural disease and symptoms.[2]

Types of HF	
Differences	**Terminology**
Location of Fluid Accumulation	Right-Sided
	Left-Sided
To fill or Discharge	Forward
	Backward
Contractility or Relaxation	Systolic
	Diastolic
Output	High-Output
	Low-Output
Onset and Duration	Acute
	Chronic

Table 1

There are various causes of HF. Precipitating causes include infection; anemia; thyrotoxicosis; pregnancy; arrhythmias; rheumatic, viral, and other forms of myocarditis; infective endocarditis; physical, dietary, fluid, environmental, and emotional excesses; systemic hypertension; myocardial infarction; and pulmonary embolism.[5]

Other causes of HF include coronary atherosclerosis, valvular and/or congenital heart disease, acute hypertensive crisis, rupture of an aortic valve cusp, impaired ventricular filling due to tricuspid and/or mitral stenosis, constrictive pericarditis without myocardial involvement, endocardial fibrosis, and some forms of hypertrophic cardiomyopathy.[5]

Many patients present with an exacerbation of previously controlled HF. Causes of decompensation may include the above causes, as well as noncompliance with medications, dietary sodium, or liquids;[6] new medications, such as antiarrhythmics, β-blockers, non-steroidal anti-inflammatory agents, verapamil, diltiazem;[6] renal dysfunction, possibly attributable to excessive diuretic use;[6] a new onset or worsening of mitral or tricuspid regurgitation;[6] pulmonary hypertension;[6] overreduction of preload, possibly caused by diuretics

and angiotensin converting enzyme (ACE) inhibitors; systemic hypertension; drug induced cardiotoxicity (ethanol, anthracyclines, trastuzumab, cocaine); hypocalcemia; and hypoxemia.

Patients presenting with HF may have many different non-specific signs and symptoms. These include dyspnea (rapid, shallow breathing), orthopnea and coughing at night, paroxysmal nocturnal dyspnea (shortness of breath and coughing with no relief sitting up), acute pulmonary edema (rales; expectoration of blood-tinged fluid; extreme shortness of breath, which can be fatal if not treated immediately), Cheyne-Stokes respiration (apneic phase→hyperventilation→hypocapnia→ apnea), fatigue, and weakness owing to decreased skeletal muscle perfusion, anorexia and nausea because of congested liver and portal venous system, and altered mental status because of decreased cerebral perfusion.[5] Physical findings can include decreased pulse pressure leading to decreased stroke volume; increased diastolic arterial pressure caused by vasoconstriction, severe hypotension, cyanosis of lips and nail beds; sinus tachycardia; increased systemic venous pressure, which appears as jugular venous distention and positive abdominojugular reflux; pulmonary rales; dullness to percussion over the lung bases because of increased pulmonary venous and capillary pressures; pulmonary edema causing expiratory wheezing; cardiac edema (especially in the ankles of ambulatory patients and the sacral region of bed-bound patients; pitting edema of arms and face is rare and occurs late in the course of HF); hydrothorax; ascites; congestive hepatomegaly; jaundice; cardiac cachexia; cold, pale, diaphoretic extremities; oliguria; albuminuria; hyponatremic urine; urine with a high specific gravity; prerenal azotemia; impotency; and depression.[5]

Decompensated HF plus fluid overload and/or elevated central venous pressures can lead to jugular venous distention, hepatomegaly, positive hepatojugular reflux, ascites, edema, and pulmonary vascular congestion.[7] Increased left ventricular preload leads to increased pulmonary capillary wedge pressure and pulmonary congestion (rales, cough, dyspnea). Increased right ventricular preload leads to jugular venous distention, hepatomegaly, splenomegaly, positive hepatojugular reflux, ascites, and edema.

The Framingham guidelines have been used in the past for diagnosing congestive HF. These guidelines use the manifestations of HF and divide them into 3 categories: major criteria, minor criteria, and major or minor criteria. The major criteria include a positive hepatojugular reflux, neck vein distention, rales, S_3 gallop, cardiomegaly, acute pulmonary edema, paroxysmal nocturnal dyspnea, and an increased venous pressure (>16 cm H_2O). Minor criteria include tachycardia (≥120 bpm), hepatomegaly, night cough, extremity edema, pleural effusion, dyspnea on exertion. and a one-third reduction of vital capacity from normal. Major or minor criteria is considered to be a weight loss ≥4.5 kg over 5 days' treatment. To establish a clinical diagnosis of congestive HF by these criteria, at least 1 major and 2 minor criteria are required.[2]

HF is the most common diagnosis for those over 65 years of age. Patients with hypertension are 2 times more likely to develop HF, and patients who have had an acute myocardial infarction are 5 times more likely.[3] There is a 25% greater HF occurrence in the black population than in the white population.[3] Today, there are almost 5 million Americans who live with chronic HF. This number represents men and women equally.[3] With each passing year, the number is expected to rise because of a longer HF life expectancy and an increase in the population of patients over 65 years old.[3]

Symptomatic HF has a poor prognosis. Studies have found several signs of HF that can help determine a more accurate prognosis. These prognostic values include heart rate variability, exercise stroke work index (SWI), left ventricular ejection fraction, plasma atrial natriuretic peptide, plasma norepinephrine, oxygen consumption (Vo_2), New York Heart Association (NYHA) classification, and B-type natriuretic peptide (BNP). One of the most useful HF prognostic information is functional capacity and clinical status. A decreased heart rate variability (HRV) is a sign of the autonomic system's inability to control the heart rate. In a study using a Poincare plot and 24-hour Holter recording, the HRV helped increase the prognostic validity of the model. HRV appears useful in classifying high-risk patients for cardiac death.[4] Exercise testing revealed that a peak SWI is a powerful prognostic indicator. The testing revealed that a peak exercise SWI of less than or equal to 30 g x m/m^2 had a 54% chance at a 2-year survival. Patients with an SWI greater than 30 g x m/m^2 had a 91% 2-year survival rate.[5]

Neurohormonal values and exercise testing show that atrial natiuretic peptides, plasma norepinephrine, and the ejection fraction are very helpful in determining long-term prognosis. A left ventricular ejection fraction of less than 45% indicated a poor prognosis.[6] A study using patients 70 years and older with HF found that the strongest prognostic factors used in this population were NYHA classification and peak Vo_2. It showed that a Vo_2 less than 14 mL/min/kg and a CO_2 consumption greater than 34.5 was related to a poor prognosis. The study also stated that the Vo_2 is a good indicator for heart transplantation.[7]

Although management of acute HF is not addressed in the new guidelines,[3] The American College of Cardiology/American Heart Association (ACC/AHA) Task Force has recently published new consensus guidelines for the treatment of chronic HF.[3] Management of acute HF had been previously discussed in published guidelines.[4]

Neurohormonal changes

Norepinephrine (NE)

Plasma NE may be greatly increased because of an increase in action of the adrenergic nervous system.[7] This elevation increases contractility[7] and causes vasoconstriction, tachycardia, and potentially arrhythmias in acute HF.[8] A long-term effect of NE is ventricular hypertrophy.[8]

The Renin-Angiotensin-Aldosterone System (RAAS)

The RAAS is stimulated in the kidney in response to a decreased cardiac output.[7] This causes an increase in angiotensin II, resulting in vasoconstriction,[7] which produces an increased systemic vascular resistance.[8] Angiotensin II also increases aldosterone,[8] which leads to sodium and fluid retention.[7,8] A long-term effect of angiotensin II is ventricular hypertrophy and remodeling.[8]

Endothelin-1

Endothelin-1 is a potent vasoconstrictor[8] and is increased in HF.[7,8] This vasoconstriction can cause an increased preload and afterload and decreased renal perfusion and consequent sodium retention.[8] Endothelin-1 can also stimulate angiotensin II, aldosterone, and epinephrine.[8] Its long-term effect is ventricular hypertrophy.[8] Use of endothelin receptor blockers may be a future therapy for the management of HF.[8]

Tumor Necrosis Factor-α (TNF-α)

TNF-α is an inflammatory cytokine that is increased in HF.[7,8] It causes negative inotropy,[8] systolic dysfunction, myocarditis, ventricular dilatation, HF, and shortened survival.[7]

Natriuretic Peptides

A-type natriuretic peptide (ANP) is released from the atria in response to activation of stretch receptors.[7] B-type natriuretic peptide (BNP) is released mainly from the ventricles and, to some extent, from the atria in response to increased volume and pressure in the left ventricle.[7] These hormones may not be sufficiently elevated in patients with decompensated HF to counter the effects of the other neurohormones.[7, 8] BNP is produced by the ventricles and released when a patient is experiencing symptomatic HF. It is released in response to an increase in ventricular volume and pressure. BNP binds to smooth muscle and endothelial cell receptors. This leads to an increase of the second messenger cGMP intracellularly, causing smooth muscle cell relaxation and vasodilation of veins and arteries. It also antagonizes the effects of the sympathetic nervous system, the RAAS, and the endothelin systems.

Diagnosis

The standard diagnostic tools for acute HF have been the physical exam, chest x-ray, echocardiogram, and measurement of the pulmonary capillary wedge pressure (PCWP) via a Swan-Ganz catheter.[9] Until recently, there was no definitive lab value to differentiate an acute episode of HF versus other causes of dyspnea. A new tool has been developed that may be beneficial in the differential diagnosis: BNP levels, which correlate with the severity of HF.[10] BNP levels, however, are not able to differentiate between systolic and diastolic HF.[3]

A study done by Dao, et al.,[11] showed that patients who were diagnosed with CHF as a cause of their dyspnea in the urgent care setting had mean BNP levels of 1076 +/- 138 pg/ml and patients who were not diagnosed with CHF had mean BNP levels of 38 +/- 4 pg/ml. In this study, the investigators determined that the BNP level was most accurate at a level of 80 pg/ml,[11] meaning that patients with a level under 80 pg/ml most likely do not have CHF, and those with a level over 80 pg/ml most likely

have some type of left ventricular dysfunction.

Treatment

Most patients experiencing acute HF require hospital admission, especially if their symptoms are severe.[4] The short-term goals of therapy include relief of symptoms, adequate perfusion of organs, and cardiovascular stabilization of the patient.[2]

The treatment of acute HF depends on the presentation of the patient. In all cases, the underlying cause needs to be addressed and corrected, if possible. In general, if the PCWP is elevated, a diuretic may be the first-line agent of choice, followed by a vasodilator, if necessary. If the cardiac index (CI) is decreased, a positive inotropic agent may be the drug of choice. Mild to moderate acute HF may be able to be managed in an outpatient setting.

Cardiogenic Shock

If a patient is experiencing cardiogenic shock and has no volume overload, the treatment of choice may include infusion of normal saline and inotropic agents. If the patient is fluid overloaded or has been repleted with fluid, dobutamine or low-dose dopamine may be the optimal treatment.[4] If a patient does not respond to these measures, then an intra-aortic balloon pump may be placed. The balloon is inserted into the aorta and inflates during diastole.[12] This causes an increased aortic diastolic pressure, allowing for more efficient coronary artery perfusion.[12] The balloon deflates during systole.[12] This dramatic decrease in pressure creates less impedance for the heart to pump blood against, thus increasing stroke volume, therefore increasing the CI.[8]

Acute Decompensation

If a patient presents with acute decompensation of previously controlled HF, the first line of treatment is to treat the reversible causes of decompensation. Once this has been accomplished, the patient should be treated as a new onset of acute HF.

Acute Cardiogenic Pulmonary Edema

Oxygen, sublingual or intravenous nitroglycerin, intravenous sodium nitroprusside, intravenous diuretics, intravenous morphine, intra-aortic balloon pump, and intravenous inotropic agents, if indicated, are the treatments of choice in patients presenting in this manner.[4]

Diuretics

Loop diuretics are the most commonly used diuretics in the treatment of HF because an increase in the loop diuretic dose causes an increased effect, regardless of the magnitude of the dose being used. Other diuretics have a ceiling effect; once a certain dose is reached, a higher dose will not cause further diuresis. Loop diuretics are also beneficial in patients who have a CrCl of > 10 ml/min, whereas most other diuretics may no longer be effective when the CrCl is < 30 ml/min. Diuretics decrease the venous return, which then decreases pulmonary congestion and CI. Patients must be monitored closely because insufficient CI can worsen the HF. Inadequate CI can cause reflex increases in sympathetic nervous system activation, renin release, norepinephrine, arteriolar and coronary constriction, tachycardia, PCWP, and myocardial oxygen consumption.

Diuretic resistance may occur, especially in HF patients. Options to overcome this

effect include switching from oral to intravenous formulations, increasing the dose, administering as a continuous infusion, adding a diuretic from a different class, and adding dopamine as a last option. Metolazone and hydrochlorothiazide are most frequently added to furosemide therapy. This may potentially cause a synergistic diuretic effect because the diuretics work in different areas of the nephron. The combination of thiazide/loop diuretic treatment may be effective even in patients with advanced renal failure.[13]

Vasodilators

Vasodilators are effective by decreasing resistance in the vasculature, leading to an increase in stroke volume and CI and a decrease in PCWP. Vasodilators are especially beneficial in patients with severe mitral valve regurgitation.[14]

Nitroglycerin

Nitroglycerin is an arterial and venous dilator. It has a short half-life, a high incidence of headache, and can cause hypotension.

Sodium Nitroprusside

Sodium nitroprusside is also an arterial and venous dilator. Its side effects are similar to nitroglycerin. In addition, one of its metabolites is thiocyanate, so patients must be monitored for toxic thiocyanate levels.

Nesiritide

Nesiritide is a recombinant form of the human B-type natriuretic peptide. It is FDA approved for "intravenous treatment of patients with acutely decompensated congestive HF who have dyspnea at rest or with minimal activity."[15] It is used in both systolic and diastolic HF. The effects of nesiritide include decrease in PCWP and systemic arterial blood pressure (BP); increase in natriuresis, diuresis, stroke volume, and CI; and blockade of aldosterone, NE, and endothelin-1.

Contraindications to its use include systolic BP < 90 mmHg, low cardiac filling pressures, and/or cardiogenic shock.[15]

In comparison with dobutamine, nesiritide may be less arrhythmogenic,[15, 17] and has an increased incidence of nausea and symptomatic hypotension.[15] In comparison with standard therapy (dobutamine, milrinone, nitroglycerin, and sodium nitroprusside), nesiritide has been shown to have similar improvements in global clinical status, dyspnea, and fatigue.[18] There is also a potential for a decreased need for diuretics and increased bradycardia.[18] Compared with nitroglycerin, nesiritide has decreased PCWP at 3 hours, a greater decrease in dyspnea at rest at 3 hours, a decreased incidence of headache, a quicker onset of action, a longer duration of action, and possibly no need for a Swan-Ganz catheter for dose titration.[15] In one study, PCWP at 3 hours was significantly better with nesiritide than with either nitroglycerin or placebo.

Inotropic Agents

Inotropic agents have shown some effectiveness in the treatment of patients with severe HF, patients who are refractory to conventional treatment, and those awaiting cardiac transplantation.[2] The agents work by increasing calcium release from the sarcoplasmic reticulum, thereby increasing contractility. Except for digoxin, it is not recommended to use positive inotropic agents in the long-

term treatment of HF, as they can increase mortality.[2]

Digoxin
Digoxin is not used in the treatment of acutely decompensated patients unless they have atrial fibrillation.[19] This may be attributable to its weak positive inotropic effects and long half-life.[8]

Dobutamine
Dobutamine is a catecholamine that works on β- and α-adrenergic receptors. It is most selective for β_1-receptors, then β_2-, and least selective for α_1-receptors. When the receptors are stimulated, adenylate cyclase is activated, which leads to an increase in cAMP. Dobutamine increases CI because of its β_1 effects and decreases systemic vascular resistance because of its β_2 effects. The α_1-mediated vasoconstriction is usually offset and overcome by the β_2-mediated vasodilation.[8] When dobutamine is administered concomitantly with β-blockers, it may be less effective or ineffective. Tolerance may occur owing to down-regulation of β-receptors, so increasing the dose may be required to obtain the same response. Some patients may not respond at all to dobutamine. Adverse effects include increased HR, increased BP, or arrhythmias.

Dopamine
Dopamine is a catecholamine that works on β_1-, β_2-, α_1-, and dopamine (D_1) receptors. At low doses (~ 0.5 to 3 mcg/kg/min), it primarily affects D_1-receptors, causing increased renal blood flow. At moderate doses (~ 3 to 10 mcg/kg/min), it affects β_1-receptors, increasing the CI and HR. At high doses (~ >10 mcg/kg/min), it mainly affects α_1-receptors, leading to vasoconstriction. These dose ranges are averages and are not patient specific, so different patients may respond differently to the same dose or may not respond at all. Because the effects may not be predictable for every patient, dopamine is not the best agent to use in the management of acute HF unless the patient is in cardiogenic shock. At high doses, dopamine may decrease coronary blood flow and increase myocardial oxygen demand and arrhythmias.[8]

Milrinone
Milrinone is a phosphodiesterase (PDE) inhibitor. It inhibits PDE, which is an enzyme that catabolizes cAMP. PDE inhibition causes positive inotropy in the heart and vasodilation in vascular smooth muscle cells.[2] It is an alternative to dobutamine, and can be used concurrently with β-blockers.[2] Like β-agonists, it can also increase HR, BP, or arrhythmias.

Inamrinone
Inamrinone is also a PDE inhibitor, and works similarly to milrinone. Milrinone may have less toxicity (e.g., GI and fever), hypotension, and thrombocytopenia than inamrinone. Milrinone has a shorter half-life. For these reasons, inamrinone is rarely used over milrinone.

When choosing between dobutamine and milrinone, several factors should be considered:

- Patients with severe renal dysfunction, severe hepatic dysfunction, and/or thrombocytopenia may be better candidates for dobutamine instead of milrinone or inamrinone.

- Patients who have concomitant HF and systemic or pulmonary hypertension may respond better to milrinone.
- Patients with bisulfite allergies should not receive dobutamine because it is formulated with bisulfite.
- Milrinone may be a better choice for patients who will be on inotropic therapy for an extended amount of time (e.g., patients waiting for a heart transplant) because less tolerance may occur.[2]
- As PDE inhibitors do not work on adrenergic receptors, milrinone may be more effective than dobutamine in patients concurrently receiving β-blockers.[21]
- Currently, milrinone is not available in a generic form, so its higher cost should also be considered.

The concurrent use of dobutamine and inamrinone has been studied.[22-24] Gage, et al.[22] showed that inamrinone administered in addition to dobutamine resulted in a greater increase in CI than dobutamine alone and a greater decrease in left-ventricular end-diastolic pressure than inamrinone alone. Guimond, et al.[23] showed that a patient receiving high doses of dobutamine was having life-threatening arrhythmias. The addition of inamrinone allowed a reduction in dobutamine dose and an increase in the CI.[23] The combination is a last-resort therapy and should be done with caution.[24]

β-blockers

Acutely decompensated patients should not be started on a β-blocker until they are stable.[20, 25] Patients who are admitted to the hospital on a β-blocker should continue on it, unless there is a contraindication for its use.[20, 25]

Discharge

It is important to make sure that patients are placed on optimal long-term treatment for their HF upon discharge. This treatment has been discussed in the 2001 ACC/AHA guidelines,[3] and will be discussed in Part 2. Additionally, patients should be educated regarding their diet, medication, and exercise.[14]

Measurement of BNP levels may be beneficial. In a study done by Cheng V, et al., NYHA class III and IV patients admitted for either decompensated HF or new-onset HF had a higher incidence of death during their stay or readmission within 30 days of discharge.[26]

The patients who had better outcomes had BNP levels of 690±103 pg/ml and a decrease in their NYHA classification upon discharge. Those who had poor outcomes had BNP levels of 1801± 273 pg/ml and a decrease in their NYHA classifications. Those who died had increasing levels of BNP and insignificant changes in their symptoms.[26]

Transplant

When the heart is no longer able to function despite optimal medications, cardiac transplantation may be the last option.[5] Each institution may develop its own inclusion and exclusion eligibility criteria.[5] IV inotropic infusion may be beneficial while a patient is awaiting transplant. Mechanical ventricular assist devices,[25] intra-aortic balloon pump, and dialysis may be necessary while patients are waiting for transplantation.[6]

Conclusion

Acute HF is the cause of many hospital admissions. It is important that health care professionals understand the optimal treatment of patients admitted for acute HF so that these patients have a reduced hospital stay and readmission rate. These objectives may be met by optimizing long-term HF management and with measuring BNP levels. The treatment regimen used is dependent on the patient's hemodynamics and symptoms. Specialized HF clinics are ideal for the long-term management of HF patients.

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