

clinical contributions

Evidence-Based Clinical Vignettes
from the Care Management Institute:

Heart Failure

Introduction

Heart failure has emerged as a major public health challenge. The condition is common, affecting from 1.5% of non-Hispanic white women to 3.5% of non-Hispanic African Americans in the United States¹ as well as 6-10% of the US population aged 65 years or older.² Approximately 50% of heart failure patients are dead within five years after diagnosis.¹ Heart failure is the most common cause of hospitalization among Medicare beneficiaries and is the single costliest Medicare diagnosis.³

Heart failure can be caused by a variety of cardiac conditions, most commonly coronary artery disease. In 30% to 40% of patients with heart failure, ventricular contractility is normal as measured by the left ventricular ejection fraction (LVEF).⁴ As many as half of elderly heart failure patients have a normal LVEF.⁵

During the past decade, several forms of therapy have been shown to prolong survival, prevent hospitalization, and improve quality of life for patients with heart failure and reduced LVEF.⁶ Because studies have shown that these forms of therapy are often underused,⁷⁻¹⁰ programs have been developed to ensure optimal care for patients with heart failure. Optimal treatment of heart failure requires correct diagnosis, identification of potentially reversible causes, appropriate use of medication, and patient education on self-care.

In this article, a vignette based on a single case presents an overview of the recently updated Care Management Institute (CMI) *Heart Failure Management Guidelines*,¹¹ available by request through the CMI Product Information Line, 510-271-6426; by e-mail at CMIproducts@kp.org; or at CMI's Web site, <http://pkc.kp.org>.

Vignette

A 78-year-old woman states that for the past two months, she has had gradually progressive fatigue; occasional cough; dyspnea during exertion; orthopnea; ankle edema; and a 10-lb (22-kg) weight gain. She denies chest discomfort, fever, or chills. She has hypertension treated with diltiazem, she quit smok-

ing 20 years ago, and she rarely drinks alcohol.

Physical examination shows an afebrile female patient with heart rate 105 beats per minute, blood pressure 130/70 mm Hg, respiratory rate 16 per minute, and oxygen saturation 94% on room air. The jugular veins are elevated with positive abdominojugular reflux. Pulmonary examination shows expiratory wheezing. The heart rate is regular without murmur, and the apical impulse is displaced and sustained. The patient has mild hepatomegaly and 2+ ankle edema. Electrocardiograms show sinus tachycardia and left ventricular hypertrophy. Chest x-ray films show cardiomegaly and pulmonary venous congestion. Levels of thyroid-stimulating hormone, albumin, ferritin, and serum creatinine are normal as are results of complete blood count.

Echocardiography shows moderate left ventricular dilation with global hypokinesis, LVEF of 30%, left ventricular hypertrophy, left atrial enlargement, mild mitral and tricuspid valve regurgitation, and pulmonary artery systolic pressure ranging from 45 mm Hg to 50 mm Hg. Angiography in this patient shows normal coronary arteries and confirms the finding of reduced left ventricular ejection fraction of 30%.

The patient begins a daily regimen of 20 mg oral furosemide, 10 mEq potassium, and 10 mg lisinopril. We phone her after three days to ensure that she is losing weight and improving. At a clinic visit one week later, she has lost 7 lb, and her symptoms have improved. (This typical response to diuretics, ie, weight loss and improved symptoms, is final confirmation that the patient does indeed have heart failure). Despite this improvement, the patient still has orthopnea and elevated neck veins—findings that indicate hypervolemia and a need for additional diuresis and vasodilation. We double her dose of furosemide and lisinopril. One week later, she has lost an additional 3 lb, denies orthopnea, and no longer has elevated neck veins.

After the patient becomes euvoletic, she starts a regimen of low-dose metoprolol (a beta-blocker) at a dosage of 12.5 mg orally twice daily. During beta-blocker

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Optimal treatment of heart failure requires correct diagnosis, identification of potentially reversible causes, appropriate use of medication, and patient education on self-care.

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Heart failure is a clinical syndrome ... no single symptom or sign is pathognomonic ...

titration, we monitor her closely for signs of worsening symptoms and weight gain and double the dose of metoprolol every two weeks as tolerated. When the dose of metoprolol is increased to 50 mg twice daily, the patient reports a 3-lb weight gain and return of orthopnea. This decompensation must be addressed before we can further increase the dosage of beta-blocker. Options for therapy include diuresis and increasing vasodilation. We choose to double the dose of furosemide. A week later, the patient has lost 3 lb, and the orthopnea has resolved. Her heart rate is 70 beats/minute, and her blood pressure is 110/70 mm Hg. We increase the dosage of metoprolol to 100 mg twice daily (the maximum dosage). The patient is now taking lisinopril (20 mg once daily), metoprolol (100 mg twice daily), furosemide, and potassium. Her heart rate is 60 beats/minute, and her blood pressure is 105/70 mm Hg.

Comment
Diagnosis

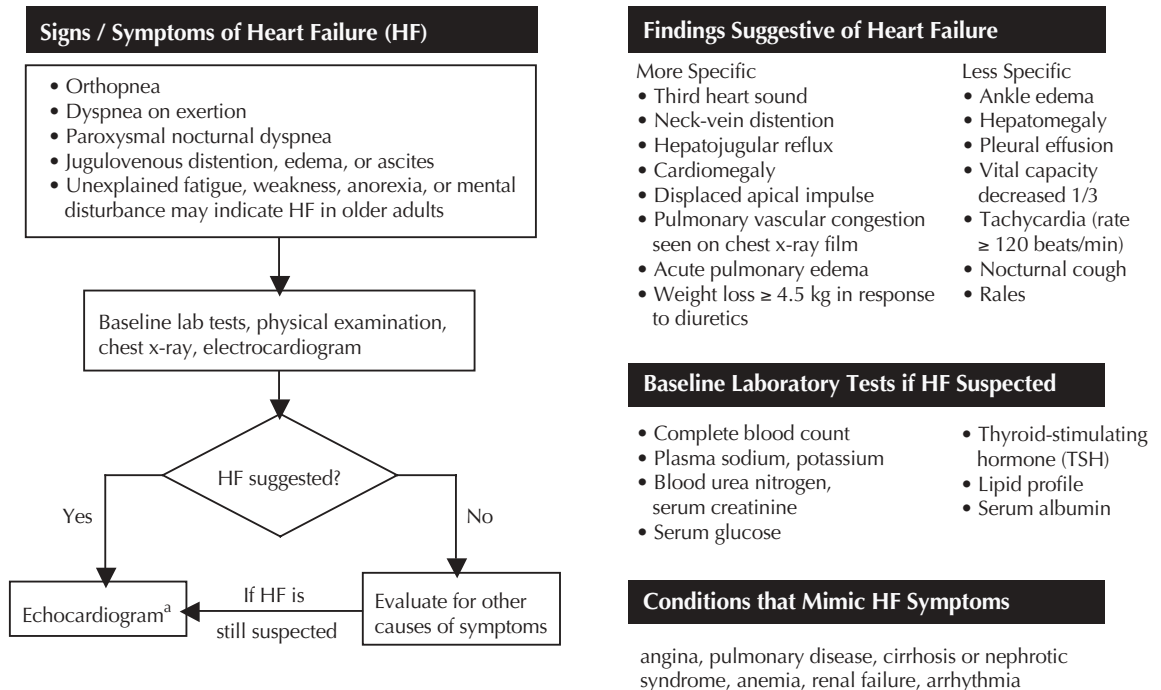
What is the diagnosis in the vignette presented? Which findings are most important for establishing

the diagnosis? Which tests should be ordered? If the patient has heart failure, what caused it? What treatment should be given?

Heart failure is a clinical syndrome where symptoms occur because the heart is either 1) unable to pump blood adequately to meet the body's needs or 2) able to do so only at high intracardiac pressure. The diagnosis of heart failure is suggested by presence of characteristic symptoms (Figure 1). Because no single symptom or sign is pathognomonic, clinicians must weigh multiple pieces of evidence and must consider conditions that mimic heart failure. However, studies have shown that, when considered together, the patient's medical history as well as results of physical examination, electrocardiography, and chest x-ray imaging can accurately indicate the diagnosis in more than 90% of cases.¹²

In the vignette presented, the clinical presentation is highly suggestive of heart failure. Results of physical examination and chest x-ray imaging show evidence of abnormal pump function (pulmonary and systemic venous congestion resulting from elevated intracardiac pressure and cardiomegaly). Two of the most suggestive findings for heart failure—an abnormal apical im-

Figure 1. Diagnosing heart failure



^a Normal echocardiogram does not rule out diastolic dysfunction

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pulse and elevated jugular venous pressure—are often overlooked. If these physical signs had not been sought, this former smoker who was wheezing might have been diagnosed with chronic obstructive pulmonary disease and been treated with bronchodilators. In this case, pulmonary venous congestion caused “cardiac asthma.” More commonly, results of lung examination are normal in patients with chronic heart failure. Rales, sometimes mistakenly believed to be a sensitive indicator of heart failure, are absent in more than 80% of patients with chronic heart failure.¹³ Similarly, patients with chronic heart failure may have normal chest x-ray films. Because jugular venous pressure is one of the most useful physical findings for diagnosing heart failure¹⁴ and is essential for assessing volume status in response to treatment, skill in examining the neck veins is important.

Examining the Neck Veins for Jugular Venous Pressure

Jugular venous pressure is estimated by measuring the vertical height of the internal jugular vein above the sternal angle (the junction of the manubrium and sternum).¹⁵ In a well-lit room, place the patient at 45 degrees from horizontal. Position the head to relax the neck muscles, and spread the skin smoothly—but not tautly—across the right side of the neck. Locate the pulsations of the right internal jugular vein, which runs between the heads of the sternocleidomastoid muscle (Figure 2). Normal pressure is located less than 4 cm vertically above the sternal angle or only about 1 inch above the clavicle when the patient is positioned at 45 degrees from the horizontal.¹⁵

Observing the external jugular vein is another way to locate the venous pressure. Although the external jugular vein may be compressed by the neck muscles

Figure 2. Neck vein examination



Compress external jugular

Collapses when released

and thus be falsely elevated, more often the external jugular mirrors the internal jugular vein. The external jugular vein can be located easily by compressing its base (causing the vein to fill) and then observing how the vein collapses when released. Table 1 contains clinical clues that can help differentiate jugular venous pulsations from carotid pulsations.

Echocardiography Used to Search for Structural Heart Disease

When heart failure is suspected on the basis of the patient's medical history and results of physical examination, evidence of abnormality in the heart should be sought. This examination usually consists of echocardiography, which assesses the structure and function of the ventricles and valves. Although heart failure rarely occurs in structurally normal hearts (eg, as occurs with high-output heart failure), abnormal results of echocardiography often provide evidence supporting the diagnosis and help identify the responsible form of cardiac dysfunction and thus allow therapy to

... patients with chronic heart failure may have normal chest x-ray films.

Maneuver	Jugular venous pulsations	Carotid pulsations
Observe	Diffuse and biphasic, rising and falling with respiration	Sharp, monophasic, unvarying
Palpate	Nonpalpable	Palpable
Compress base of external jugular vein	Pulsations higher in neck are obliterated	Pulsations will persist
Reposition patient	Vary with position of patient	Does not vary with position of patient
Apply pressure to midabdomen	Pulsations rise. Sustained rise of 4 cm for more than 10-15 seconds of pressure constitutes positive abdominojugular reflex.	No change

All patients with heart failure should be screened for coronary artery disease ...

be directed appropriately. The normal range for left ventricular ejection fraction (LVEF) is 50% to 70%. Systolic dysfunction is defined as LVEF less than 45%. Valvular dysfunction should be considered a potential cause of heart failure when the echocardiogram shows moderate or severe valvular stenosis or regurgitation. Mildly elevated pulmonary pressure (40-60 mm Hg) is characteristic of heart failure and does not necessarily suggest a separate disease process.

Cardiac dysfunction sufficient to cause heart failure can exist even when the echocardiogram appears normal. This situation is seen most often in diastolic dysfunction. Echocardiography is not mandatory if valvular and left ventricular systolic function has been assessed by other measures, such as by cardiac catheterization.

Table 2. Causes of heart failure in patients with reduced left ventricular ejection fraction
Coronary artery disease
Alcohol use
Valve dysfunction, especially mitral and aortic regurgitation
Hypertension
Tachycardia-induced cardiomyopathy
Hypothyroidism
Hemochromatosis
Idiopathic disease

An Assay Used to Diagnose Heart Failure

Recently, heart failure was accurately diagnosed by using an assay for B-type natriuretic peptide (BNP), a hormone released from the ventricles in response to stretch and pressure overload.¹⁶ The exact role of this assay is still being defined, but the assay will probably be most useful when heart failure is suspected but remains unconfirmed after the medical history has been carefully obtained and physical examination, electrocardiography, and radiography of the chest have been done.

Causes of Systolic Heart Failure

The echocardiogram for the 78-year-old woman in the vignette shows a reduced LVEF (systolic dysfunction) without clinically significant valve disease. Moderate elevation of pulmonary pressure helps to confirm heart failure. After heart failure is diagnosed, the cause must be identified by focusing on a short list of conditions that are common or potentially treatable (Table 2). In addition to basic laboratory tests (Figure 1), measurement of the plasma ferritin level may be used to screen for hemochromatosis, a condition which

is relatively common and potentially treatable. Other tests, such as rheumatologic serology, should be obtained only if the medical history and results of physical examination suggest a specific diagnosis.

Screening for Coronary Artery Disease

All patients with heart failure should be screened for coronary artery disease because it is the most common cause of reduced LVEF. However, no consensus exists on the best screening strategy.⁵ Options include clinical assessment consisting of medical history, physical examination, stress imaging,⁴ or coronary angiography. In general, proceeding directly to angiography should be considered for heart failure patients who have typical angina, flash pulmonary edema, or multiple coronary risk factors (eg, tobacco use combined with diabetes). Angiographic results for the 78-year-old woman in the *Vignette*—normal coronary arteries and confirmation of the 30% left ventricular ejection fraction—were consistent with nonischemic cardiomyopathy.

Criteria for Hospital Admission

Should this patient be admitted to the hospital? Common reasons to consider admission are listed in Table 3.¹⁷ Admission often depends on how ill a patient appears. In this *Vignette* of a 78-year-old female patient with gradual onset of symptoms, outpatient management was appropriate.

Treating Systolic Heart Failure: Countering Compensatory Reflexes Gone Astray

Our understanding of the treatment for systolic heart failure has been greatly advanced by understanding its pathophysiology. When ventricular function is compromised, neurohormones (including norepinephrine and the renin-angiotensin-aldosterone system) are activated. These hormones lead to vasoconstriction and

Table 3. Indications for hospital admission for patients with heart failure¹⁷
Abrupt onset of heart failure
Coexisting pneumonia
Evidence of ischemia
Severe exacerbation of chronic obstructive pulmonary disease
Oxygen saturation <90%
Poorly tolerated cardiac arrhythmia
Severe respiratory distress
Severe hypertension
Hypotension
Acute renal failure
Syncope

fluid retention—reflexes which are meant to counteract dehydration but which tend to worsen heart failure. Gradually, these neurohormones cause myocyte death and fibrosis, further worsening ventricular function. Untreated ventricular dysfunction tends to progress, causing worsening heart failure and, eventually, death.

Countering these reflexes with diuretic agents and vasodilators has emerged as a main component of heart failure management. Moreover, agents that block the damaging neurohormones have proved more beneficial than agents that merely correct the hemodynamic derangement which occurs in heart failure. For example, angiotensin-converting enzyme (ACE) inhibitors affect survival more favorably than direct-acting vasodilators such as calcium channel blockers. Similarly, norepinephrine-antagonist beta-blockers (formerly contraindicated because of their negative, inotropic effects) actually improve survival more than any other drug class. In patients with systolic heart failure, survival has been improved by three categories of medication,

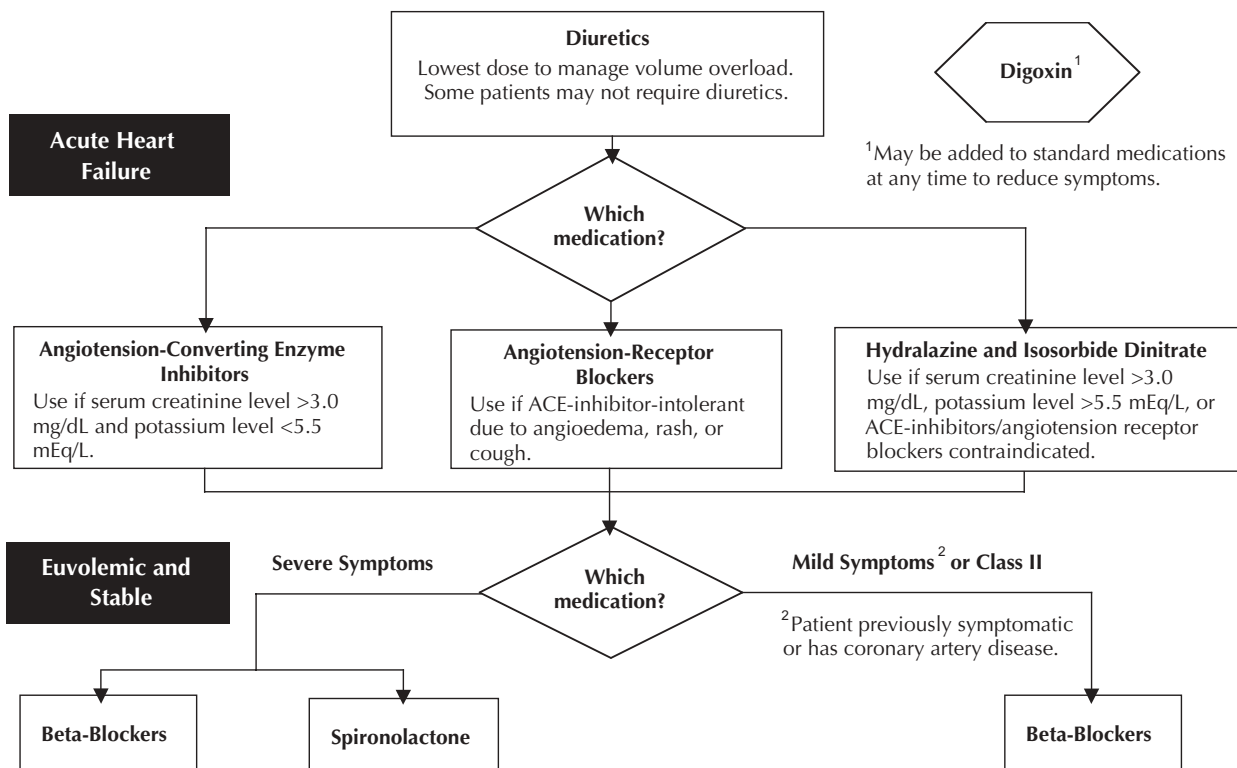
sometimes referred to as “triple therapy”: vasodilators (especially ACE inhibitors, which can lead to 23% relative reduction in mortality);¹⁸ beta-blockers (which can lead to a 35% relative reduction in mortality);¹⁹ and spironolactone (which can lead to a 30% relative reduction in mortality).²⁰

Sequence of Medication Titration

Initially, diuretic agents and vasodilators should be used to stabilize the condition of patients with heart failure (Figure 3).¹¹ ACE inhibitors are the vasodilators preferred on the basis of multiple clinical trials that showed mortality benefit.¹⁹ Use of angiotensin receptor blockers is an alternative for patients who have intolerance to ACE inhibitors (eg, because these patients have cough, angioedema, or allergy). Patients with renal dysfunction or hyperkalemia should be treated with hydralazine and isosorbide dinitrate.

After the patient’s condition has stabilized, a regimen of beta-blockers is added. Beta-blockers may initially worsen heart failure and therefore must be initiated at a low dose

Figure 3. Management of heart failure with decreased systolic function



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and titrated slowly—and only after volume overload is corrected. Patients should be instructed that although beta-blockers may initially worsen symptoms, this effect is almost always transient and correctable. Patients can be told that their long-term quality and quantity of life will be improved. Teaching patients the signs and symptoms of deterioration before beta-blocker titration is begun can help prevent problems during titration.

For patients with LVEF less than 35% and severe symptoms (such as fatigue or dyspnea with minimal activity), spironolactone (an aldosterone blocker) also decreased mortality.²⁰ Renal insufficiency and hyperkalemia are contraindications to spironolactone, and potassium levels of patients receiving spironolactone must be monitored closely.²⁰ When administered at higher doses, spironolactone is a potassium-sparing diuretic; however, in patients with heart failure, spironolactone is used at subdiuretic doses and does not replace furosemide.

Digoxin does not improve survival but has been shown to reduce hospitalization rates slightly.²¹ Thus, use of digoxin may be appropriate for patients who remain both symptomatic and at risk for hospitalization despite other therapeutic measures.

Monitoring Response to Treatment

During medication titration, the clinician must assess response to treatment, particularly the patient's volume status. For this assessment, examining the neck veins for jugular venous pressure is crucial. Elevated venous pressure indicates hypervolemia and the need for further diuresis. Orthopnea suggests continued elevation of pulmonary wedge pressure and the need for further diuresis, vasodilation, or both. Treatment can be monitored by phone and occasionally at clinic visits as long as the patient is doing well and has vital signs checked and blood tests drawn.

Has the medication regimen of the patient in the *Vignette* been finally adjusted? No. Even though she is doing well with the current regimen of furosemide, potassium, lisinopril, and metoprolol, we should increase the lisinopril to the maximum dose of 40 mg if tolerated, because higher doses of ACE inhibitors have shown reduced rates of hospitalization and mortality more effectively than lower doses.¹⁹ Spironolactone should then be added if the patient remains substantially symptomatic, because this drug can further decrease mortality.²⁰ Spironolactone's mortality benefit is uncertain in patients with mild symptoms. Digoxin can be added to the medication regimen if the patient remains substantially symptomatic despite all other measures taken.²¹

The importance of patient education in treating heart failure cannot be overstated.

Anticoagulation

Accepted indications for anticoagulation therapy in patients with heart failure include atrial fibrillation, left ventricular thrombus, and previous diagnosis of thromboembolism.¹¹ Routine use of warfarin in patients with severely reduced LVEF has not conclusively shown decreased risk of stroke.²²

Patient Education:

Self-Care for Heart Failure

The *Vignette* illustrates the essential role of patient self-care during medication titration. The patient helped guide medication adjustments by weighing herself daily and by reporting worsening symptoms. The importance of patient education in treating heart failure cannot be overstated. Education alone, independent of any changes in medical therapy, has shown reduced rates of rehospitalization by 39%²³ and gives patients a sense of empowerment and control over their health. Among the most valuable teaching points is that sudden weight gain—2 lb (4.4 kg) in one day or 5 lb (11 kg) in one week—is the earliest sign of fluid retention. To prevent hospitalization resulting from bowel edema (which may impair absorption of oral medication), patients with heart failure should respond to fluid-based weight gain by increasing their dose of diuretic agents. Other important components of patient education for heart failure are listed in Table 4.

Table 4. Important educational components for patients with heart failure

Early recognition of signs and symptoms of decompensation
Understanding the importance of medication compliance
Sodium intake \leq 2 g daily (requires teaching to read food labels)
Flexible diuretic dosing on basis of patient's weight (measured daily)
Physical activity

Considerations for Patients with Advancing Illness

Frank discussion of prognosis is an important aspect of patient education. Heart failure has a high mortality rate, and half of deaths occur suddenly and unpredictably. Therefore, planning for the end of life is essential even when the patient feels well. Patients should be told that heart failure is a serious disease that is often not curable. Although treatment can improve both quality and length of life, many patients die of heart failure

Table 5. Medication-related causes of refractory or worsening systolic heart failure

Medication to avoid in patients with systolic heart failure	Reason
Nonsteroidal anti-inflammatory drugs and COX-2 inhibitors	May promote fluid retention and worsen renal function
Calcium channel blockers (except felodipine and amlodipine)	May accelerate heart failure progression and increase exacerbation
Antiarrhythmic drugs (except amiodarone and dofetilide)	Can increase risk of sudden death
Use with caution:	
Metformin	Must be discontinued when serum creatinine level >1.5 mg/dL (132.6 μ mol/L)
Thiazolidinediones (eg, pioglitazone, rosiglitazone)	Can cause fluid retention (usually mild) requiring additional diuresis

nonetheless. Health decline in heart failure may be sudden or gradual, and timing of deterioration is often unpredictable; therefore, planning early for advancing illness—including medical, financial, legal, and personal needs—is important.¹¹

Patients with Refractory Symptoms

Hemodynamic state should be reassessed in patients who remain severely symptomatic despite medical therapy. Examining jugular venous pressure is an invaluable method of determining the patient's volume status.¹⁵ If the jugular venous pressure is normal (indicating adequate diuresis), additional vasodilation to reduce vascular resistance and to improve cardiac output should be considered. A second vasodilator may be required; however, few clinical trials have examined the long-term effects of adding a second vasodilator, and no studies have found mortality benefit. In fact, in a recent trial of the angiotensin receptor blocker valsartan, mortality rates were increased when valsartan was added to an ACE inhibitor and beta-blocker.²⁴ This finding suggests that all three of these drugs should not be used simultaneously. For patients already receiving an ACE inhibitor and beta-blocker, alternative second vasodilators include hydralazine plus isosorbide dinitrate; or the calcium channel blockers amlodipine or felodipine. Compared with placebo initial therapy with hydralazine and isosorbide has shown improved survival,²⁵ but the combination of hydralazine and isosorbide has not been studied when added as a second vasodilator. Amlodipine and felodipine have no proven effect on mortality.²⁶ Digoxin does not improve mortality²¹ but should be considered for patients with refractory symptoms, especially patients who are hypotensive.

In patients with persistent hypervolemia, sodium intake should be carefully reviewed. Hypervolemia that persists despite sodium restriction may be caused by

decreased renal responsiveness to loop diuretic agents, possibly caused by hypertrophy of the distal nephron and resultant increased sodium resorption distal to the loop of Henle. Adding a thiazide diuretic (such as hydrochlorothiazide or metolazone) to block distal sodium resorption can restore the effectiveness of loop diuretic agents²⁷ but necessitates close monitoring of potassium levels (because potassium loss is exacerbated by addition of the thiazide diuretic agent).

Medication-related causes of refractory or worsening systolic heart failure are listed in Table 5.

Referral to Heart Failure Programs

Kaiser Permanente has developed heart failure programs that provide many of the components of management described in this article, including patient education and medication titration. These disease management programs are supervised by physicians, conducted by nurses and clinical pharmacists, and modeled on programs shown to reduce rates of hospitalization while improving quality of care. Interested patients who meet local eligibility criteria should be referred to these programs.

Heart Failure in Patients with Normal Left Ventricular Ejection Fraction

The 78-year-old woman in the *Vignette* was based on an actual patient but was altered in one important respect: The actual echocardiogram revealed normal chamber sizes, left ventricular hypertrophy, mildly elevated pulmonary artery systolic pressure (between 45 mm Hg and 50 mm Hg), and *normal LVEF ranging from 70% to 75%*.

Was the diagnosis of heart failure mistaken? No. This patient presented with classic heart failure: orthopnea, weight gain, elevated neck veins, and a characteristic response to diuresis. Moreover, moderately elevated

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pulmonary pressure was a finding consistent with heart failure. Population studies have found that 30% to 40% of patients (and 50% of very elderly patients) with heart failure have normal LVEF.^{4,5}

Heart failure with normal systolic function may occur for a number of reasons, such as valve dysfunction or pericardial disease. The most common cause is diastolic dysfunction, impaired ability of the ventricle to fill at normal pressure. Heart failure resulting from diastolic dysfunction may be clinically indistinguishable from systolic heart failure. Abnormal diastolic pressure shown on echocardiograms suggests the diagnosis of diastolic heart failure. However, echocardiography is not completely sensitive for diastolic dysfunction, and *heart failure is therefore not excluded by normal results of echocardiography.*

Potentially treatable causes of heart failure with normal LVEF should be sought. Common causes are listed in Table 6.

To date, few mortality trials have been done to guide therapy for heart failure when the ejection fraction is normal. Thus, therapy is empirical and is aimed at relieving symptoms and treating exacerbating conditions.

Table 6. Causes of heart failure in patients with normal left ventricular ejection fraction
Coronary artery disease ^a
Hypertension (especially with left ventricular hypertrophy) ^a
Diabetes mellitus ^a
Valve dysfunction
Atrial fibrillation
Cor pulmonale (right-sided heart failure)
Hypertrophic cardiomyopathy
Pericardial disease

^acontributes to diastolic dysfunction

Table 7. Treatment of heart failure in patients with normal systolic function
Treat fluid retention: Remove excess fluid with loop diuretics.
Control blood pressure: Treat hypertension aggressively with a goal blood pressure of <i>less than</i> 130/85 mm Hg. Agents are preferred which regress LVH, such as ACE inhibitors (angiotensin-receptor blockers if patient has intolerance to ACE inhibitors), beta-blockers, and diuretics.
Control heart rate: For patients with atrial fibrillation and heart failure, restore sinus rhythm if possible. To lengthen diastolic filling time, slow ventricular response rate with beta-blockers, digoxin, verapamil, or diltiazem. For patients in sinus rhythm, slow rate to 55 to 75 beats per minute with beta-blockers.
Treat ischemia: Treat ischemia with anti-anginal medication, such as beta-blockers, nitrates, or calcium channel blockers. Refer for revascularization when appropriate.
Educate patients: Self-care education is the same as for systolic dysfunction and is equally important.

Principles of management are listed in Table 7,¹¹ and key points of heart failure management are summarized in Table 8.

Many patients with heart failure and normal LVEF receive some of the same medications as those with systolic heart failure—loop diuretics, ACE inhibitors, and beta-blockers. However, beta-blockers may be used before ACE inhibitors and may be titrated more rapidly in patients with normal systolic function. Spironolactone has not been studied in patients with normal LVEF and is generally not recommended.

Conclusion

The past decade has seen many advances in the treatment of heart failure, and these advances offer our patients improved survival and quality of life. Heart failure is a clinical diagnosis, made after weighing multiple pieces of evidence. Examination of the neck veins for jugular venous pressure is useful both for diagnosing heart failure and for monitoring response to therapy. Once heart failure is diagnosed, treatable causes should be sought.

Three classes of medication—beta-blockers, vasodilators (especially ACE inhibitors), and spironolactone—have shown improved survival in patients with heart failure caused by reduced LVEF. Survival data for patients with heart failure and normal LVEF are lacking, and treatment for these patients is empirical but ultimately includes many of the same medications used to treat systolic heart failure. For all patients with heart failure, education on prognosis and self-care is essential. ❖

^a Stress testing without imaging is usually not sensitive enough to accurately diagnose coronary artery disease in patients with heart failure.

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Table 8. Key points in diagnosis and treatment of patients with heart failure

Heart failure is a clinical diagnosis.
Examination of jugular venous pressure is important for diagnosing heart failure and monitoring response to therapy.
Seek reversible cause.
Angiotensin-converting enzyme (ACE) inhibitors, beta-blockers, and spironolactone improve survival in patients with heart failure and decreased left ventricular ejection fraction.
Treatment for heart failure patients with normal left ventricular ejection fraction is empirical but uses many of the same medications as for systolic heart failure (except spironolactone).
Patient education and self-management are essential components of heart failure treatment.

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