Take-Home Points

The most common cause of heart failure is impaired contractility of the left ventricle (LV), due to CAD, MI, dilated cardiomyopathy, valvular heart disease, or hypertension. Right ventricular (RV) failure also can occur, most commonly due to LV failure, RV infarction, pulmonary hypertension, or tricuspid regurgitation.

Signs/symptoms of advanced disease include dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. Fatigue, chest pain/pressure and palpitations also may occur. With RV failure, jugular venous distention, peripheral edema, hepatomegaly, and ascites are common. Auscultation may reveal a gallop rhythm (with S3 and often S4 sounds) and in valvular disease, heart murmurs. Patients' functional impairment is categorized by the level of dyspnea they experience, as outlined in the New York Heart Association Heart Failure Symptom Classification System:

<table>
<thead>
<tr>
<th>Class</th>
<th>Level of Impairment</th>
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<tbody>
<tr>
<td>I</td>
<td>No symptom limitation with ordinary physical activity</td>
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<tr>
<td>II</td>
<td>Ordinary physical activity somewhat limited by dyspnea (e.g., long-distance walking, climbing two flights of stairs)</td>
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<tr>
<td>III</td>
<td>Exercise limited by dyspnea with moderate workload (e.g., short-distance walking, climbing one flight of stairs)</td>
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<tr>
<td>IV</td>
<td>Dyspnea at rest or with very little exertion</td>
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With good management, even patients with Class III or IV heart failure can remain relatively stable most of the time. However, patients can decompensate and develop acute dysfunction, most typically resulting in pulmonary edema or hypotension/shock. The following pointers address the basics in the management of stable heart failure and acute decompensation.

Assessment/Information Gathering

- To identify stable CHF:
  - Assess for the signs and symptoms previously noted.
  - Recommend electrolytes (fluid balance, sodium levels), BUN and creatinine (renal function), and brain natriuretic peptide or BNP (useful in diagnosing CHF)
  - Recommend chest X-ray; look for cardiomegaly, pulmonary vascular congestion, Kerley B lines, pleural effusion.
  - Recommend 12-lead ECG to assess for LV or RV hypertrophy or ischemia/CAD.
  - If ECG indicates ischemia/CAD, recommend stress test, cardiac catheterization or coronary CT angiography (CTA) to confirm or exclude CAD as the cause
  - Recommend an echocardiogram to assess for systolic and diastolic function, hypertrophy, chamber size, and valve abnormalities (discussed subsequently).

- To assess for decompensation/pulmonary edema:
  - Assess for signs of acute decompensation, e.g., restlessness, confusion, dyspnea, ↑ work of breathing, diaphoresis, tachypnea, tachycardia, low pulse pressure.
  - Assess peripheral perfusion; look for cool, pale, cyanotic or mottled skin, slow capillary refill.
  - Assess cough/sputum production; look for frothy and/or blood-tinged sputum.
  - Assess breath sounds (bilateral crackles + wheezing -> acute decompensation).
Assess for chest pain (presence suggests acute myocardial ischemia/infarction).
Monitor SpO2 and obtain an ABG (hypoxemia with respiratory alkalosis typical).
Recommend a STAT chest X-ray (typically reveals bilateral fluffy infiltrates).
Recommend CBC, serum electrolytes, BUN and creatinine, BNP (see above), and cardiac biomarkers (troponin, CK, CK-MB) to assess for MI.
Recommend an echocardiogram (helps determine possible mechanical causes such as cardiac tamponade or valve problems).
In patients who have respiratory distress or clinical evidence of impaired perfusion, recommend pulmonary artery (Swan-Ganz) catheterization to assess heart function and guide therapy.

Treatment/Decision-Making

- To manage stable CHF:
  - Recommend disease management education, with an emphasis on sodium and fluid restriction, smoking cessation, daily monitoring of BP, weight control and moderate aerobic exercise.
  - Recommend the following medications for all CHF patients:
    - an angiotensin-converting enzyme (ACE) inhibitor (e.g., captopril) or angiotensin receptor blocker (e.g., valsartan)
    - a beta-blocker (e.g., carvedilol)
  - Depending on the severity of symptoms additional medications may include digoxin (especially with A-fib), a diuretic (preferably a loop diuretic like furosemide or torsemide) and an aldosterone antagonist such as spironolactone.

- To manage decompensation/pulmonary edema:
  - Initiate O2 therapy with the highest FIO2 possible (nonrebreather at 12-15 L/min or high-flow cannula at 30-40 L/min) to obtain SpO2 > 90%.
  - Recommend CPAP or BiPAP with 100% O2 (improves gas exchange, decreases venous return and ventricular preload)
  - Recommend short-term treatment with a beta-2 agonist (e.g., albuterol) for patients experiencing dyspnea (may improve pulmonary function, cardiovascular hemodynamics, and help resorption of edema fluid)
  - Recommend morphine or a benzodiazepine such as lorazepam to reduce anxiety (morphine also may decrease preload via venous dilation).
  - Recommend the appropriate ACLS protocol for any associated arrhythmia or MI.
  - Recommend the following medications (assumes the patient is not hypotensive):
    - a vasodilator such as nitroglycerin, sodium nitroprusside, nesiritide (to decrease preload and afterload)
    - a rapid acting loop diuretic such as furosemide or torsemide
  - If patient is hypotensive recommend an inotrope such as dobutamine (adrenergic) or milrinone (PDE inhibitor) to maintain mean arterial pressure > 70-75 mm Hg.
  - Recommend intubation and invasive ventilation if patient develops severe respiratory acidosis on CPAP/BiPAP.
  - In the patient with persistent hypotension and pulmonary edema due to an acute MI, recommend intra-aortic balloon pumping (if available) until angioplasty or cardiac surgery can be performed.
Follow-up Resources

Standard Text Resources:


Useful Web Links:


