Acute Heart Failure: Pathophysiology and Management Pearls

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Abstract

Acute heart failure (AHF) is a global pandemic with more than one million admissions to hospital annually in the US and more millions all-over the world. Despite several critical steps forward in the management of chronic HF (CHF), the area of AHF has remained relatively stagnant. Treatment of AHF remains largely opinion-based with little good evidence to guide therapy. This review well summarized the epidemiology, underlying etiology and management of acute heart failure syndromes.

Keywords: Acute Heart Failure; Acute Pulmonary Edema; Acute Decompensated Heart Failure

Abbreviations

ACS: Acute Coronary Syndrome; ADCHF: Acute Decompensated Chronic Heart Failure; ADHERE: Acute Decompensated HF National Registry; AF: Atrial Fibrillation; AHF: Acute HF; BNP: Brain Natriuretic Peptides; BUN: Blood Urea Nitrogen; CHF: Congestive Heart Failure; COPD: Chronic Obstructive Pulmonary Disease; CPAP: Continuous Positive Airway Pressure; cTns: Cardiac Troponins; CVA: Cerebrovascular Accident; EHFS: European HF Survey; EF: Ejection Fraction; HFrEF: HF with Reduced EF; HfPEF: HF with Preserved EF; ICD: Intra-cardiac Defibrillator; LVEF: Left Ventricular Ejection Fraction; NIPPV: Non-invasive Positive Pressure Ventilation; NIV: Non-invasive Ventilation; NPs: Natriuretic Peptides; NTproBNP: N Terminal proBNP; NSAID: Non-steroidal Anti-Inflammatory Drugs; OPTIMIZE-HF: Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with HF; VAD: Ventricular Assist Device

Definition

Acute heart failure (AHF) is the term used to describe the rapid onset of, or acute worsening of symptoms and signs of HF, associated with elevated plasma levels of natriuretic peptides. It is a life threatening condition that requires immediate medical attention and usually leads to urgent hospital admission [1].

Epidemiology

- AHF is a prevalent condition, as it represents the first reason for hospitalization in advanced age.
- Furthermore, AHF is a condition with an adverse prognosis, characterized by high mortality and rehospitalization rates.
- Finally, AHF represents a significant financial burden to health systems, as the enormous health care expenditure required for heart failure is mainly related to hospitalizations for AHF [2].
- It is characterized by an adverse prognosis, with an in-hospital mortality rate of 4 - 7%, a 2 - 3-month post-discharge mortality of 7 - 11% and a 2 - 3-month readmission rate of 25 - 30% [2].
- Based on acute HF registries (ADHERE [3], OPTIMIZE-HF [4], EHFS II [5]), most who are admitted with HF are over age 70, have a prior history of admission for HF and 40 - 52% have preserved LVEF.

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Pathophysiology

1. Substrate: myocardial structure and function [6]
   a. Normal myocardial substrate that has suffered an acute injury
      i. Ischemia/infarction.
      ii. Inflammation (myocarditis, autoimmune).
      iii. Could be completely reversible, partially reversible or irreversible.
   b. Abnormal underlying substrate
      i. Stage B with first symptomatic event.
      ii. Those with chronic compensated HF who present with an acute decompensation Most common presentation.

2. Triggering mechanisms [7]
   a. Acute coronary syndrome.
   b. Tachyarrhythmia e.g. AF others.
   c. Excessive rise in blood pressure.
   d. Infection (e.g. pneumonia, infective endocarditis, sepsis).
   e. Non-adherence salt intake and medications.
   f. Bradyarrhythmia.
   g. Toxic substances (alcohol, recreational drugs).
   h. Drugs (e.g. NSAIDs, corticosteroids, negative inotropic substances, cardiotoxic chemotherapeutics).
   i. Exacerbation of chronic obstructive pulmonary disease.
   j. Pulmonary embolism.
   k. Surgery and perioperative complications.
   l. Increased sympathetic drive, stress-related cardiomyopathy.
   m. Metabolic/hormonal derangements (e.g thyroid dysfunction, diabetic ketois, adrenal dysfunction, pregnancy and peripartum related abnormalities).
   n. Cerebrovascular insult.
   o. Acute mechanical cause: myocardial rupture complicating ACS chest trauma or cardiac intervention, acute native or prosthetic valve incompetence secondary to endocarditis, aortic dissection or thrombosis.

3. Amplifying mechanisms [6]:
   a. Myocardial dysfunction.
   b. Neurohormonal and inflammatory activation.
   c. Renal dysfunction.
   d. Vascular endothelial dysfunction, arterial stiffness, VC.

Cardiac versus vascular failure [2]

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Cardiac failure</th>
<th>Vascular failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart failure type</td>
<td>Fluid retention</td>
<td>Fluid redistribution</td>
</tr>
<tr>
<td>Onset</td>
<td>AD/CHF</td>
<td>De novo</td>
</tr>
<tr>
<td>Signs</td>
<td>Peripheral and pulmonary congestion</td>
<td>Normal or low</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal or low</td>
<td>Normal or increased</td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>Reduced</td>
<td>Preserved</td>
</tr>
<tr>
<td>Filling pressures</td>
<td>Lower</td>
<td>High</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>Low</td>
<td>Higher</td>
</tr>
<tr>
<td>Main therapy</td>
<td>Diuretics</td>
<td>Vasodilators</td>
</tr>
<tr>
<td>Mortality rate</td>
<td>High</td>
<td>Lower</td>
</tr>
<tr>
<td>Rehospitalization rate</td>
<td>High</td>
<td>High</td>
</tr>
</tbody>
</table>

Table 1: Cardiac Versus Vascular failure.

Classifications [2]:

1. Past history of heart failure
   a. Acute decompensated CHF (ADCHF).
   b. De novo AHF.

2. Blood pressure upon presentation
   a. Hypertensive AHF
   b. Normotensive AHF.
   c. Hypotensive AHF.

3. LVEF
   a. AHF with reduced LVEF < 40%.
   b. AHF with preserved LVEF > 50%.
   c. AHF with borderline or mid-range EF 41 - 49%.
   d. AHF with improved EF > 41 with history of treated HFrEF.

4. Congestion and peripheral perfusion
   a. Warm and dry.
   b. Warm and wet.
   c. Cold and dry.
   d. Cold and wet.

5. Clinical profile upon presentation
   a. Decompensated heart failure.
   b. Acute pulmonary oedema.
   c. Cardiogenic shock.
   d. Hypertensive heart failure.
   e. Right heart failure.
   f. ACS with AHF.

Prognostic factors in AHF

1. Clinical:
   a. Age
   b. Heart rate
   c. Systolic blood pressure
   d. $O_2$ saturation
   e. 6-min walked distance
   f. Need of inotropic agents
   g. Ischemic electrocardiogram (ECG) changes.

2. Medical history:
   a. Recurrent hospitalizations
   b. Renal dysfunction
   c. COPD
d. Anemia

e. Cerebrovascular events

f. Peripheral vascular disease.

3. Laboratory:

a. Natriuretic peptides (NPs)
b. Cardiac troponins (cTns)
c. Serum urea or BUN
d. Serum creatinine
e. Serum Na+
f. Hemoglobin
g. Liver function tests
h. LVEF
i. Restrictive physiology (echo Doppler).

Evaluation of the patient with acute heart failure see figure 1 and 2.

Figure 1: Algorithm for management of AHF ESC HF guidelines 2016 ref 7.
Clinical assessment:

1. **Presentation**
   
a. Dyspnea on exertion
   
i. Most sensitive symptom
b. Paroxysmal nocturnal dyspnea
   
i. Most specific symptom
c. Peripheral edema
   
i. Less common (66%)
d. Fatigue
e. Cough, particularly nocturnal
f. Chest discomfort

2. **Physical examination**: A rapid initial assessment should be performed to identify:
   
a. Evidence of congestion
   
i. Left sided:
      1. $S_3$ and $S_4$ gallop
      2. Prominent $P_2$
      3. Bilateral basal rales
   
ii. Right sided:
      1. JVP $> 10$ cm
      2. Hepatoglandular reflux
      3. Hepatomegaly
      4. Edema LL
      5. Ascites

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**Figure 2**: Mechanistic approach to heart failure in the vulnerable phase and thereafter. ACEI: Angiotensin-Converting Enzyme Inhibitor; ARB: Angiotensin Receptor Blocker; ARNI: Angiotensin Receptor/Nephrilysin Inhibitor; BB: Beta-Blocker; COPD: Chronic Obstructive Pulmonary Disease; CRT: Cardiac Resynchronization Therapy; ICD: Implantable Cardioverter-Defibrillator; MRA: Mineralocorticoid Receptor Antagonist ref 1.
b. Evidence of low output/cardiogenic shock
   i. Low pulse pressure < 25 mm hg
   ii. Sinus tachycardia
   iii. Pulsus alternans
   iv. Cool extremities
   v. Lethargy/ altered mentation
   vi. Hypotension

c. Presence of co-morbidities and precipitating factors.

3. Investigations:
   a. X-ray chest:
      i. Normal in 25% of cases
      ii. My show:
         1. Dilated upper lobe vessels
         2. Interstitial edema
         3. Enlarged pulmonary arteries
         4. Pleural effusion
         5. Alveolar edema
         6. Prominent superior vena cava
         7. Kerley B lines
   b. ECG: For
      i. Coronary artery disease
      ii. Chamber enlargement
      iii. Dysrhythmias
      iv. Device mal function
   c. Laboratory:
      i. Biomarkers:
         1. Natriuretic Peptides BNP and NT pro BNP for diagnosis in suspected AHF (good negative predictive value) and for prognosis
         2. Cardiac troponin
      ii. Other laboratory tests:
         1. Electrolytes, including sodium (Na), calcium (Ca), potassium (K) and magnesium (Mg)
         2. Renal function (blood urea nitrogen (BUN), Creatinine (Cr))
         3. Liver function tests (LFT’s)
         4. Thyroid function tests
         5. Complete blood picture
   d. Echocardiography:
      i. Assess LV and RV Function
         1. Preserved or reduced
         2. Ventricular structure
3. Size
4. Wall thickness
   ii. Other structural abnormalities
      1. Valvular
      2. Pericardial
      3. Right ventricle
      4. Atrial size.

**Indications for hospitalization (HF society of America)**

1. Hospitalization is recommended for patients with ADHF who present with the following clinical circumstances:
   a. Hypotension
   b. Worsening renal function
   c. Altered mentation
   d. Rest dyspnea
   e. Tachypnea
   f. Hypoxia
   g. Hemodynamically significant arrhythmias
   h. New onset rapid atrial fibrillation
   i. ACS

2. Consideration of hospitalization should be made if:
   a. Evidence of worsening pulmonary or systemic congestion (even in the absence of dyspnea or weight gain)
   b. Marked electrolyte disturbances
   c. Multiple implantable cardioverter defibrillator (ICD) firings
   d. Co-morbid conditions
      i. Pneumonia
      ii. Diabetic ketoacidosis
      iii. Pulmonary embolus
      iv. Transient ischemic attack (TIA)/cerebrovascular accident (CVA).

**Goals of management**

- Rapidly relieve symptoms of congestion
- Identify reversible causes, particularly ischemia
- Restore hemodynamics
- Ensure adequate oxygenation
- Prevent end organ damage
- Identify patients with low output states.

**Phases of management:**

1. Phase I: Urgent/Emergent Care
   Rapid diagnostic workup with control of symptoms and life threatening conditions.

2. Phase II: Hospital Care
3. Phase III: Pre-discharge Planning
Optimize the chronic oral HF therapy and evaluation for readiness for discharge.

4. Phase IV: Post-discharge Management
Close monitoring in this vulnerable period. HF clinics and nurse visits may help.

Management of Congestion [8,9]:
- It will depend on 3 factors
  - Volume state
  - Blood pressure
  - Renal functions
- Diuretics are the corner stone of congestion management.
- IV loop diuretic either repeated bolus doses or continuous infusion.
- Addition of thiazide or thiazide like metolazone may be helpful
- Use of vaso-dilators may be needed if the BP is high.
- Ultrafiltration may be needed for patients with poor diuretic response.

O₂ therapy and assisted ventilation
My be needed in patients with hypoxia either be masks or non-invasive ventilation NIV with use of CPAP or non-invasive positive pressure ventilation NIPPV.

Vasodilator therapy:
- Recommended for rapid symptom relief in those with pulmonary congestion or hypertension.
- Use when symptoms persist despite aggressive diuretic and standard oral regimens.
- Do not use if the patient has symptomatic hypotension.
- The most commonly used vasodilators are
  - Nitro-glycerine
  - Na nitroprusside
  - Nesiritide
  - Serelaxin.

Hemodynamic support
- By use of inotropic agents
  - Dobutamine
  - Dopamine
  - Milrinone,
  - Norepinephrine
  - Enoximone
  - Levosimendan
- Mechanical circulatory support
  - Intra-aortic balloon
- Ventricular assist devices
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- Percutaneous VAD
  - Impella
  - Tandem heart
  - Extracorporeal Membrane Oxygenation (ECMO).

Conclusion

Acute heart failure is a common health problem with a gloomy prognosis on short and long term outcome. Its pathophysiology includes an interplay of underlying cardiac structural abnormality with large list of precipitating factors that results in a characteristic clinical presentation. This presentation include a combination of congestion and hypoperfusion manifestation. Management of AHF is usually based on physician's clinical decision rather than evidence based strategy due to the paucity of clinicial trials guideline recommendations.

Disclosure

No conflicts of interest.

Bibliography


