Acute Heart Failure

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Worsening or decompensated chronic HF (peripheral oedema/congestion): there is usually a history of progressive worsening of known chronic HF on treatment, and evidence of systemic and pulmonary congestion. Low BP on admission is associated with a poor prognosis.

Pulmonary oedema: patients present with severe respiratory distress and laboured breathing. Many patients with AHF present with a clinical picture and laboratory findings suggestive of increasing left ventricular filling pressures.

Isolated right HF: is characterized by a low output syndrome in the absence of pulmonary congestion with increased jugular venous pressure, with or without hepatomegaly, and low LV filling pressures.

Hypertensive HF: signs and symptoms of HF accompanied by high BP and usually relatively preserved LV systolic function. There is evidence of increased sympathetic tone with tachycardia and vasoconstriction. The patients may be euvolaemic or only mildly hypervolaemic, and present frequently with signs of pulmonary congestion without signs of systemic congestion.

Cardiogenic shock is defined as evidence of tissue hypoperfusion induced by HF after adequate correction of preload and major arrhythmia. There are no diagnostic haemodynamic parameters. However, typically, cardiogenic shock is characterized by reduced systolic blood pressure (SBP: <90 mmHg or a drop of mean arterial pressure > 30 mmHg) and absent or low urine output (<0.5 mL/kg/h). Rhythm disturbance are common. Evidence of organ hypoperfusion and pulmonary congestion is rapidly.
Therapeutic Goals in AHF

Improve patient hemodynamic status to relieve symptoms and stabilize organ function

Reduce fluid volume and filling pressures

Reduce systemic vascular resistance (SVR)

↑ cardiac output (CO)

Reduce neurohormones
Assessment of Hemodynamic Profile

4 Possible Hemodynamic Profiles of AHF

Sign of low perfusion:
- Narrow pulse pressure, cool extremities, sleepy, suspect from ACEI hypotension, low Na, renal worsening

Sign of congestion:
- Orthopnea, elevated JVP, edema, pulsatile hepatomegaly, ascites, rales, louder S3, P2 radiation left ward, abdomino-jugular reflex, Valsava square wave

Low Perfusion?  

<table>
<thead>
<tr>
<th></th>
<th>Warm/Dry</th>
<th>Cold/Dry</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>YES</td>
<td>L</td>
<td>C</td>
</tr>
</tbody>
</table>

Adapted from LW Stevenson
Fluid Challenge

Inotropic drugs:

Diuretic Vasodilator

Warm

Cold

Dry

A

Warm/Dry

Cold/Dry

B

Warm/Wet

Cold/Wet

C

Fluid Challenge
Initial assessment of patient suspected AHF

Suspected acute heart failure

History/examination
(including blood pressure and respiratory rate)
- Chest X-ray
- Echocardiogram or NP (or both)
- Blood chemistry

ECG
Oxygen saturation
Full blood count

Simultaneously assess for
- Ventilation/systemic oxygenation inadequate
- Life-threatening arrhythmia/bradycardia
- Blood pressure <85 mmHg or shock
- Acute coronary syndrome
- Acute mechanical cause/severe valvular disease

Urgent action if present
- Oxygen
- NIV
- ETT and invasive ventilation
- Electrical cardioversion
- Pacing
- Inotrope/vasopressor
- Mechanical circulatory support (e.g. IABP)
- Coronary reperfusion
- Antithrombotic therapy
- Echocardiography
- Surgical/percutaneous intervention

ESC Guideline: For diagnosis and treatment of Acute and chronic HF. 2012
A clinical assessment of patients with AHF

Clinical classifications

Tissue perfusion

Dry and warm

Wet and warm

Dry and cold

Wet and cold

Pulmonary congestion

ESC Guidelines for the Diagnosis and Treatment of Acute and Chronic Heart Failure

www.escardio.org

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ESC Guideline. For diagnosis and treatment of Acute and chronic HF. 2008
Pharmacologic option in AHF

- **Diuretics**: Reduce fluid volume
- **Vasodilators**: Decrease preload and afterload
- **Inotropes**: Augment contractility
- **Natriuretic peptides, ACE, aldactone**: Vasodilate; reduce fluid volume; counteract RAAS/SNS

RAAS = renin-angiotensin-aldosterone system; SNS = sympathetic nervous system
Acute Heart Failure with Systolic Dysfunction

Oxygen/CPAP
Furosemide ± vasodilator

Clinical evaluation (leading to mechanistic therapy)

- SBP > 100 mmHg
  - Vasodilator (NTG, nitroprusside, BNP)
  - Good response
  - Oral therapy
  - Furosemide, ACE-I

- SBP 90-100 mmHg
  - Vasodilator and/or Inotropic (dobutamin PDEI or Levosimendan)
  - No response
  - Reconsider mechanistic therapy
  - Inotropic agent

- SBP < 90 mmHg
  - Volume loading?
  - Inotrope (Dopamin > 5mcg/kg/mnt)
  - And/or norepinephrine

ESC, Acute Heart Failure, 2005
Initial treatment algorithm in AHF

Acute Heart Failure

Immediate symptomatic treatment

Patient distressed or in pain

YES

Analgesia, sedation

Pulmonary congestion

YES

Medical therapy
Diuretic/vasodilator

Arterial oxygen saturation < 95%

YES

Increase FiO₂, Consider CPAP, NIPPV, mechanical ventilation

Normal heart rate and rhythm

NO

Pacing, antiarrhythmics, electroversion

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ESC Guidelines for the Diagnosis and Treatment of Acute and Chronic Heart Failure
Acute pulmonary oedema/congestion

- Intravenous bolus of loop diuretic
  - Hypoxaemia
    - Yes: Oxygen
    - No
  - Severe anxiety/difficulty
    - Yes: Consider i.v. opiate
    - No
  - Measure systolic blood pressure
    - SBP < 85 mmHg or shock
      - Add non-vasoconstricting inotrope
    - SBP 85-110 mmHg
      - No additional therapy until response assessed
    - SBP > 110 mmHg
      - Consider vasodilator (e.g., NTG)
  - Adequate response to treatment?
    - Yes: Continue present treatment
    - No: Re-evaluation of patient's clinical status
      - SBP < 85 mmHg
        - Yes: Stop vasodilator
        - No: Stop beta-blocker if hypoperfused
      - SpO₂ < 90%
        - Yes: Oxygen
        - No: Consider NIV
      - Urine output < 20 mL
        - Yes: Bladder catheterization to confirm
        - No: Increase dose of diuretic or use combination of diuretics
        - Consider low-dose dopamine
        - Consider right-heart catheterization
        - Consider mechanical circulatory support
Acute management

- Oxygen
- Diuretics
- Opiates
- Vasodilators
- Inotropes
- Vasopressor
Diuretics

For achieving **optimal volume status** to eliminate or minimize congestion:

- **High doses** of i.v diuretics 2-3 times daily
- More **effective with continuous i.v.**
- **Combination diuretics**
- **Resistant diuretics** is a common problem
## Indication and dosing of diuretics in AHF

<table>
<thead>
<tr>
<th>Fluid retention</th>
<th>Diuretic</th>
<th>Daily dose (mg)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate</td>
<td>Furosemide or</td>
<td>20–40</td>
<td>Oral or i.v. according to clinical symptoms</td>
</tr>
<tr>
<td></td>
<td>bumetanide or</td>
<td>0.5–1</td>
<td>Titrate dose according to clinical response</td>
</tr>
<tr>
<td></td>
<td>torasemide</td>
<td>10–20</td>
<td>Monitor K, Na, creatinine, blood pressure</td>
</tr>
<tr>
<td>Severe</td>
<td>Furosemide</td>
<td>40–100</td>
<td>i.v. Increase dose</td>
</tr>
<tr>
<td></td>
<td>Furosemide infusion</td>
<td>(5–40 mg/h)</td>
<td>Better than very high bolus doses</td>
</tr>
<tr>
<td></td>
<td>Bumetanide</td>
<td>1–4</td>
<td>Oral or i.v.</td>
</tr>
<tr>
<td></td>
<td>Torasemide</td>
<td>20–100</td>
<td>Oral</td>
</tr>
<tr>
<td>Refractory to loop diuretic</td>
<td>Add hydrochlorothiazide</td>
<td>50–100</td>
<td>Combination better than very high dose of loop diuretics</td>
</tr>
<tr>
<td></td>
<td>or metolazone</td>
<td>2.5–10</td>
<td>More potent if creatinine $\text{cl}r &lt; 30 \text{ ml/min}$</td>
</tr>
<tr>
<td></td>
<td>or spironolactone</td>
<td>25–50</td>
<td>Spironolactone best choice if no renal failure and normal or low K</td>
</tr>
<tr>
<td>With alkalosis</td>
<td>Acetazolamide</td>
<td>500</td>
<td>i.v.</td>
</tr>
<tr>
<td>Refractory to loop diuretics and thiazides</td>
<td>Add dopamine (renal vasodilation) or dobutamine</td>
<td></td>
<td>Consider ultrafiltration or haemodialysis if co-existing renal failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hyponatraemia</td>
</tr>
</tbody>
</table>
Morphine and its analogues

In patient present with restlessness and dyspnoea

Morphine induces
• Venodilatation
• Mild arterial dilatation
• Reduce heart rate

Dose: 3 mg IV bolus, rate 1 mg/min. Repeated if required
Vasodilators
Nitroprusside, Nitroglycerin, Nitrate family

- Work by cGMP mediated smooth muscle relaxation -> vasodilation
- Decrease myocardial work by afterload and preload reduction
- May cause hypotension
- May cause headache
Intravenous Vasodilator used to treat AHF

<table>
<thead>
<tr>
<th>Vasodilator</th>
<th>Dosing</th>
<th>Main side effects</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerine</td>
<td>Start with 10–20 μg/min, increase up to 200 μg/min</td>
<td>Hypotension, headache</td>
<td>Tolerance on continuous use</td>
</tr>
<tr>
<td>Isosorbide dinitrate</td>
<td>Start with 1 mg/h, increase up to 10 mg/h</td>
<td>Hypotension, headache</td>
<td>Tolerance on continuous use</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>Start with 0.3 μg/kg/min and increase up to 5 μg/kg/min</td>
<td>Hypotension, isocyanate toxicity</td>
<td>Light sensitive</td>
</tr>
<tr>
<td>Nesiritide</td>
<td>Bolus 2 μg/kg + infusion 0.01 μg/kg/min</td>
<td>Hypotension</td>
<td></td>
</tr>
</tbody>
</table>

*Not available in many European Society of Cardiology countries.*
Nitrate

- Not evaluated by large scale studies
- Many studies shown their favorable effect
- Limitation
- Side effect
- Nitrate Resistance
- Nitrate Tolerance
  - Prevention
  - Intermittent dosing: 12 hour nitrate free interval
  - Escalating dose
  - Concomitant use of hydralazine
Role of Inotropic Therapy in Acute Heart Failure

The use of inotropes as a treatment of:

- cardiogenic shock
- diuretic/ACE inhibitor– refractory heart failure decompensations
- a short-term bridge to definitive treatment, such as revascularization or cardiac transplantation, is potentially appropriate
Inotropic Agent

Indication:
Peripheral hypoperfusion (hypotension, decrease renal function) with or without congestion

Patients with CHF:
Clinical course, symptom and prognosis may depend on haemodynamics parameter

Improvement of haemodynamics may become a goal of treatment

Beneficial effect of improvement haemodynamics potentially counteract by the rise of arrythmia (increase oxygen demand, Ca^{++} loading, excessive increase in energy) may potentially harmful

ESC, Acute Heart Failure, 2012
Inotropes:
Dopamine, Dobutamine, Milrinone

- **Improve cardiac output**
  - by directly increasing cardiac contractility
- **Significant proarrhythmic effects**
- **May precipitate ischemia**
- **Not recommended for routine use** in AHF, but clearly have a role in specific patients
Inotropic Agents

Dopamine

- Is dose dependent and they involve in three different receptors.

- **In low dose (< 2 µg/kgBW/min),**
  - *vasodilatation* occurs predominantly in renal, coronary, and cerebral vascular beds.

- At doses > 5 µg/kgBW/min dopamine
  - will increase peripheral vascular resistance via α adrenergic receptors

- However if no response is seen in diuresis the therapy **should be terminated**
  (Level of evidence C, class IIb)

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ESC, Acute Heart Failure, 2005
Inotropic Agents

**Dobutamine**

- Clinical action is dose dependent positive inotropic and chronotropic effects.
- In low dose induce arterial vasodilatation and in higher induce arterial vasoconstriction.

ESC, Acute Heart Failure, 2005
Inotropic Agents

Phosphodiesterase inhibitors

- Block the breakdown of cyclic AMP into AMP (milrinone, enoximone)
- In advance HF, associated with inotropic, lusitropic, vasodilating effects
- Intermediate between vasodilator and predominant inotrope
Drugs used to treat AHF that are positive inotropes or vasopressor or both

<table>
<thead>
<tr>
<th>Drug</th>
<th>Bolus</th>
<th>Infusion rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine</td>
<td>No</td>
<td>2–20 μg/kg/min (β⁺)</td>
</tr>
<tr>
<td>Dopamine</td>
<td>No</td>
<td>&lt;3 μg/kg/min; renal effect (β⁺)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3–5 μg/kg/min; inotrop (β⁺)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt;5 μg/kg/min; (β⁺), vasopressor (α⁺)</td>
</tr>
<tr>
<td>Milrinone</td>
<td>25–75 μg/kg over 10–20 min</td>
<td>0.375–0.75 μg/kg/min</td>
</tr>
<tr>
<td>Enoximone</td>
<td>0.5–1.0 mg/kg over 5–10 min</td>
<td>5–20 μg/kg/min</td>
</tr>
<tr>
<td>Levosimendan²</td>
<td>12 μg/kg over 10 min (optional)³</td>
<td>0.1 μg/kg/min, which can be decreased to 0.05 or increased to 0.2 μg/kg/min</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>No</td>
<td>0.2–1.0 μg/kg/min</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Bolus: 1 mg can be given i.v. during resuscitation, repeated every 3–5 min</td>
<td>0.05–0.5 μg/kg/min</td>
</tr>
</tbody>
</table>
After stabilization

- ACE-I
- Beta blocker
- Minelarocorticoid receptor inhibitor
- Digoxin
- Device therapy
Conclusion

Rapid assessment and treatment of ADHF could decreased mortality and morbidity rate

Management strategies including

- Ensure oxygenation
- Reduce pain
- Reduce fluid volume
- Reduce preload and or afterload
- Increase cardiac output
- Identify and treat the cause of CHF
Terima kasih!