TREATMENT OF HEART FAILURE AND CARDIOGENIC SHOCK

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ในปัจจุบัน อัตราการเสียชีวิตจากภาวะ Acute MI ?

- 40-50%
- B. 60-70%
- C. 70-80%
- D. 80-90%
OVERVIEW OF TOPICS

- Hemodynamic disturbances
  - Heart failure (HF)
  - Cardiogenic shock
  - RV infarction
- Cardiac complications e.g. cardiac rupture
- Management of HF following ST-segment elevation MI
HEART FAILURE

- Development of HF after STEMI is an indication for angiography with revascularization needed.

- LV myocardium may be ischemic, stunned, hibernating, or injured.

- Viability assessment may be needed depending on timing of revascularization.

- Ischemic (functional) mitral regurgitation due to LV remodeling may progress over time, and may require surgery.
ACUTE DECOMPENSATED HF (ADHF)

**Definition**

- Rapid onset of S&S due to abnormal cardiac function
- With or without previous cardiac disease
- Systolic or diastolic dysfunction
- Often life threatening or require urgent Rx
Right heart failure

- low output syndrome
- JVD, hepatomegaly
- hypotension

LV dysfunction is the strongest predictor of mortality following STEMI
Heart Failure Pathophysiology

Myocardial injury → Fall in LV performance

Activation of RAAS, SNS, endothelin, vasopressin, cytokines, etc.

Myocardial toxicity → ANP, BNP → Peripheral vasoconstriction

Remodeling and progressive worsening of LV function → Heart failure symptoms

Morbidity and mortality

Pathophysiologic Role of RAAS

Angiotensinogen → Renin → Angiotensin I → Angiotensin II

- Chymase cathepsin G converting Angiotensin I to Angiotensin II
- ACE converting Angiotensin II to Bradykinin (VD)
- Conversion of Angiotensin II to Inactive fragments

Angiotensin II results in:
- Vasoconstriction
- ↑ Vasopressin
- ↑ Aldosterone
- ↑ SNS Activity

Results in:
- Na⁺ and H₂O Retention
Pathophysiologic Effects of Aldosterone

Blood Vessels
- Endothelial dysfunction
- Vascular inflammation
- HTN, atherosclerosis

Kidneys
- Na⁺ retention
- K⁺ and Mg²⁺ excretion
- Renal fibrosis
- HTN, ESRD, HF, sudden death

Heart
- Ventricular hypertrophy
- Myocardial fibrosis
- Ventricular remodeling
- ↑ SNS activity
- HF, sudden death, MI

Brain
- Vascular damage
- Baroreceptor dysfunction
- HTN, stroke

Pathophysiologic Effects of the Sympathetic Nervous System

Myocardial injury → ↑ SNS activity

- ↑ RAAS and vasopressin
- Vasoconstriction
- ↑ Heart rate and contractility
- Direct cardiotoxicity

- ↑ Wall stress

- ↑ Na⁺ and H₂O retention

↑ Myocardial O₂ demand → Myocyte damage, ↓ contractility, myocardial hypertrophy

Pathophysiologic Effects of Endothelin-1

- **Blood Vessels**
  - Potent VC
  - Collagen formation
  - Nitric oxide release (ETα)
  - ↓ Renal blood flow

- **Kidneys**
  - Afferent/effferent VC

- **Heart**
  - ↑ Heart rate
  - ↓ Contractility
  - Ventricular hypertrophy
  - Ventricular remodeling

- **Neurohormonal**
  - ↑ SNS
  - ↑ Angiotensin II
  - ↑ Aldosterone
  - ↑ Vasopressin

Pathophysiologic Effects of AVP

Blood Vessels
- Potent VC ($V_{Na}$)

Kidneys
- Free H2O reabsorption ($V_{2}$)
- Dilutional hyponatremia

Heart
- ↓ Contractility
- Ventricular hypertrophy

Physiologic Effects of Natriuretic Peptides

**Blood Vessels**
- VD (arterial/venous)
- Inhibits vascular smooth muscle proliferation

**Kidneys**
- ↑ Na⁺ and H₂O excretion

**Heart**
- Coronary artery VD
- Myocardial relaxation
- ↓ Myocardial fibrosis

**Neurohormonal**
- ↓ Angiotensin II
- ↓ Aldosterone
- ↓ SNS
- ↓ Endothelin

Release of C-type natriuretic peptides from vascular endothelium

Release of B-type natriuretic peptides from ventricles

Release of A-type natriuretic peptides from atria

Suppression of renin–angiotensin and endothelin

Decreased peripheral vascular resistance (decreased blood pressure)

Increased natriuresis
Degree of HF following MI:

- Class I - no HF (no rales, no S3 gallop)
- Class II - pulmonary congestion with rales < 50% of lung fields, sinus tachycardia, or S3 gallop
- Class III - pulmonary edema with rales > 50% of lung fields
- Class IV - cardiogenic shock
HEMODYNAMIC ASSESSMENT

Based on:

- physical examination
- continuous ECG monitoring
- oxygen saturation
- BP monitoring
- hourly urine output
INVESTIGATION

- Transthoracic echocardiography/ Doppler*
  - LV function assessment
  - look for mechanical complications
- CXR
RV INFARCTION

- found in 1/3 of inferior STEMI patients.
- most often due to proximal occlusion of RCA, and is associated with a higher mortality risk.
- **Clinical Triad**: hypotension, clear lung fields, and elevated JVP
- **ECG** marker of RV injury*
MOST SENSITIVE ECG*
CARDIOGENIC SHOCK

- complicates 6-10% of all STEMI cases, remains a leading cause of death, with hospital mortality rates 50%.

- For those with pump failure, 15% occur at time of presentation, and 85% during hospitalization.

- often develops early after the onset of acute MI, within 6 hrs in 50% and within 24 hrs in 75% (data from SHOCK trial registry).

- typically present with “cold and wet”
CLASSIC SHOCK PARADIGM
Hemodynamic Subsets in ADHF

HF

CARDIOGENIC SHOCK

Subset I (normal) - "Warm & Dry"
Subset II (congestion) - "Warm & Wet"
Subset III (hypoperfusion) - "Cold & Dry" 5%
Subset IV (hypoperfusion and congestion) - "Cold & Wet" 28%

Cardiac Index (L/min/m²)

Pulmonary Capillary Wedge Pressure (mm Hg)

HEMODYNAMIC CRITERIA: CARDIOGENIC SHOCK

- cardiac index < 2.2 l/min/m²
- PCWP > 18 mmHg
- diuresis usually < 20 ml/h
- inotropes and/or an IABP is needed to maintain SBP > 90 mmHg
LV FAILURE IS MOST COMMON ETIOLOGY
Baseline and follow-up stroke volume index --> the most powerful predictor of 30-day mortality.

Cardiogenic shock management do not necessarily need invasive measurement of LV filling pressure and CO, but LVEF and mechanical complications should be assessed by TTE.
MECHANICAL COMPLICATIONS

- Bimodal, temporal distribution: most occur in first 24 hrs, and remainder present within 1st week.

- The new systolic murmur indicates either ventricular septal rupture (VSR) or mitral regurgitation (MR).
MITRAL REGURGITATION

- 2 mechanisms:
  - papillary muscle rupture
  - postinfarction LV remodeling with displacement of pap. muscles, leaflet tethering, and annular dilatation

- acute rupture affects PM papillary muscle (single blood supply)
- acute severe MR --> pulmonary edema and/or shock
VENTRICULAR SEPTAL RUPTURE

- usually heralded by a loud systolic murmur and HF or shock, depending on defect size and degree of RV/LV dysfunction.

- **GUSTO-1, SHOCK registry**: occurs most often within 24 hrs in STEMI who treated with fibrinolytic Rx, incidence 0.2% with 30-day mortality 73-87%, and high surgical mortality 83%
LV FREE-WALL RUPTURE

- characterized by recurrent chest pain and ST-T changes.
- rapid progression to hemodynamic collapse, arrhythmias, and death
- occur most frequent in first MI, anterior infarction, elderly, and women.
- Other risk factors: HT during acute phase of MI, absence of collateral blood flow, Q wave ECG, steroid or NSAIDs use, received fibrinolytics > 14 hrs after onset.
- Pseudoaneurysm with contained rupture* was formed.
LV ANEURYSM

- occurs in < 5% of patients, more frequent in anterior infarction.

- LV thrombi should be treated with anticoagulant therapy.
MANAGEMENT OF HF & CARDIOGENIC SHOCK
Initial assessment of patient with suspected acute heart failure

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
- Echo-cardiography
- Surgical / percutaneous intervention

European Heart Journal (2012) 33, 1787–1847
European Journal of Heart Failure (2012) 14, 803–869
KILLIP CLASS II (MILD HF)

- loop diuretics and/or i.v. nitrates to reduce preload and relieve congestion/dyspnea.
- HT : should be treated promptly with ACEI/ARBs/AAs* to improve survival.
KILLIP CLASS III (MODERATE HF)

- morphine reduces dyspnea and anxiety

- i.v.loop diuretics and/or i.v.vasodilators (use in non-hypotension cases, SBP > 90 mmHg).

- non-invasive ventilation with CPAP (if tolerate), or ET-intubation with ventilatory support.

- Choices of inotropes/vasopressor: dopamine or dobutamine or levosimendan (if SBP > 90 mmHg).

- Ultrafiltration: refractory to diuretics (esp. hyponatremia)
KILLIP CLASS IV (SEVERE HF WITH SHOCK)

- try to detect **alternative causes of hypotension** e.g. hypovolemia, drug-induced, arrhythmias, tamponade, mechanical complications or RV infarction.

- aim to maintain SBP > 90 mmHg and adequate cardiac output and renal perfusion.
Algorithm for management of acute pulmonary oedema/congestion

1. Intravenous bolus of loop diuretic

2. Hypoxaemia
   - Yes: Oxygen
   - No:
     - Severe anxiety/distress
       - Yes: Consider i.v. opiate
       - No: Measure systolic blood pressure

3. Measure systolic blood pressure
   - SBP < 85 mmHg or shock: Add non-vasodilating inotrope
   - SBP 85-110 mmHg: No additional therapy until response assessed
   - SBP > 110 mmHg: Consider vasodilator (e.g. NTG)
Algorithm for management of acute pulmonary oedema/congestion

1. **Acute pulmonary oedema / congestion**
   - Intravenous bolus of loop diuretic
     - Hypoxaemia
       - Oxygen
       - Yes
       - No
       - Severe anxiety/distress
         - Consider i.v. opiate
         - Yes
         - No
         - Measure systolic blood pressure
           - SBP <85 mmHg or shock
             - Add non-vasodilating inotrope
           - SBP 85-110 mmHg
             - No additional therapy until response assessed
           - SBP >110 mmHg
             - Consider vasodilator (e.g. NTG)

2. **Adequate response to treatment**
   - Yes
     - Continue present treatment
   - No
     - Re-evaluation of patient's clinical status
       - SBP <85 mmHg
         - Stop vasodilator
         - Stop beta-blocker if hypoperfused
         - Consider non-vasodilating inotrope or vasopressor
         - Consider right-heart catheterization
         - Consider mechanical circulatory support
       - SpO₂ <90 %
         - Oxygen
         - Consider NIV
         - Consider ETT and invasive ventilation
       - Urine output <20 mL/h
         - Bladder catheterization to confirm
         - Increase dose of diuretic or use combination of diuretics
         - Consider low-dose dopamine
         - Consider right-heart catheterization
         - Consider ultrafiltration

used due to their favourable hemodynamic effects.

Norepinephrine is the first choice (use lowest dose, and keep SBP at least 80 mmHg).

*from a recent RCT trial: Dopamine was associated with higher mortality in cardiogenic shock subgroup and more adverse events (mainly arrhythmic events) than Norepinephrine.

none has produced consistent symptomatic improvement,
<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatment of mild heart failure (Killip class II)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxygen is indicated to maintain a saturation &gt;95%.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Loop diuretics, e.g. furosemide: 20–40 mg i.v., is recommended and should be repeated at 1–4 h intervals if necessary.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>i.v. nitrates or sodium nitroprusside should be considered in patients with elevated systolic blood pressure.</td>
<td>IIa</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>An ACE inhibitor is indicated in all patients with signs or symptoms of heart failure and/or evidence of LV dysfunction in the absence of hypotension, hypovolaemia, or renal failure.</td>
<td>I</td>
<td>A</td>
<td>309–312</td>
</tr>
<tr>
<td>An ARB (valsartan) is an alternative to ACE inhibitors particularly if ACE inhibitors are not tolerated.</td>
<td>I</td>
<td>B</td>
<td>281</td>
</tr>
<tr>
<td>An aldosterone antagonist (epironone) is recommended in all patients with signs or symptoms of heart failure and/or evidence of LV dysfunction provided no renal failure or hyperkalaemia.</td>
<td>I</td>
<td>B</td>
<td>282</td>
</tr>
<tr>
<td>Hydralazine and isosorbid dineitrates should be considered if the patient is intolerant to both ACE inhibitors and ARBs.</td>
<td>IIa</td>
<td>C</td>
<td>313</td>
</tr>
<tr>
<td><strong>Treatment of moderate heart failure (Killip class III)</strong></td>
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<tr>
<td>Oxygen is indicated.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Ventilatory support should be instituted according to blood gases.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Loop diuretics, e.g. furosemide: 20–40 mg i.v., are recommended and should be repeated at 1–4 h intervals if necessary.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Morphine is recommended. Respiration should be monitored. Nausea is common and an antiemetic may be required. Frequent low-dose therapy is advisable.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Nitrates are recommended if there is no hypotension.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Inotropic agents:</td>
<td></td>
<td></td>
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<tr>
<td>• Dopamine</td>
<td>IIa</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>• Dobutamine (inotropic)</td>
<td>IIa</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>• Levosimendan (inotropic/vasodilator).</td>
<td>IIb</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>An aldosterone antagonist such as spironolactone or epleronone must be used if LVEF ≤40%.</td>
<td>I</td>
<td>B</td>
<td>282, 314</td>
</tr>
<tr>
<td>Ultrafiltration should be considered.</td>
<td>IIa</td>
<td>B</td>
<td>315</td>
</tr>
<tr>
<td>Early revascularization must be considered if the patient has not been previously revascularized.</td>
<td>I</td>
<td>C</td>
<td></td>
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</table>
SHOCK trial - STEMI with cardiogenic shock, undergoing emergency PCI or CABG have improved long-term survival, compared with initial intensive medical therapy followed by no-or late in-hospital revascularization.
Treatment of Cardiogenic Shock

Emergency revascularization with either PCI or CABG is recommended in suitable patients with cardiogenic shock due to pump failure after STEMI irrespective of the time delay from MI onset.

In the absence of contraindications, fibrinolytic therapy should be administered to patients with STEMI and cardiogenic shock who are unsuitable candidates for either PCI or CABG.
Treatment of Cardiogenic Shock

The use of intra-aortic balloon pump counterpulsation can be useful for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacological.

Alternative LV assist devices for circulatory support may be considered in patients with refractory cardiogenic shock.
IABP COUNTERPULSATION

Picture courtesy: http://www.eonet.ne.jp/~hidarite/ce/sinpai06.html
from TACTICS trial, and another small pilot trial: no benefit in short term mortality outcome, but showed a favourable decrease in 6-month mortality for more severe hemodynamic impairment in IABP group.

LVADs: benefit evidence is limited.
TREATMENT OF RV INFARCTION

- Maintenance of RV preload*
- Reduction of RV afterload
- Inotrophic support (if needed)
- Immediate reperfusion
- **Should avoid Nitrates & Diuretics**
- Cardiac pacing (if AV block) or cardioversion (if AF)
MANAGEMENT OF MECHANICAL COMPLICATIONS

- All patients with mechanical MR and subacute rupture should be emergently considered for surgical intervention.

- Superior surgical outcome will be gained when performed before onset of cardiogenic shock.