Acute MI Complicated by Cardiogenic Shock

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Disclosures

- NONE
- Except to say that...

Objectives

- Cardiogenic Shock (CS)
  - Definitions
  - Incidence/prognosis
  - Diagnosis
  - Causes of CS
    - Acute MI
    - Mechanical complications of MI
    - Others
  - Management of CS
    - Pharmacologic and supportive
    - Mechanical circulatory support (MCS)
    - New directions for improving survival
Definition

- Clinical syndrome manifested by a sudden reduction in cardiac output, leading to systemic hypotension and end-organ hypoperfusion.

Incidence and Prognosis

- Most common cause of death in patients hospitalized with AMI
  - Mortality rates ~40-50%
- Complicates ~5-8% of STEMI, 2-5% of NSTEMI cases
  - ~40,000-50,000 cases annually in the US
- National Cardiovascular Data Registry (NCDR)
  - CS in STEMI: ~33.1% mortality
  - CS in NSTEMI: ~40.8% mortality
- ACTION Registry-GWTG
  - Hospital survivors of AMI + CS
  - Higher risk of death and/or hospitalisation during the 1st yr post-discharge

Pathogenesis

- Classic Panorgan and the Role of Systemic Inflammation in the Pathogenesis of Cardiogenic Shock
More common to develop CS after hospitalization
National Registry of Myocardial Infarction (NRMI)
Collects prospective outcomes on AMI patients in US
Only 29% of CS patients in shock on presentation
SHOCK Trial
Only 9% of CS patients in shock on arrival
Median time to development of CS: 4.6 hrs
What causes appearance of CS later?
- Myocardial reinfarction
- Recurrent ischemia
- Mechanical complications
- Infarct expansion

Clinical Presentation
Classic syndrome
- Systemic hypotension
  - SBP <90 mmHg or MAP 30 mmHg below baseline
- Systemic hypoperfusion
  - Oliguria, cool extremities, altered mental status, etc.
- Pulmonary congestion
- Variable presentation more common
Lab findings

- ECG
  - Evidence of ischemia
- CXR
  - Pulmonary congestion
- BMP
  - Metabolic acidosis, elevated lactate, decreased serum bicarbonate
  - Renal hypoperfusion - increased BUN and Cr
- Echocardiogram
  - LV dysfunction and/or mechanical complications

Ventricular Septal Defect

- Traditionally ~2.2% of AMI cases
- Improved reperfusion - ~0.2% of cases
- Usually within 7 days
- Higher incidence in woman and elderly
- Suspect when rapid evolution from normal to depressed EF
- Can be asymptomatic for months or years
- Pulmonary congestion not prominent
- Echocardiography
- Inotropes and/or inotropic support to maintain MAP
- IABP to reduce afterload and magnitude of left-to-right shunting
- Definitive treatment
  - Surgical repair (high operative mortality)
  - 53 vs 6 percent mortality (medical vs surgical)

Ventricular Septal Rupture

- Traditionally ~1-2% of AMI cases
- Improved reperfusion - ~0.2% of cases
- Usually within 7 days
- Higher incidence in woman and elderly
- Suspect when rapid evolution from normal to depressed EF
- Can be asymptomatic for months or years
- Pulmonary congestion not prominent
- Echocardiography
- Inotropes and/or inotropic support to maintain MAP
- IABP to reduce afterload and magnitude of left-to-right shunting
- Definitive treatment
  - Surgical repair (high operative mortality)
  - 53 vs 6 percent mortality (medical vs surgical)
**Papillary Muscle Rupture**
- Posteromedial papillary muscle rupture most common
- Dual blood supply of anterolateral papillary muscle
- Inferior wall infarct leads to PMR rupture and severe MR
- ~5% of deaths in AMI
- Severe pulmonary edema
- New holosystolic murmur (generally no thrill)
- Echo for Dx

**Initial therapy**
- Afterload reduction (sodium nitroprusside) and IABP
- Decreased regurgitant fraction and improved forward flow

**Definitive treatment**
- Surgical repair or replacement (20-25% mortality)

**Free Wall Rupture**
- Varying incidence in studies, 1-4%
- 11.2% of deaths from AMI
- Sudden hypotension from pericardial effusion/tamponade
- Hypotension, elevated JVP, pulsus paradoxus
- Echo for Dx

**Initial therapy**
- Pericardiocentesis to stabilize

**Definitive treatment**
- Surgical repair
Revascularization for AMI complicated by CS

- 2013 ACC/AHA STEMI Guidelines
- **CLASS I**
  - Emergency revascularization with either PCI or CABG is recommended in suitable patients with cardiogenic shock due to pump failure and/or shock irrespective of the time delay from MI onset (Level of Evidence: B)
- ~70-80% of patients with CS have **multivessel** disease
- Higher mortality compared with single vessel
- Does multivessel PCI improve outcomes?

### CULPRIT-SHOCK Trial

706 patients with AMI + COⅢ + Multivessel disease
Culprit lesion only vs Multivessel PCI
Primary Endpoint: Composite of death or renal-replacement therapy at 30 days
Lower Death (p=0.03) and Lower RRT (p=0.07) with culprit only PCI
_NEJM 2017_
Management Options

- Pharmacologic therapy
- Antiplatelet and antithrombotic agents
- Avoid beta-blockers (negative inotropes)
- Avoid nitroglycerin (vasodilators)
- Vasopressors for initial circulatory support
  - Dopamine - positive inotrope and vasodilator (tachycardia)
  - Norepinephrine - potent vasoconstrictor (increased SVR)
  - Dobutamine - positive inotrope (peripheral vasodilation)

Table 4: Mechanism of Action and Hemodynamic Effects of Common Vasopressor Medications in CS

<table>
<thead>
<tr>
<th>Medication</th>
<th>Vasopressor Dose</th>
<th>Beta Effect</th>
<th>Alpha Effect</th>
<th>Dopaminergic Effect</th>
<th>Hemodynamic Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>0.5-5 mcg/kg/min</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+ (positive inotrope)</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>10-50 mcg/kg/min</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+ (positive inotrope)</td>
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</tbody>
</table>

Note: CS indicates coronary sinus, CS, cardiogenic shock; Pgp, P-glycoprotein; SVR, systemic vascular resistance; and LV, systemic vascular resistance.
What other options do we have?

- **2013 ACC/AHA STEMI Guidelines**
  - **Class Ia**
    - The use of intra-aortic balloon pump (IABP) counterpulsation can be useful for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacological therapy. (Level of Evidence: B)
  - **Class Ib**
    - Alternative LV assist devices for circulatory support may be considered in patients with refractory cardiogenic shock. (Level of Evidence: C)

Intra-aortic Balloon Counterpulsation Pump (IABP)

- STEMI and Non-STEMI
- 45% Anterior infarct
- CO/S: hypotension / poor perfusion
- IABP use pre- or post PCI
- Median LVEF : 36%
- No PA-catheter indices
Impella Trials

- **ISAR-SHOCK**
  - Randomized trial comparing Impella 2.5 vs IABP in 26 CS patients
  - Greater increase in cardiac index with Impella
  - Similar mortality
- **Uspella Registry**
  - 154 PCI patients
  - Early use of Impella 2.5 associated with more complete revascularization and improved survival in AMI with CS
Exhausting Coronary Approaches for a Ventricular Problem?

AMI Shock Mortality Unchanged in >20 years

Hemodynamic Efficacy Without Clear Clinical Benefit

Acute MCS: A Field in its Infancy

What are we missing?

This is why we ask for a minimum of 2 views
"Comfort is the Enemy of Progress"
- P.T. Barnum

2013 STEMI Guidelines:
Class I
EMS transport directly to a PCI-capable hospital for primary PCI is the recommended strategy for patients with STEMI, with an ideal FMC-to-device time system goal of 90 minutes or less. (Level of Evidence: B)

More Rapid Balloon Angioplasty is NOT the answer
We’ve maximized the benefits of DTB

Does Size Matter?

Are There Other Ways to Limit Infarct Size?

Myocardial O2 Supply
- Coronary Occlusion
- Multivessel Disease
- Microvascular Dysfunction
- Systemic Hypotension
- Collateral Blood Flow

Myocardial O2 Demand
- Heart Rate
- LV Wall Stress
- LV Stroke Work

Factors Influencing Infarct Size Following Experimental Coronary Artery Occlusions

Of greatest interest, from the clinical point of view, is the finding that the severity and extent of myocardial ischemic injury resulting from coronary occlusion could be radically altered not only by pretreatment of the animal but also by an appropriate intervention as late as 3 hr after the coronary occlusion. This suggests that measures designed for reduction of myocardial oxygen demands and improvement of coronary perfusion, when effected promptly after a patient has been brought to a hospital, might potentially reduce the ultimate size of the infarction.
Unloading Impact 1
Reduced LV Wall Stress and Myocardial O2 Consumption

Unloading Impact 2
Reduced LA Pressure and RV Afterload

Unloading Impact 3
LV Unloading Increases Coronary Collateral Blood Flow
Unloading Impact 4
Unloading Promotes Protective Myocardial Signaling

KEY OBJECTIVES:
- Reduce exposure to high dose inotropes
- Impella use prior to PCI
- Protocol using hemodynamic monitoring to guide escalation and weaning

GOAL
- Improve outcomes in CS with standardized protocols

40 Years of LV Unloading Science

Unloading before not after reperfusion is required to reduce infarct size

Detroit Cardiogenic Shock Initiative
DSCI

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Paradigm Shift

- Time to rethink quality metrics
- Should Doorto Unload replace Doorto Balloon
- Can delaying reperfusion with unloading first reduce infarct size

Future Directions

[Legend and data graph]

Door To Unload: STEMI Safety & Feasibility Trial
Anterior STEMI Referred for Primary PCI (WITHOUT SHOCK)

Inclusion Criteria:

- Age ≥ 18
- Non-ST elevation MI
- High cardiac urgency
- No sign of shock
- ST-segment elevation > 2 mm in ≥ 2 adjacent or > 1.5 mm LAD

Exclusion Criteria:

- Contraindication to PCI
- Active coagulopathy
- Prior PCI in the same leg
- Revascularization failure

Procedure:

1. Unloading Time
   - Impella CP per protocol
   - 30 min protocol
2. Unloading Time
   - Impella CP per protocol
   - 24 h to 48 h

Procedure:

- Impella CP after a minimum of 3 hours support.
Thanks for Your Attention!

The best way to a man's heart is through the groin
- The Cath lab