Project: Ghana Emergency Medicine Collaborative

Document Title: Achy Breaky Heart: Cardiogenic Shock, A Historical Perspective and Current Therapy Guidelines

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Objectives

- Fulfill a requirement for graduation
- Present a case that we can all learn from
- Discuss the various treatment options available for cardiogenic shock
- Discuss what we can do in the ED to potentially increase survivability
Case Presentation

- CC: Chest pain, Shortness of breath
- HPI: 44 y.o. M unknown PMH, chest pain and SOB for 2 days. Worsening dyspnea. Brought in by family. Difficult to obtain history secondary to DIB and language barrier.
Vitals

- HR: 167
- BP: 89/64
- RR: 37
- SaO2: 99% NRB
- Temp: NR
Physical Exam

- **General:** Overweight gentleman, visibly short of breath, agitated, unable to sit still.
- **Cardiovascular:** Irregularly irregular. Tachycardic. No murmurs, rubs, or gallops appreciated. No JVD. Rapid but palpable radial pulses present.
- **Pulmonary:** Diffusely decreased air entry bilaterally with minimal wheezing noted.
Physical Exam

• Extremities: Warm, well-perfused. No evidence of lower extremity edema or swelling.

• Neurologic: Awake, alert, speaking to family members in 1-2 word sentences. Mostly nodding or shaking head to questions.
Lab work

CBC

16.9

9.2 >---< 258

48.2

Basic

140 102 12

--------- | --------- | --------- < 118

4.1 26 0.98
Lab work

Myoglobin 163.7 ng/mL
Troponin 1.18 ng/mL

BNP 161 picogram/mL
D-dimer < 200 ng/mL

ABG: 7.31/42/304/21
Repeat: 7.21/51/168/20
Therapies

- IV fluids
- Anti-arrhythmics
- Pressors
- BiPap
- Intubation
- Echocardiogram
- CT scan
- Cath lab
Phone a friend

a. Call your attending
b. Call the cardiologist
c. Call the cardiothoracic surgeon
d. Call your mother
Differential Diagnosis of Chest Pain and SOB
Differential Diagnoses (limited)

- MI
- Tension PTX
- Aortic dissection
- PE
- Cardiac tamponade
- Ruptured viscus
- Valvular abnormalities (mitral/aortic stenosis)
Some of the Many Causes of Cardiogenic Shock

- MI (most common)
- Aortic dissection
- PE
- Cardiac tamponade
- Ruptured viscus
- Hemorrhage
- Sepsis
- Cardiomyopathy (restrictive or dilated), myocarditis
- Medication overdose (beta/calcium-channel blockers)
- Cardiotoxic drugs (doxorubicin)
- Electrolyte abnormalities (calcium, phosphate)
- Valvular abnormalities (mitral/aortic stenosis)
- Papillary muscle or ventricular free wall rupture
A Lil’ History

• 1700s: Shock first defined as a sequelae of severe trauma

• 1935, 1940: Harrison and Blalock classified types of shock

• 1950: Treatment of CS with O₂, phlebotomy, morphine. Also in favor was ethyl alcohol vapor, digitalis, quinidine

• 1960: Introduction of CCUs; improvement in mortality from arrhythmia, but not CS

• 1962: First IABP designed

• 1968: IABP placed by Dr. Kantrowitz in 5 patients with CS
Cardiogenic Shock

• 5-15% of ACS cases
• Small percentage with NSTEMI have CS (GUSTO II-B, PURSUIT trials)
• Loss of 40% of ventricular muscle mass
• Myocytes adjacent to infarct are susceptible to expanding ischemia
Risk Factors for Developing CS

• Older age
• Multivessel CAD
• Anterior MI location
• STEMI or LBBB
• HTN
• DM
• Prior MI
• Prior CHF
Diagnosing CS

• Clinically
  – SBP <90mmHg
  – HR >100 beats/min
  – RR >20 breaths/min (Paco$_2$<32 mm Hg)
  – Evidence of hypoperfusion
  – C.I <2.2L/min/m$^2$
  – LVEDP or PCWP >15mmHg

• Echocardiogram
Treatment for Cardiogenic Shock

- ABCs still take precedence
- 250-mL saline boluses over 5 to 10 minutes.
- Vasopressors or inotropic support
- Revascularization
- Consider IABP for refractory shock
Box 1. Conventional therapy for cardiogenic shock

Maximize volume (right atrial pressure 10 to 14 mm Hg, PCWP 18 to 20 mm Hg)
Maximize oxygenation (eg, ventilator)
Control rhythm (eg, pacemaker, cardioversion)
Correct electrolyte and acid–base imbalances
Sympathomimetic amines (eg, dobutamine, dopamine, norepinephrine, phenylephrine)
Phosphodiesterase inhibitors (eg, milrinone)
Vasodilators (eg, nitroglycerin, nitroprusside)
Diuretics (eg, furosemide)
Antiarrhythmics (eg, amiodarone)
Intra-aortic balloon counterpulsation
Clinical Signs: Shock, Hypoperfusion, CHF, Acute Pulm Edema
Most likely major underlying disturbance?

- Acute Pulmonary Edema
  - Administer
    - Furosemide
    - Morphine
    - Oxygen intubation
    - Nitroglycerin
    - Dopamine
    - Dobutamine

- Hypovolemia
  - Administer
    - Fluids
    - Blood transfusions
  - Cause-specific interventions

- Low-output cardiogenic shock
  - Check Blood Pressure
    - Systolic BP (>100 mm Hg)
      - Nitroglycerin
    - Systolic BP (NO signs/symptoms of shock)
      - Dobutamine
    - Systolic BP (signs/symptoms of shock)
      - Dopamine
    - Systolic BP (<70 mm Hg + signs/symptoms of shock)
      - Norepinephrine

- Arrhythmia
  - Brady cardia
  - Tachycardia

See Sec. 7.7 in ACC/AHA Guidelines for patients with STEMI

Further Diagnostic/Therapeutic Considerations (for non-hypovolemic shock)

**Diagnostic**
- Pulmonary artery catheter, echo, angiography, etc

**Therapeutic**
- Intra-aortic balloon pump, reperfusion revascularization
Pharmacologic Treatment of Cardiogenic Shock

- SBP <70 mm Hg + shock
  → Norepinephrine
- SBP 70-100 mm Hg + shock
  → Dopamine
- SBP 70-100 mm Hg – shock
  → Dobutamine
- Refractory hypotension + shock
  → Amrinone or milrinone may improve cardiac output
The New England Journal of Medicine

- Multicenter, randomized, blinded study comparing Dopamine to Norepinephrine
- 1679 patients from 2003 – 2007
- Primary end point was rate of death at 28 days
The New England Journal of Medicine

- Multicenter, randomized, blinded study comparing Dopamine to Norepinephrine
Levosimendan

- Novel inodilator; calcium-sensitizing agent
- Hemodynamic improvement
- The Survival of Patients with Acute Heart Failure In Need of Intravenous Inotropic Support (SURVIVE) trial.
Quick Review of ED Treatments

- Rapid assessment of history, PE, CXR
- Echo-Doppler to assess LV function, RV size, MVR, effusion, septal rupture
- Pressors/inotropes for hypotension
- ASA
- β-blockers and nitrates should be avoided in acute phase
Hypotension
Shock? (RR < 90 mm Hg)
- Correction of fluid deficit
  - Vasopressors

Hypotension
Cold extremities
Oliguria

Ventilation unstable?

Intubation
Controlled Ventilation

Suspected right ventricular infarction?
(Echo?)
- Volume

Circulation unstable?

Dobutamine
Norepinephrine

Circulation unstable?

Circulation unstable?

Revascularization

Lung-Protective Ventilation
Pinspmax <= 30
Tidal Volume <= 6 mL/kg est. BW

Estimated Body Weight
M: 50+0.91 * (Height in cm-152.4)
F: 45+0.91 * (Height in cm – 152.4)

Criteria of Cardiogenic Shock
RRsys <= 90 mmHg and HF > 90/min
RRmean <= 65 mmHg
Signs of organ insuff: oliguria, cold extremities
CI < 2.2 L/min/m^2
PCWP > 15 mmHg

Abbreviations
BW = Body Weight
CI = Cardiac Index
N-/STE-ACS = Non-/ST elevation acute coronary syndrome
PCWP = Pulm Capillary Wedge Pressure
Pinspmax = max inspiratory peak ventilation pressure
RR = blood pressure

Therapy according to N-/STE-ACS Guidelines
Therapies Beyond the ED

- IABP
- LVAD
- ECMO
- PCI
- CABG
Intra-Aortic Balloon Pump

• Increases coronary blood flow, decreases LV afterload and LV EDP without increasing O2 demand.

• Currently Class I recommendation for patients with low C.O. states, hypotension and CS not responding quickly to other measures.

• IABP-SHOCK II Trial
http://www.youtube.com/watch?v=o11fhdVOYWA&feature=player_detailpage

DSCP, Wikimedia Commons
Left Ventricular Assist Device

![Diagram of a left ventricular assist device](https://commons.wikimedia.org/wiki/File:HeartMateII_tube.png)

- **Aorta**
- **Heart**
- **Diaphragm**
- **Blood pumped out to body**
- **HeartMate Blood Pump**
- **Blood flow out to HeartMate**
- **Connection to external air power supply**

Steven M. Gordon, Centers for Disease Control, [Wikimedia Commons](https://commons.wikimedia.org/wiki/File:HeartMateII_tube.png)
SHOCK Trial

- 1190 patients in SHOCK trial registry
- 60% mortality in CS
- Revascularization associated with decreased mortality
SHOCK Trial

- Emergency revascularization neutralizes impact of CAD
- CABG performed in 39% of SHOCK trial patients; overall improved 1-year survival
- In presence of CS, LVEF, initial TIMI and culprit vessel were independent correlates of 1-year survival
GUSTO-1 Trial

- 41,021 from 15 countries
- Streptokinase vs. tPA
- tPA more efficacious than Streptokinase in preventing shock.
- However, if CS is already established, not as useful.
Fibrinolytics

- Fibrinolytic therapy not as effective in accomplishing reperfusion in STEMI with CS.
- Mortality benefit of IABP + thrombolytics is additive
- Still, IABP + thrombolytics worse than PCI or CABG
Unadjusted mortality (Kaplan-Meier) first year after index admission for the 26,205 patients with ST-segment elevation myocardial infarction receiving reperfusion therapy between 1999-2004.
Predictors of Death in CS (partial)

<table>
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<th>Table XII. Miscellaneous risk factors (without right heart catheterization)</th>
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<td>Altered sensorium</td>
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<th>Table X. Risk corresponding to total points (without right heart catheterization)</th>
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Failed therapies

- Tilarginine (NO synthase inhibitor) TRIUMPH trial, 2007 showed no survival benefit
- GIK (high-dose glucose, insulin, potassium)
Question #1

A 60y.o.m with PMH HLP presents to the ED with c/o 2 hours crushing substernal CP radiating to L arm, N/diaphoresis. BP 82/48 mmHg, HR 110 bpm, O2 95% 4L. Severe respiratory distress, cold clammy extremities, S3 gallop, bilateral crackles. EKG reveals STE in anterolateral leads and ST depression in inferior leads. Pt given ASA, nitroglycerin, heparin, IVF. Vasopressors started to maintain BP, but he remains hypotensive despite 2 pressors. Which of the following is the most appropriate next step in management until pt reaches cath lab?

- Add a phosphodiesterase inhibitor
- Initiate cardiac glycosides
- Insert an IABP
- More aggressive fluid resuscitation
- Sodium nitroprusside infusion
Review Questions

Question #1

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- Add a phosphodiesterase inhibitor
- Initiate cardiac glycosides
- **Insert an IABP**
- More aggressive fluid resuscitation
- Sodium nitroprusside infusion
Review Questions

- IABP is recommended for patients with MI when cardiogenic shock is not quickly reversed with pharmacologic therapy. Used as a stabilizing measure prior to angiography and prompt revascularization.
- Phosphodiesterase inhibitors have some vasodilatory properties and should not be used in patients with low mean arterial pressure.
- Nitroprusside also has a vasodilatory effect and should not be used in low cardiac output states.
- Aggressive fluid resuscitation may be limited by acute pulmonary edema.
- Digoxin can be used in shock to control HR but only if atrial arrhythmias exist.
Review Questions

Question #2

Which of the following steps has been shown to have a mortality benefit in patient with cardiogenic shock cause by MI?

– Addition of glycoprotein IIb/IIIa inhibitors
– B-adrenergic agonists
– Early cardiac cath followed by revascularization by PCI or surgical revascularization
– Initial medical stabilization with blood pressure control prior to catheterization
– Thrombolytic infusion
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- Addition of glycoprotein IIb/IIIa inhibitors
- B-adrenergic agonists
- **Early cardiac cath followed by revascularization by PCI or surgical revascularization**
- Initial medical stabilization with blood pressure control prior to catheterization
- Thrombolytic infusion
Review Questions

• The SHOCK trial compared emergent revascularization for cardiogenic shock due to MI with initial medical stabilization and delayed revascularization. This showed a mortality benefit at 30 days that increased over time at 6 months an 1 year. The ACC/AHA recommend early revascularization for pts aged 75yrs or younger with STE or LBBB who develop shock within 36 hours of MI and suitable for revascularization that can be performed within 1 hours of shock.
References