emDocs

The EM Educator Series

Challenging Diagnosis of Cardiogenic Shock

Author: Alex Koyfman, MD (@EMHighAK) // Edited by: Brit Long, MD (@long_brit) and Manpreet Singh, MD (@MprizzleER)

Case #1:
A 62-year-old male presents with chest pain, dyspnea, and diaphoresis. He appears ill, with VS RR 24, HR 90, BP 72/41 mm Hg, Sat 90% on RA, T 37C. His ECG demonstrates ST elevation in V1-V4, with reciprocal changes.

Questions for Learners:

1) What are the etiologies of cardiogenic shock?
2) What does the RUSH exam entail?
3) What will the physical exam demonstrate, and what are its limitations?
4) What is the medical management of cardiogenic shock?
5) How do you optimize this high-risk intubation?
6) When the ship is sinking, who else can help?

Suggested Resources:

- ✓ Articles:
  - emDOCs – Cardiogenic Shock
  - R.E.B.E.L. EM – Cardiogenic Shock
  - EM in 5 – Cardiogenic Shock
  - emDOCs – RUSH ("Rapid Ultrasound for Shock") Protocol
  - First 10 EM – Undifferentiated hypotension
  - emDOCs – The Hypotensive ED Patient: A Sequential Systematic Approach
- ✓ Podcasts:
  - EMCrit RACC – Podcast 10 – Cardiogenic Shock

Answers for Learners:

1) **What are the etiologies of cardiogenic shock?**

While acute myocardial infarction (AMI) is the major cause of cardiogenic shock, other diagnostic considerations should always be entertained. These include valvular heart disease (particularly flail leaflet), myocarditis, myocardial contusion, and cardiomyopathies.

- Rate-related
- Valve Disorder
- Ischemic (Right sided infarct, STEMI, NSTEMI)
- Cardiomyopathy
- Toxicologic

2) **What does the RUSH exam entail?**

- **Introduction**

One of multiple described ultrasound protocols for evaluation of patients presenting to ED with undifferentiated hypotension
Provides a framework for rapid and systematic evaluation of cause of hypotension
Three categories
  - Pump – Cardiac evaluation
  - Tank – Volume status
  - Pipes – Vascular system

Equipment: Ultrasound machine with phased array (3.5-5MHz) and linear probes (7.5 – 10MHz)

- **Pump: Cardiac Evaluation**

Determine the Presence of Pericardial Effusion
Appears as anechoic fluid surrounding the heart
Possible pitfall is to misdiagnose an effusion
Distinguish pericardial fat pad from pericardial effusion by mild echogenicity of fat pad and know that fat pads tend to move in concert with myocardium
Pericardial effusion appears anterior to descending aorta in parasternal long axis view as opposed to pleural effusion that appears posterior to descending thoracic aorta
Look for evidence of cardiac tamponade
Right ventricle and atrium may have diastolic collapse, plethoric IVC

Assess LV Contractility
Assessing LV ejection fraction – hyperdynamic, normal, moderately or severely decreased
Visual assessment by emergency physicians generally accurate
E point Septal Separation (EPSS; distance of E wave of anterior MV leaflet from septum in M mode) or calculating fractional shortening may allow more objective assessment

Assess RV Strain
Normal RV/LV ratio is 0.6:1
RV/LV ratio >0.9 suggestive of right heart strain, may suggest acute pulmonary embolism

Parasternal short axis may underestimate size of RV, use other views
Bowing of septum into LV suggests elevated right heart pressure ("D sign")
Thickened RV wall suggests chronic right heart strain (e.g. pulmonary htn, COPD)

- **Tank: Volume Status**

"Tank Fullness" – IVC Evaluation
Measure IVC 2cm distal from cavoatrial junction or immediately superior to insertion of hepatic veins
IVC diameter < 2cm with >50% collapse correlates with CVP < 5mmHg and suggests fluid responsiveness
IVC diameter > 2cm with < 50% collapse correlates with CVP > 10mmHG, argues against fluid responsiveness

"Tank Leakiness" – FAST Exam and Lung Ultrasound
Evaluates peritoneal compartment for free fluid
In non-traumatic setting and depending on the clinical scenario, presence of free fluid may suggest ruptured AAA, ectopic pregnancy, or ruptured hemorrhagic cyst

"Tank Overload" – Assessment for Pleural Effusion and Pulmonary Edema
As part of the FAST exam, the views of the RUQ and LUQ should include views above the diaphragm to assess the presence of pleural effusion
To assess for pulmonary edema, use the phased array probe in the anterolateral chest, between the 2nd and 5th intercostal spaces
Presence of multiple B lines (vertical reverberation artifact extending from the pleural line to the far field) suggests pulmonary edema
Finding of poor LV contractility, multiple B lines, and plethoric IVC is suggestive of cardiogenic shock

"Tank Compromise" – Assessment for Pneumothorax as Cause of Obstructive Shock
Place linear probe in mid-clavicular line between 3rd and 5th intercostal spaces
Assess for normal lung sliding or "waves on a beach" pattern on M mode
Lack of lung sliding or "bar code" sign is suggestive of pneumothorax

- **Pipes: Circulatory System**

Aorta (AAA)
Measurement of the aorta should begin at epigastrium and extend distally to bifurcation of iliac arteries
Measurement of the aorta should be from outer wall to outer wall; abnormal if >3cm
In a hypotensive patient with AAA > 3cm, acute rupture should be considered

Aortic Dissection
Thoracic aortic dissection may be detected on parasternal long axis view with aortic root measuring > 3.8cm
Aortic root best seen in parasternal long axis. You may see an intimal flap if dissecting

"Clogged Pipes" – Deep Vein Thrombosis
Compression US of lower extremities using linear probe
Should be performed at the level of the common femoral vein to the bifurcation of the deep and superficial femoral veins and at the popliteal vein extending to the trifurcation of the calf veins
The RUSH exam provides a framework for approaching the non-traumatic patient in the emergency department presenting with undifferentiated hypotension. While the exam generally should start with

the cardiac exam, the clinician's judgment and clinical context should guide the progression through the different components of the exam.

3) **What will the physical exam demonstrate, and what are its limitations?**

Signs + Symptoms:

- Shortness of breath
- Dyspnea on exertion
- Diaphoresis
- Cough with pink sputum
- Chest pain
- Air hunger
- Hypoxia
- Tachycardia
- JVD
- Rales
- Skin pallor/mottling
- Altered Mental Status
- Decreased Urine Output

4) **What is the medical management of cardiogenic shock?**

Start with what you know: ABCs, IV, O2, Cardiac Monitor and Ultrasound.

It is critical to obtain a 12-lead ECG while ABCs are being secured. An AMI not only represents the major cause of cardiogenic shock but also represents one of the fixable etiologies. These patients usually will not have subtle AMIs. They'll typically have large anterior STEMIs although inferior STEMI with extension into the RV is common as well. Don't forget to look closely for elevations in aVR, which represent significant left main coronary artery (LMCA) or left anterior descending (LAD) artery disease. Once a STEMI is identified, get your cath team on the phone immediately. Opening of the culprit vessel is the intervention most likely to save the patient. Unfortunately, thrombolytics are not very effective in STEMI with cardiogenic shock. These patients have intense thrombolytic resistance likely secondary to marginal drug delivery (secondary to low diastolic pressure and thus low coronary artery filling pressures) as well as acidosis. The SHOCK registry demonstrated that thrombolytics did not significantly change mortality (Hochman 1995).

5) **How do you optimize this high-risk intubation?**

Patients with cardiogenic shock will have severe respiratory distress. Unlike patients with ADHF, they often will not tolerate non-invasive positive pressure ventilation (NIPPV) and will require emergent intubation. Unfortunately, these patients are challenging to intubate as they have all three of the "HOp killers" (hypotension, hypoxia and acidosis) as discussed by Scott Weingart. The intubation strategy for these patients is beyond the scope of this post and has been covered here. The big critical pieces are maximizing preintubation hemodynamics (small fluid bolus, push dose pressors) and preoxygenation as well as oxygenation during intubation (NO DESAT).

6) **When the ship is sinking, who else can help?**

Concomitant to addressing airway, breathing and getting an ECG to aid with diagnosis, circulation must be addressed. Enhancing perfusion to the brain and coronary vessels is critical. Enter vasoactive medications. The optimal agent would be one that increased coronary artery perfusion, had minimal effects on heart rate, decreased afterload and decreased myocardial oxygen demand while enhancing cerebral perfusion pressure. Unfortunately, no such agent exists.

The gut instinct of many physicians is to reach for an inotrope like dobutamine. However, caution must be practiced here. Dobutamine has beta-1 and beta-2 agonist activity, which may augment cardiac output but will also cause vasodilation. The balance between increased output and peripheral vasodilation leads to the classic teaching that one-third of patients will drop their blood pressure, one-third will have no change in blood pressure and one-third will have increased blood pressure. Unfortunately, there's no good way to predict which patient will have which response.

This can be combated by initiating a vasopressor first and then adding the inotrope when blood pressure has become relatively stable (shooting for a MAP = 65 mm Hg). There is no optimal vasopressor for this indication. The ACC/AHA recommends the following (Overgaard 2008):

SBP 70-100 (w/o signs of shock) Start dobutamine
SBP 70-100 (w/ signs of shock) Start dopamine
SBP < 70 Start norepinephrine

However, an RCT of patients with undifferentiated shock showed that norepinephrine was superior to dopamine specifically in the subgroup of patients with cardiogenic shock (De Backer 2010). Although it is recommended that norepinephrine be given through a central line, temporary infusion through a good peripheral line while central access is being obtained is reasonable. Epinephrine (adrenaline) is a viable alternate option as well as it may increase cardiac contractility as well as increasing MAP.

Is there a role for intra-aortic balloon pumps (IABP) in these patients? In theory, IABP placement makes sense. It should increase myocardial oxygen supply by increasing coronary artery perfusion and decrease myocardial oxygen demand. The largest study of IABP in cardiogenic shock was published in the NEJM in 2012. In this prospective, randomized, unblinded (hard to blind a patient or doctor to the presence or absence of a large catheter in the groin) trial, the authors demonstrated no mortality benefit to IABP placement (Thiele 2012). This trial had many flaws but challenges the potential benefits of IABP placement.