Decompensated CHF & Cardiogenic Shock

NYEMU Europe 2009
Michael J. Betzner MD FRCPc
Emergency Physician
Calgary
Senior Medical Director
STARS

Learning Objectives

• Review strategies to avoid intubation in pts with severe CHF (with some BP to work with & with no BP to work with at all)
• Review intubation strategies should treatments fail to stabilize the patient

Overview Cardiogenic Shock

• JVD + pulmonary edema + cardiac gallop + altered LOC + ↓ BF + ↓’d urine output
• Very ominous presentation
• Sickest of the sick - common underlying factor in ‘clean kills’
• Mortality 60-80% despite all efforts

Overview Cardiogenic Shock

• AMI most common initiating event
• Acute contributory mechanical complications include:
  • Acute MR 2° to papillary muscle dysfn or rupture
  • Ventricular septal rupture or free wall rupture
Pathophysiology

Myocardial Ischemia

LV Dysfunction → ↓'d BP → worse myocardial ischemia

Impaired LV fn → decreased CO → activation of neurohormonal compensatory mechanisms which can actually accelerate the progression of CHF

Poor LV compliance

DDx

- ACS
- Aortic Regurgitation
- Cardiomyopathies
- Acute on Chronic CHF
- Mitral Regurgitation
- Myocarditis
- Pericarditis & Tamponade
- PE

Pathophysiology

- Sympathetic system activation & eventual depletion
- Activation of RAAS
- Increased natriuretic peptides
- Increased ADH
- Increased Endothelins
**Sympathetic Activation**

- Increases cardiac output, HR, & causes peripheral vasoconstriction
- Activates RAAS → increases preload & afterload
- Increased NorEpi levels → myocardial cell death & areas of focal necrosis → further impairing LV fn

**Activation of RAAS**

- Leads to increased Angiotensin II which leads to:
  - Increased aldosterone
  - Increased NorEpi
  - Inhibition of vagal tone

**Diagnostics in Cardiogenic Shock**

- Orthopnea, dyspnea, edema
- S3, tachycardia, & elevated JVP
- EKG - abnormal 90% of time
- CXR - cardiomegaly, vascular redistribution, interstitial edema, effusions
- Bedside US

**What about BNP?**
Nitrates

- ↓’s preload & afterload (slightly)
- ↓’s mortality & improves symptoms
- sublingual, patch, or IV
- Titrate IV up q 3-5 mins
- Hypotension can be aggravated

Nesiritide

- No role for this agent
- 50 X more expensive than nitro with NO benefit & potentially increased mortality
- Increased risk of subsequent renal dysfunction

Diuretics

- Venodilate (immediate) + Diuresis (delayed)
- Cause ↑’d plasma renin & ↑’d NorEpi levels → increased SVR

ACE Inhibitors

- Captopril sublingual can ↓ PCWP within 10 minutes (peak 30 min)
- Early use decreases admission to ICU
- No role for ARBs over ACEi inhibitors acutely
- Consider in hypertensive CHF pts if Nitro maximized
Other Afterload Reducers

- Nitroprusside - may have a role in pts with borderline BP, but risky
- May be useful if Nitro failing to reduce BP in hypertensive patients with CHF
- Useful in pts with MR

Morphine

- Classically taught as useful for decreasing anxiety and preload
- Causes sedation & respiratory depression
- Increases ICU admission rate

β-Blockers in CHF

- Unquestionable benefit chronic severe CHF pts
- Counteract the harmful effects of the sympathetic nervous system in CHF
- But - no role acutely in ED unless ↑HR playing a huge role
- Esmolol agent of choice for rare acute use

IABP

- Last ditch effort as a bridge gap to acute plasty or bypass surgery
- Superior to pressor therapy in terms of net effect on myocardial O₂ consumption
NIV

- NIV appears to improve hemodynamics & reduce intubation rate, but doesn’t affect mortality
- No proven benefit in AMI patients with CHF → intubation preferred

Pressors

- Dopamine
- Dobutamine
- NorEpi
- Milrinone/Amrinone

- Consider pressor use in pts presenting with SBP < 85 & poor end organ perfusion

Dopamine

- Increases HR & myocardial O₂ consumption
- May have a role in hypotensive patients with poor end organ perfusion
- Double edged sword

Dobutamine

- Can vasodilate & improve contractility
- Increases HR
- Increases arrhythmia
- Mortality benefit unclear
**NorEpi**

- Same issues as Dopamine

**Milrinone/Amrinone**

- Similar effects to Dobutamine
- Longer half life
- No evidence of acute benefit
- More for chronic use in patients awaiting transplant

**Intubation/RSI PEARLS in Cardiogenic Shock**

- Keep patient in upright/sitting position
- Topicalize - 4 cc 4% Lidocaine neb
- Minimize, minimize, minimize drugs given...

**Intubation/RSI PEARLS in Cardiogenic Shock**

- Stay with hemodynamically stable isolated agents:
  - **Fentanyl** 25 mcg IVP q 2-3 min to endpoint, OR
  - **Ketamine** 0.25-0.5 mg/kg IVP q 3-5 mins to max 2 mg/kg, OR
  - **Etomidate** 0.15 mg IVP q 5 min prn to max 0.3 mg/kg
Intubation/RSI PEARLS in Cardiogenic Shock

- Paralytics:
  - Usual choices & contraindications
  - Try to avoid if you can - taking away tone will take away pressure
  - Have inotropes primed & ready for use

- Slowly titrate up PEEP to improve oxygenation as pressure allows
- May have to titrate up pressors to allow more PEEP
- Follow serial gases/lactates to ensure you're keeping up with acidosis

References

- Chen/Somarovsky. Advanced Cardiac EDS Chapter - The EDS II Course.
- Peacock, W.F. Impact of Intravenous Loop Diuretics on Outcomes of Patients Hospitalized with Acute Decompensated Heart Failure - Insights from the Adherence Registry Cardiology 113 (1):11, Jan 2009.