



Foundations Frameworks

Approach to CHF Exacerbations

Author: Quentin Reuter, MD

Editors: Emilie Powell, MD; Kristen Grabow Moore, MD, MEd

HPI

- SOB/cough/orthopnea, swelling in feet, worsening fatigue and exercise intolerance, decreased appetite

PE

- Assess volume status: dry vs current weight, JVD, crackles on pulm exam, r/o valve catastrophe
- Assess perfusion: mental status, BP, oxygen saturation, warm (perfused) vs cold and clammy extremities

Labs

- CBC, chem, trop, BNP, lactate
- BNP <100 (or 300 in dialysis) = no heart failure, BNP > 500 = heart failure

Imaging

- EKG
- Chest XR to assess for cardiomegaly and pulmonary edema
- Bedside ECHO to r/o effusion, assess LV function, assess RV for signs of PE, evaluate for pulmonary edema (B lines)

Causes of CHF exacerbations:

1. Ischemic heart disease: STEMI, NSTEMI → EKG, trop
2. Rate: tachy/brady dysrhythmias → EKG
3. Valvular catastrophe: typically MR, listen for a murmur → echo
4. Tox/Metabolic: cocaine, amphetamines, thyrotoxicosis, CCB/BB overdose, digitalis toxicity, EtOH w/d
5. Infectious: myocarditis, sepsis induced cardiomyopathy → echo
6. Dietary indiscretion/medication non-compliance: diagnosis of exclusion
7. Not all fluid overload is CHF: make sure pulm edema isn't from AKI

Types of CHF exacerbations:

1. "Stable" CHF exacerbation
 - a. Warm and well perfused, stable BP, peripheral edema, sob/mild pulmonary edema
 - b. Admit to floor vs home with close follow-up, there is a wide spectrum of disease severity in this "stable" group
 - c. Consider treating the following clinical derangements
 - i. Volume overloaded: give one-time dose of diuretic
 1. 40mg IV furosemide if diuretic naïve or
 2. IV doses of home diuretic (could do 2 times home dose)
 3. preload/afterload reduction with nitrates (SL 400mcg/tab, paste 1-2in)
 - ii. Hypertension: preload reduction with nitrates (SL 400mcg/tab, paste 1-2in) or ACE-I (such as enalapril)
 - iii. Hypoxia/SOB: oxygen, nitrate, BIPAP/CPAP
 - d. Easy way to remember -> UNLOAD: upright, nitrates, furosemide, oxygen/NIPPV, ACE-I, digoxin

2. SCAPE: Sympathetic Surge, Crashing, Acute Pulmonary Edema- AKA "flash pulmonary edema" often in patient with diastolic failure
 - a. Hypertensive (SBP > 180 typically), hypoxic, tachypneic, in extremis
 - b. Goal is to lower afterload and preload aka abolish sympathetic overdrive and redistribute pulmonary edema.
 - c. Heart failure leads to fluid on lungs, this increases sympathetic surge, increasing afterload, worsening forward flow and increasing preload on a poorly relaxing heart, leading to more fluid backup onto lungs, worsening hypoxia.
 - d. Treatment:
 - i. IV access, cardiac monitor, O2 monitor, supplemental O2, defibrillator pads in room
 - ii. Nitroglycerin: works to decrease preload, afterload at high doses
 1. Sublingual 0.4mg equates to 50-80 mcg/min if given every 3-5 minutes
 2. Drip -> make sure to start high enough if requiring sublingual nitro every 3-5 minutes: 50-80 mcg/min, quickly titrate to improvement of WOB and BP
 - iii. CPAP/BIPAP: positive pressure will help redistribute fluid back into vascular space as well as decrease preload and afterload
 - iv. ACE-I or nicardipine: if patient still hypertensive despite high doses of IV nitroglycerine, consider sublingual or IV ACE-I or IV nicardipine, easy and rapid way to decrease afterload
 - v. Diuretics: not always necessary in ED, sometimes a fluid distribution (lungs) problem rather than overload problem

3. Cardiogenic Shock
 - a. Hypotensive, cold and clamped down, poor end organ perfusion (AMS, CP, SOB), elevated lactate, low SVO2 (if you have a central line)
 - b. Systolic failure
 - c. These patients need inotropes (more specifically inodilators) because you typically can't decrease their afterload or preload because they have no room to go on their BPs
 - d. Can start with small fluid bolus – 250cc to 500cc and see response, careful giving more
 - e. What is your choice pressor? -> dobutamine
 - i. If BP still low with dobutamine (MAP <65) you can start a vasopressor (e.g., norepinephrine) but remember this will worsen the afterload that their weak heart has to pump against and will likely not improve perfusion, it will only clamp them down further which may improve the BP but not their forward flow.
 - f. Monitor resuscitation for signs of improved perfusion
 - i. Look for increased urine output, improving mental status, improving pulmonary edema, down-trending lactate
 - g. Mechanical support: call cardiology for potential balloon pump, impella, LVAD, etc.

References:

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