emDocs

# The EM Educator Series

## The EM Educator Series: Heart Failure and Flash Pulmonary Edema

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**Case 1:**
**A 79-year-old female with a history of heart failure, coronary artery disease, diabetes, and hypertension is brought in by family with worsening shortness of breath and peripheral edema over 8 days. She denies fevers, chest pain, abdominal pain, cough, and back pain. Her daughter accompanies her and states she has had multiple admissions for similar issues "When she doesn't take her medications and eats a lot of soup." She is overall well appearing and nontoxic, and her vital signs include BP 162/92 mm Hg, RR 19, HR 77, saturation 97% on room air, and T 97.4F. She has crackles at the bases of her lungs, and her legs demonstrate 2+ pitting edema.**

**Case 2:**
**A 63-year-old male with a history of heart failure, coronary artery disease, and hypertension presents with severe shortness of breath. He is brought immediately to the resuscitation bay. He is diaphoretic and in respiratory distress. His vital signs include BP 221/107 mm Hg, RR 28, HR 104, saturation 89% on room air, and T 98.2F. He has diffuse crackles on pulmonary exam, but there is no apparent peripheral edema.**

**Questions for Learners:**

1. What are the different types of heart failure?
2. How can patients present with heart failure, and what is flash pulmonary edema? Are all patients with flash pulmonary edema truly volume overloaded?
3. What laboratory testing is recommended? What is the utility of BNP?
4. What imaging is recommended? How does ultrasound change your management?
5. What management is recommended when it comes to medications and the airway?

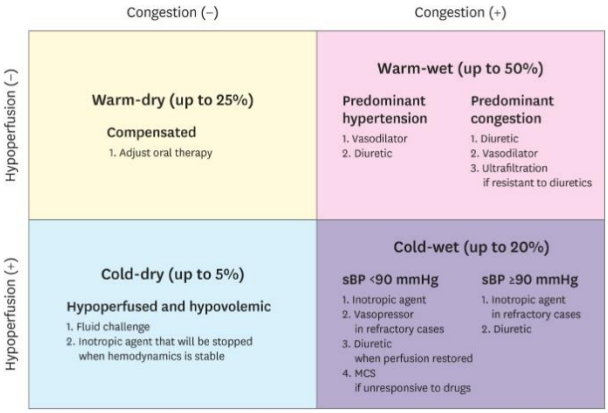
**Suggested Resources:**

- Articles
  - Core EM - Acute Pulmonary Edema
  - EMCrit - SCAPE
  - Emergency Medicine Cases
  - emDOCs - HF Myths Part 1
  - emDOCs - HF Myths Part 2
  - emDOCs - HF management pearls and pitfalls
  - emDOCS - BNP in the ED
  - emDOCs - Furosemide for acute pulmonary edema treatment
- Journal Articles
  - Diagnosis of HF in the ED
  - Management of HF in the ED
  - HF Misconceptions HF Disposition

**Answers for Learners:**

**1. What are the different types of heart failure?**

Categorized by left vs right, systolic vs diastole, reduced vs preserved.




Practical Classification of CHF to Direct ED Management

- Hypertensive patients (50% of patients with acute pulmonary edema): typically older patients, often women, with diastolic dysfunction and symptoms developing acutely over 24-48hrs due to fluid misdistribution and increased afterload
    - Clinical features of pulmonary edema (weight gain and edema may not be present), crackles but good systolic function, and needs to be treated aggressively with nitroglycerin to decrease preload and afterload, with secondary consideration to diuretics if volume overloaded
- Normotensive patients: typically younger patients with systolic dysfunction and subacute worsening of their baseline status over days to weeks as a result of gradual total body fluid overload (as opposed to isolated pulmonary congestion causing respiratory distress)
    - Clinical features of peripheral edema (+/- pulmonary edema), weight gain and gradual onset, and needs to be treated up front with diuretics, with a secondary consideration for nitroglycerin

**2. How can patients present with heart failure, and what is flash pulmonary edema? Are all patients with flash pulmonary edema truly volume overloaded?**

**Symptoms**

- Shortness of breath
- Dyspnea on exertion
- Diaphoresis
- Cough with pink sputum
- Chest pain

**Signs**

- Air hunger
- Hypoxia
- Tachycardia
- JVD
- Rales
- Skin pallor/mottling
- Altered Mental Status
- Decreased Urine Output

Flash pulmonary edema is a general clinical term used to describe a particularly dramatic form of acute decompensated heart failure. In this setting, patients are often hemodynamically unstable and develop severe pulmonary congestion, although they frequently are not fluid overloaded. These patients are pharmacologically treated similarly to the decompensated patients described earlier; however, specific therapy should be guided by the underlying precipitating factor.

**3. What laboratory testing is recommended? What is the utility of BNP?**

- BNP is a vasoactive hormone that is released by strained myocardium from a variety of causes
- The use of BNP as a cardiac biomarker has been shown to reduce hospitalization rates and length of stay, and may be useful for prognostication of CHF
- There has been much interest in the use of BNP in the ED to help improve diagnostic accuracy of CHF, since ED physicians are only about 80% accurate in their diagnosis of CHF without BNP
- Currently, the categorical use of cardiac markers such as BNP or N-Terminal-Pro-BNP (NT-Pro-BNP) for the diagnosis of CHF in the ED may not be very useful: while a BNP level of 500 has a moderately good specificity to rule in CHF, in both cases these patients are usually not presenting with a diagnostic dilemma (ie physician gestalt is already very accurate in patient's with very low or high BNP)
- Steinhart et al. recently published an article using a novel approach to biomarkers, which might help exactly where we need help – patients in the "grey" zone (where there is a diagnostic dilemma, and the clinician's pre-test probability for the diagnosis of CHF is 20-80%: the mathematical prediction model uses the clinician's pre-test probability (ie. gestalt) along with the absolute NT-Pro-BNP value (continuous, not categorical value)
    - The model appropriately redirected 44% of patients with intermediate clinical probability to either low or high probability, without inappropriately redirecting clinicians towards the 'wrong' eventual diagnosis
- Troponin should be considered in every patient presenting with possible CHF given that the differential for possible etiologies includes ischemic heart disease (especially in elderly patients with atypical symptoms presenting with dyspnea, or in new-onset CHF)
- Even though troponin might be elevated due to other causes (eg, renal failure, sepsis), patients with elevated troponin levels, regardless of the cause, have higher morbidity and mortality

➔ Natriuretic peptides should only be used in combination with clinical evaluation. Other causes of elevated BNP should be considered.

**4. What imaging is recommended? How does ultrasound change your management?**

Chest x-ray Interpretation Acute Congestive Heart Failure

- JAMA Rational Clinical Exam article lists these chest x-ray (CXR) findings (with likelihood ratios) as the most important in AHF: pulmonary venous congestion (distension of pulmonary veins and redistribution to the apices; LR 12), interstitial edema (LR 12) and cardiomegaly (LR 3.3), although all have wide confidence intervals
- Others to keep in mind: Kerley B lines (See Image Below), and "bat wing"or "butterfly" pattern in severe AHF
- A comparison with a baseline CXR (eg, best-looking CXR just before discharge from hospital during the last admission) is often useful
- Signs of pulmonary congestion on might not show up on CXR until a few hours have elapsed after the onset of flash pulmonary edema (therefore patients with AHF may have a normal CXR)
- A normal CXR does not rule out AHF

POCUS is an important diagnostic modality in patients with suspected APE. In patients with APE, POCUS will demonstrate the presence of "B-lines." The presence of > 3 B-lines per rib space suggests the presence of interstitial pulmonary fluid. Evidence demonstrates that physicians more accurately identify pulmonary edema on lung US than with CXR. Additionally, POCUS may identify a ruptured valve causing the patient's symptoms leading to an alternate management pathway (i.e. cardiovascular surgery for valve repair)

**5. What management is recommended when it comes to medications and the airway?**

### Medications for Acute Congestive Heart Failure

- Nitroglycerin (NTG): Used to decrease preload (and afterload at higher doses), can be started as sublingual spray but should be switched to continuous infusion early (start at 30mcg/min, but may need to increase it by 10mcg/min every 10min, up to 150-200mcg/min – consider an arterial line for monitoring)
    - Be cautious in preload-dependent patients such as inferior MI, pulmonary hypertension and aortic stenosis
    - Acutely decompensated heart failure patients with hypertension and shortness of breath who received intermittent IV nitroglycerin boluses (1-2mg), showed an associated lower ICU admission rate and shorter hospital LOS compared to continuous nitroglycerin infusion.

- Furosemide (Lasix): Works by targeting kidneys, which are often poorly supplied in conditions of hypotension or catecholamine overdrive due to the splanchnic vasculature vasoconstriction
    - Therefore, it might be reasonable to wait for restoration of better renal perfusion while other modalities are instituted (eg, BiPAP, NTG) before using it; the patient's daily dose as an IV bolus through saline lock is a reasonable first dose, with doubling 30-60min later if no clinical effects
    - Use furosemide judiciously in patients with renal failure or low serum sodium: there is an association between high creatinine, furosemide use and higher long-term mortality
    - Continuous infusion of furosemide as opposed to bolus therapy has been shown to reduce all- cause mortality in AHF

- Morphine: Although the ADHERE registry (the largest HF registry to date) has shown worse outcomes when it is used in high doses (30-40mg), leading to increased rates of intubation and ICU admission, days of hospitalization and mortality, small doses may be considered (benzodiazepines are an alternative to decrease anxiety and the catecholamine drive)

- ACE Inhibitors: While there is evidence from small trials that SL Captopril and IV Enalapril decrease the need for intubation and rapidly improve symptoms, the 2007 Canadian Guidelines for Heart Failure suggest that they should not be used routinely; oral ACE inhibitors should be considered after the patient is stabilized, usually by the specialist at 12-24hrs after presentation

- Consider Nitroprusside in patients with a hypertensive emergency and AHF who do not respond to high doses of IV Nitroglycerin

## Non-Invasive Positive Pressure Ventilation (NIPPV)

- BiPAP has revolutionized the management of AHF, and has been shown to significantly reduce the need for intubation by increasing oxygenation and cardiac output through maintenance of alveoli patency
    - Consider its use early in the management of AHF

## Vasopressors in Acute Congestive Heart Failure

- The 2007 Canadian Guidelines for the Management of Heart Failure suggest dobutamine over milrinone for patents in cardiogenic shock