



Episode 4 – Acute Heart Failure

Prepared by Dr. Lucas Chartier

-Acute Heart Failure (AHF) is both prevalent and difficult to diagnose, owing to a clinical presentation having much overlap with other conditions, the lack of reliable test or imaging modality, and the absence of a practical classification system

-General Approach to Shortness of Breath in the ED:

1. Cardiac (AHF, pericardial effusion and tamponade, arrhythmias, ischemia, etc)
2. Respiratory (COPD, asthma, pneumonia, pneumothorax, primary pulmonary hypertension)
3. Hematological (anemia)
4. Neuromuscular (ALS, Guillain-Barré, etc)

History

-JAMA review article lists these historical features (with likelihood ratios) as the most important in AHF: past history of congestive heart failure (CHF; LR 5.8) or of myocardial infarction (LR 3.1), paroxysmal nocturnal dyspnea (LR 2.6), and orthopnea (LR 2.2)

-Others to keep in mind include: increased weight gain, peripheral edema, and lack of compliance with medications or diet (eg, salt)

Physical

-JAMA review article lists these physical examination features (with likelihood ratios) as the most important in AHF: presence of third heart sound (ventricular filling gallop; LR 11 – although poor interobserver agreement, especially in a noisy ED), jugular venous distension (LR 5.1) esp. with hepatojugular reflex, pulmonary rales (LR 2.8), any cardiac murmur (LR 2.6) and leg edema (LR 2.3)

-The absence of crackles does not exclude pulmonary congestion

-Other key physical exam point: new-onset cardiac murmur (eg, a new mitral regurgitation murmur in the setting of AHF may require urgent surgical management)

-A wide pulse pressure may indicate a low cardiac output and portend a worse prognosis vs. a narrow pulse pressure may signify high output causes of AHF such as anemia and thyrotoxicosis

ECG

-JAMA review article lists these ECG features (with likelihood ratio) as the most important in AHF: atrial fibrillation (LR 3.8), new T-wave changes (LR 3.0) or any abnormal ECG findings (LR 2.2) increase the likelihood of AHF, whereas a completely normal ECG (13% of patients with AHF) decreases the likelihood of AHF (LR 0.64)

-Others to keep in mind: any other arrhythmia (atrial fibrillation present in 25% of patients with AHF), left bundle branch block signifying possible poor LV function, and evidence of ventricular hypertrophy, which may signify untreated chronic hypertension or valvular disease

-Non-cardiogenic pulmonary edema has been associated with giant, diffuse, symmetrical inverted T waves with QT prolongation



Chest x-ray

- JAMA review 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, 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

Cardiac markers: B-type Natriuretic Peptide (BNP) and Troponin

- 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 AHF
- There has been much interest in the use of BNP in the ED to help improve diagnostic accuracy of AHF, since ED physicians are only about 80% accurate in their diagnosis of AHF without BNP
- Currently, the categorical use of cardiac markers such as BNP or N-Terminal-Pro-BNP (NT-Pro-BNP) for the diagnosis of AHF in the ED may not be very useful: while a BNP level of <100 has a good sensitivity to rule out AHF, and a level of >500 has a moderately good specificity to rule in AHF, 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 AHF 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 AHF 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 AHF)
- 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



Echocardiography

- Most patients will eventually need formal echocardiography, but some might mandate it sooner or on an urgent basis directly from the ED, such as young patients with no prior history or patients with new-onset cardiac murmur who might require urgent surgical management
- Consider a bedside ED Ultrasound in all patients with distended neck veins or “shortness of breath NYD” to rule out pericardial effusion and cardiac tamponade
- There is little/no role for EM physicians to determine the degree of diastolic or systolic function with bedside ultrasound in the ED since this is a very difficult skill to acquire and it will not change the ED management significantly

Important Causes of AHF

- Ischemia, arrhythmias, severe hypertension, valvular dysfunction, cardiomyopathy, thyrotoxicosis or myxedema coma, myocarditis and anemia

Practical Classification of AHF 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

Management

- More than one disease (eg, AHF and COPD exacerbation) often needs to be addressed simultaneously in an undifferentiated dyspneic patient until a clearer picture emerges (eg, if wheezes are present, it may be reasonable and relatively safe in AHF to use bronchodilators early in the course of an undifferentiated patient)
- Don't forget to find and treat underlying causes as listed above



Medications

- 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
- 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

- 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

Pressors

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



Disposition

- Multiple factors have been shown to predict worse prognosis and the need for admission: systolic BP <120, low sodium, high BUN, elevated creatinine, elevated troponin, ECG changes, BNP >500 and poor response to initial therapy; other considerations for admission include patients with first-episode AHF (who need investigations looking for an etiology)
- Several prediction models for those who can be discharged from the ED have unacceptably high mortality rates at 30d (~8%), but may include patients with frequent visits to the ED for exact same presentations with good response and clear diagnosis, that have made inappropriate use of prescription medications, NSAIDs or salt intake; these patients still need prompt follow-up with CHF clinic or their primary care provider

Future research efforts

- Emergency department-based research (led by EPs, not cardiologists) with short-term outcomes (1-2wk outcomes, not 60 days or 1 year) are needed
- A validated prediction model for safe discharge from the ED is needed

KEY REFERENCES

- Canadian Cardiovascular Society Consensus Conference recommendations on heart failure update 2007: Prevention, management during intercurrent illness or acute decompensation, and use of biomarkers. *Canadian Journal of Cardiology*. Vol 23, No.1. Jan 2007
- Rational Clinical Exam Series: Wang CS, et al. Does This Dyspneic Patient in the Emergency Department Have Congestive Heart Failure? *JAMA* 2005;294(15):1944-56
- Steinhart B et al. Improving Diagnosis of Acute Heart Failure Using a Validated Prediction Model. *J Am Coll Cardiol* 2009;54(16):1515-21