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EM:RAP C3 Continuous Core Content

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C3: CHF

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* *Drug doses are a guide only, always check a second source and follow local practice guidelines*

Take Home Points:

- Congestive heart failure (CHF) is one of the commonest diagnoses in ED and hospitalized patients.
- CHF is a clinical diagnosis and it make co-exist with or mimic other common (and serious) cardiopulmonary conditions such as COPD, PE, MI and pneumonia.
- Initial treatment for pulmonary edema due to CHF is with nitroglycerin and non-invasive ventilation (CPAP or BiPAP).
- Initial treatment for cardiogenic shock due to CHF is with inotropes and vasopressors such as norepinephrine and epinephrine.
- An attempt to determine the underlying reason for the new or recurrent episode of CHF is a critical part of the assessment. This may lead to specific emergency interventions such as cardiac catheterization in the event of a coronary occlusion or to the operating room in the case of an acute valvular emergency.

Introduction

Congestive heart failure (CHF) is a big deal for emergency and primary care providers alike. For many decades now, it has been among the leading causes of hospitalization in the industrialized world. The most common presentation to the emergency department is an acute decompensation in a patient with a known diagnosis of chronic CHF. This is because patients with this disease tend to present multiple times to the hospital as their disease progresses. In addition to the millions of patients that are living with this CHF, 1 million new diagnoses are made each year in the United States alone.

We often underestimate how very serious a diagnosis CHF is. Sudden cardiac death from arrhythmia is a real risk in patients with CHF and overall mortality, especially in patients with repeat hospitalizations, exceeds that for many types of advanced cancer.

Background And Classification

Congestive heart failure (CHF) is a broad term that encompasses the pathophysiological end result of a multitude of disease processes. It can be most simply defined as a structural or functional disorder of the heart causing an impaired ability of the ventricles to fill or pump blood. There are many way to classify CHF, but it is commonly described using these descriptors:

- Acute vs. Chronic
  - Chronic CHF is much more common and develops over time, often months to years
    - The heart typically grows in size over time as it fails

- In ischemic heart disease, chronic valvular disease and chronic uncontrolled hypertension, this growth and remodelling process is its attempt to compensate for its decreasing function
- In an acute exacerbation of chronic CHF, cardiomegaly is typically present, with the additional features, such as pulmonary edema and peripheral edema superimposed

- Acute CHF may occur in the setting of an acute myocardial infarction or an acute valve rupture where the heart is suddenly unable to generate the same stroke volume it did before
  - This might present as sudden ("flash") pulmonary edema and/or shock
  - The heart size may be normal, as it has not had time to change in size

- Left vs. Right-sided
  - Left-sided failure
    - This refers to failure of the left ventricle to pump blood forward (causing hypotension and hypoperfusion) and the resulting backup of blood into the lungs (pulmonary edema)
  - Right-sided failure
    - This refers to failure of the right ventricle to pump blood forward
    - This results in a failure of the right ventricle to "feed" the left ventricle its preload) and a back-up of blood (causing jugular venous distension, hepatomegaly and peripheral edema).
    - The commonest cause of right-sided failure is left-sided failure, and patients often present with manifestations of both
    - Isolated right side failure can occur acutely (for example, in pulmonary embolism) or chronically (for example, in COPD)
- Systolic vs. Diastolic
  - Systolic failure is usually what we refer to when we talk about CHF, this refers to a failure of the ventricle to empty its blood or its "squeeze"
  - Some patients, especially those with longstanding hypertension and left ventricular hypertrophy, might not have a problem with squeeze (e.g. their ejection fraction will be normal) but with their ventricle's ability to relax and hence fill with blood during diastole
  - This so-called "diastolic dysfunction" typically results in the pump backup symptoms of CHF: pulmonary and peripheral edema
- Low output vs. High output
  - Low failure is much more common and usually what we refer to when we talk about CHF
  - In both types of CHF (high and low output) the cardiac output is insufficient to meet the body's needs but in high-output failure the problem is not the heart, which is working overtime, it is increased demand (like thyroid storm or massive peripheral vasodilatation)
- By Etiology
  - The big three causes of CHF throughout most of the world are: ischemia, chronic hypertension and chronic valvular disease (e.g. chronic mitral regurgitation or aortic stenosis)

- Other causes:
  - Infectious causes
    - Chagas disease (common in South America)
    - Post-myocarditis
  - Postpartum
  - Cardiomyopathy
    - Genetic types (either hypertrophic or dilated cardiomyopathy)
    - Chronic cocaine/methamphetamine/alcohol use
    - Nutritional (e.g. beri-beri)

Clinical Assessment

- History
  - Typically presents as undifferentiated dyspnea with lower extremity edema but could be more subtle
  - Other common symptoms:
    - exercise intolerance
    - generalized fatigue
    - clothes/shoes fitting tighter
    - chest discomfort
    - orthopnea (shortness of breath lying flat)
  - Risk factors: CAD, HTN, diabetes, obesity, smoking, high sodium diet, valvular heart disease
- Exam
  - Lungs
    - Tachypnea, increased work of breathing, dyspneic with short phrases
    - Crackles, wheezing decreased air entry at bases0
  - Heart
    - Tachycardia, S3 (a third heart sound is highly specific for CHF)
    - Abdominojugular reflux or jugular venous distention (JVD)

      Watch on EMRAP-HD – JVD
    - Edema - usually lower extremity but it can rise up to abdomen and cause scrotal edema and ascites
    - Should be symmetric edema – if unilateral consider DVT/PE
    - RUQ tenderness from hepatic congestion and distended hepatic capsule

- B lines seen with pulmonary edema
  EMRAP-HD video by Jacob Avila on ultrasound for pulmonary edema – *LINK*
- Other important things to look for:
  - Pericardial effusion
  - Pleural effusion
  - Signs of large PE
- Dilated RV (larger than LV) and dilated IVC
- Formal Echocardiogram
  - A recent formal echocardiogram in the patient's chart can be very helpful and give information beyond what is typically obtained from a bedside echo
- Blood tests
  - Electrolytes and renal function are important, especially when multiple medications are involved
  - Troponin is frequently measured and is important to trend in the setting of an acute onset of CHF without an obvious explanation
- Troponin assays are increasingly sensitive and will often be slightly positive at baseline in many patients with chronic CHF
  - BNP (Brain Natriuretic Peptide) must be interpreted with caution:
- BNP <100 pg/mL or NT-proBNP <300 pg/mL makes acute heart failure unlikely (proBNP cut-off is higher b/c half life is higher). LR- = 0.1
- BNP >500 pg/mL or NT-proBNP >1000 pg/mL acute heart failure syndrome likely. LR+ = 6
- Patients with chronic CHF often have chronically elevated BNP levels, making it hard to interpret
- BNP can also be elevated in COPD and pulmonary embolism because of their effects on the right ventricle
- We recommend using clinical signs rather than the BNP to make clinical decisions
  - Troponin is frequently measured and is important to trend in the setting of an acute onset of CHF without an obvious explanation
- Non-STEMI commonly occurs together with a CHF exacerbation (both cause and effect!)

Differential Diagnosis And Finding The Underlying Cause

- CHF may co-exist with or mimic any of the following:
  - COPD
  - PE
  - Pericardial effusion
  - Arrhythmia
  - Heart block

- Valvular disease
- Hyper/hypothyroidism
- Sepsis
- Alcohol withdrawal
- Severe anemia
- Aortic dissection
- Pneumonia
- If rapid onset of CHF think:
  - Ischemia
  - Valvular pathology
  - Arrhythmia

- Identifying any one of the following causes of CHF will result in very unique interventions:
  - STEMI
    - emergent cardiac catheterization
  - Acute valvular rupture or septal wall rupture
    - emergent surgical intervention
  - Pericardial tamponade
    - emergent pericardiocentesis
  - Alcohol withdrawal
    - sedation and supportive therapy with active airway management
  - Arrhythmia (e.g. atrial fibrillation with rapid ventricular rate)
    - Rate and possibly rhythm control
  - Medication non-adherence/Dietary indiscretion
    - A very common cause for presentation
  - Missed dialysis
    - Treated initially with nitrates and NIV but dialysis will be necessary in patient who do not produce urine
  - Physiologic stress (e.g. sepsis, DKA, pneumonia)
    - Treat comorbid conditions
  - Postpartum cardiomyopathy
    - Supportive
  - Thyrotoxicosis
    - Treatment of CHF in thyrotoxic states can be difficult

- Beta-blockers are generally contraindicated in the acute phase of a CHF exacerbation but they may be given with caution in thyrotoxic states
- A short-acting, titratable beta-blocker, such as esmolol, may be helpful here, diltiazem for rate control is another option

Initial Resuscitation Of CHF With Pulmonary Edema

- Flash pulmonary edema is acute left sided "backward" failure
- Classic clinical appearance
  - Patient sitting up, tachypneic, diaphoretic, cool and ashen skin, coughing, +/- pink frothy sputum, tachycardic, usually hypertensive
  - Was this in the setting if chest pain/ischemia?
    - Maybe this is a STEMI that needs cath?
  - Can you hear a murmur?
    - Is this a valve rupture that may require a surgical intervention?
  - Assess volume status
    - Loop diuretics are indicated in patients with volume overload
- Two treatments to start immediately on arrival
  - Nitroglycerin
  - Non-invasive ventilation (NIV)
- Nitroglycerin
  - IV or SL
  - IV start at 10 mcg/min but titrate up quickly to 100 mcg/min and then to 300-400 mcg/min if needed (and the patient's blood pressure tolerates)
  - Many providers start much higher than this
    - Can start with sublingual load while drip is getting setup -- a sublingual tab is usually 0.4 mg -- that is 400 mcg. Probably about 100 mcg is absorbed and probably less than that if they are in shock
    - Many EMS protocols allow up to 3 SL nitroglycerin tabs 5 minutes apart -- that's a lot of nitro!
  - Hypotension with nitroglycerin
    - Usually transient, just stop or decrease the dose
    - Also think about RV infarct, recent sildenafil use, hypovolemia, pericardial tamponade, and aortic stenosis -- nitro is contraindicated in these cases
- Non-invasive ventilation (NIV)
  - Reduces work of breathing
  - PEEP opens up alveoli and improves oxygenation

- Both CPAP and BiPAP are typically delivered via a face mask that seals tightly around the patient's mouth and nose
  - CPAP - continuous positive airway pressure
    - Just gives one continuous pressure (PEEP)
    - Can also set the FiO_2
  - BiPAP - bilevel positive airway pressure
    - Senses when the patient is inhaling and provides inspiratory pressure
    - Senses when exhaling and provides an expiratory pressure (PEEP)
    - You set both of these as well as the FiO_2
  - BiPAP vs CPAP?
    - EMRAP February 2014 Paper Chase – *LINK*
    - Neither is clearly superior to the other
    - If the patient is not tolerating BiPAP try switching to CPAP since it is less for them to coordinate with
- To consider non-invasive ventilation patient should be:
  - Awake
  - Able to follow basic commands
  - Protect their airway
  - Not actively vomiting
  - Not having seizures
- Initial CPAP Settings
  - Initial pressure settings are usually around 10 cm H20 and then adjust based on patients comfort or clinical response. Pressures range from 4 to 20 cm H20.
  - Max PEEP of 15. Above 15 cm H20 can open the esophageal sphincter
- Initial BiPAP Settings
  - Common initial inspiratory positive airway pressure (IPAP) is 10 cm H20 (larger patients may need 15 cm H20)
  - Expiratory positive airway pressure (EPAP) is 5 cm H20
  - Adjust from there usually by 2-5 cm H20 at a time
- Rate of 10-12 breaths per minute (can increase rate if needing to get rid of more CO_2)
  - Rate is a backup - if they need BiPAP to initiate a breath they may need to be intubated
- FiO_2 initially is set at 100% and then titrated down once stabilized

- Angiotensin Converting Enzyme Inhibitors (ACE-Is)
  - ACE-Is reduce afterload and may have a role in patients is not improving with NIV and nitro
  - Commonly used is enalapril

- 1.25 mg/dose IV over 5 minutes q6hr; doses up to 5 mg/dose IV q6hr have been administered
- Lower doses used in poor renal function

- Loop diuretics
  - The role for loop diuretics like furosemide is still evolving, with many experts seeing them as routinely indicated and others questioning their first line role
  - These appear to be most appropriate in patients who are volume overloaded
    - Moreover, patients may need redistribution of fluids before diuresis (achieved with NIV and nitrates)
    - Not all patients with acute decompensated CHF are hypervolemic, some are euvolemic, and even worse some are hypovolemic and may actually need a little volume or pressors if developing shock
    - Less than half of patients with flash pulmonary edema are hypervolemic
    - In an exacerbation we typically double their home dose and since the conversion is 2:1 for oral to IV, you just give their same home dose but by IV route. Ex: they take 40 mg PO, give 40 mg IV and this is equivalent to double their dose
    - Furosemide infusions are recommended by some
    - Other Controversies:
      - Do high doses of furosemide worsen renal function?

        EMRAP Sept 2017 – *LINK*
      - Does it matter how quickly you administer the furosemide?
        - EMA January 2018 Abst 4 – Door to furosemide Time – *LINK*
    - Options other than furosemide (other loop diuretics)
  - Torsemide 10-20 mg IV
  - Bumetanide 1-4 mg IV
    - Check for and treat hypokalemia and hypomagnesemia
- Vasopressors
  - Indicated in shock states (see next section)
- Nesiritide
  - B natriuretic peptide
  - This was recently a very popular drug for CHF until the balance of evidence turned against it
- Digoxin
  - Still used in many countries
  - Mild improvement in cardiac output and rate control in atrial fibrillation while at rest
  - May improve symptoms but no effect on mortality
- Rotating tourniquets and phlebotomy

- These are old techniques to help reduce the circulating blood volume and, in the case of phlebotomy, reduce the viscosity of the blood
- Both may still have a role in austere (e.g. field) environments

- Endotracheal Intubation
  - We can usually avoid intubation with aggressive medical therapy
  - Pearls for intubation
    - Keep them sitting upright for preoxygenation, until you push meds
    - Go fast, use apneic oxygenation, and have push dose pressors ready
    - Resuscitate before you intubate, have suction ready

Initial Resuscitation Of CHF With Cardiogenic Shock

- CHF with cardiogenic shock is acute left sided "forward" failure
  - This may occur together with or without backward failure (pulmonary edema)
- Assess for causes that need immediate intervention (see above)
- Important potential causes:
  - Massive PE
  - Sepsis
  - Hypovolemia
- Treatment
  - Try small fluid boluses (e.g. 250 cc of saline), frequently re-assessing respiratory status
  - Vasopressors
    - There are many choices here
    - Norepinephrine, epinephrine and dobutamine are all considerations
  - An Intra-aortic balloon pump (IABP) can be temporarily placed as a way to stabilize the patient (as a "bridge") to catheterization or surgery

New Onset CHF

- New onset CHF may either be:
  - an acute presentation new onset heart failure or, much more commonly,
  - the first presentation of a process that has been slowly getting worse over time and and now finally causing symptoms
- In general, patients presenting with signs of CHF for the first time are admitted for further evaluation

Disposition

- The management of acute, life-threatening failure, as described above, is the domain of the emergency provider
- However, once a patient is stabilized, the ongoing inpatient management, which involves the complex interplay of multiple medications and co-morbidities, is the the domain of the internist
- Admit vs discharge
  - Patients with severe presentations, such as acute pulmonary edema or shock, are generally admitted to intensive care or monitored settings
  - Patients with milder exacerbations can often be managed on a regular ward or even in an ED observation unit
  - Well controlled patients with milder presentations and excellent follow-up may be able to go home in some cases
    - Try increasing their home meds for a few days, close outpatient follow up, and strict return precautions

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