Pulmonary Edema

Cline: ch. 00 ; ch. 00
Brady pp 583; 694
(+/or other standard
paramedic text)

Objectives

Upon completion of this lecture you should be able to discuss :

- Epidemiology and causes
- Pathophysiology and classifications
- Clinical presentation of CHF/ Pul. Ed.
- History (present illness, medications)
- Physical findings
- Management differences

Epidemiology

Frequency of CHF

- More than 3 million cases in US
- More than 400,000 new patients yearly

Prevalence of CHF

- 2% of general population

Race

- African Americans : 1.5 times more likely to die of CHF than whites

Prognosis and Mortality

- Mortality rates range from 10% in patients with mild symptoms to 50% with advanced
- Median survival
  - 3.2 years for males
  - 5.4 years for females
- Short term results of initial treatment
  - usually good, regardless of cause.

Causes of CHF

- Coronary artery disease : most common cause
  - Loss of left ventricular muscle +/or
  - Ongoing ischemia
  - Chronic CHF that has been exacerbated
    - Important to identify the precipitating cause
      - Diet
      - Noncompliance
      - Hypertension
      - Other (PE)

Precipitating Events

- Medications changes or noncompliance
- Dietary indiscretion (salt)
- Cardiac ischemia
- Cardiac dysrhythmias
- Pulmonary embolus
- Physical or environmental stresses (heat stroke deaths)
- Iatrogenic volume overload
- Systemic processes
  - Pregnancy
  - Hyperthyroidism
  - Hypothyroidism
- Other
  - Hypertension
  - Valvular heart disease
  - Myocarditis

Congestive Heart Failure (CHF) Pathophysiology

- Loss of Starling forces
  - Loss of the ability to keep the interstitium and alveoli dry
  - When blood oncotic or "pulling" pressure (generally 25 mm Hg) is no longer greater than pulmonary capillary pressure (normally 10 mm Hg)

CHF Pathophysiology

- Failure of pump function
- Failure to maintain adequate circulation of blood.
  - Inadequate renal blood flow
    - Increased renin-angiotensin
    - Increased ADH (vasopressin)
- Pulmonary edema
  - Most severe CHF
  - Develops because of increase of lung fluid secondary to leakage from pulmonary capillaries into the interstitium and alveoli of the lung

Functional Classification of CHF

NY Heart Association

- Class I : not limited with normal physical activity
- Class II : ordinary physical activity results in fatigue, dyspnea.
- Class III : marked limitation in normal activity
- Class IV : symptoms at rest or with any physical activity.

Acutely Ill Patients with Possible CHF

Diagnosis before therapy ??

- No
- The diagnosis not needed before initial therapy
  - all critical or unstable medical patients receive:
  - ABCDE / O2 M3 Bld IV Glu
- COPD and Pulmonary Edema receive much the same initial therapy except high-flow O2
- The standard of care has been shotgun therapy
  - treating for both CHF and acute pulmonary process such as asthma
  - with both diuretics and beta agonist inhalation

Diagnosis of CHF Diagnosis in Acutely Ill Patients

- Diagnosis after therapy has been initiated
- Traditionally, CHF diagnosis was based mostly on:
  - clinical criteria [Framingham; others]
    - History and Physical
    - Evidence of ventricular failure
    - Response to therapy
- New diagnostic test

New Diagnostic Approaches to Treating Acute CHF

New rapid BNP: Beta Natriuretic Peptide

- Test for distinguishing "heart vs lung"
  - High BNP levels help distinguish CHF from other causes of acute dyspnea
- At "point of care" [like an accucheck …]
- BNP levels :
  - 40 : non-CHF dyspnea (pneumonia, COPD / RAD,…)
  - 1000 + : CHF

Clinical Presentation of CHF / Pulmonary Edema

History

cc : SOB

- SOB, at rest
- +/- chest pain
- Sudden onset
- Dyspnea on exertion (DOE)
  - most sensitive symptom
  - yet not specific (60%)
- Anxiety
- Orthopnea
- Paroxysmal nocturnal dyspnea (PND)
- Cough productive of pink, frothy sputum
- Edema

Presentation of CHF History

Nonspecific symptoms:

- Weakness
- Lightheadedness
- Abdominal pain
- Wheezing
- Nausea

PMH

- Cardiac medications
  - Norvas
  - Lisinopril (Prinivil and Zestri)
- Hypertension
- Angina
- Prior MI
- Valvular heart disease
- Alcohol use

Presentation of CHF PE

Across the Room

- Upright
- Agitated
- Using accessory muscles of respiration

Airway:

- Pink frothy

Breathing:

- +/- wheezing
- " sniff " ; pursed lips

Circulation:

- Pale, cool
- Diaphoretic
- Mottled: gray and cyanotic

Vitals:

- **O2 sat**: often less than 86%
- **BP**: often elevated; 250/140
- **HR**: ST (or A-fib with RVR)
- **RR**: increased
- **T** cold / clammy

Continued PE in CHF

NECK

- Jugular venous distention (JVD)

CHEST:

- wheezes
- wet rales

CV:

- aortic or mitral murmurs
- "S_3 ; S_4" (if SBP 220+)

EXTR:

- Edema
- Lower extremity edema may not be present in the acute phase

PULSES

- Pulsus alternans
  - alternating weak and strong pulse
  - indicates: depressed LV function

EKG in CHF

- Nonspecific
- R/O cardiac ischemia, MI
- R/O prior myocardial infarction (MI)
- R/O cardiac arrhythmias
- LV hypertrophy / chronic hypertension changes

Emergency Care of CHF

- High flow O2 NRB
- cardiac monitoring and continuous pulse oximetry.
- elevate the head of the bed (to reduce venous return)
  - Even legs dangling
- Start nitrates and then nitrates and then more nitrates and then diuretics
- Continuous positive airway pressure (CPAP)
- Consider
  - Nesiritide (BNP)
  - Inotrope dobutamine

NTG

- NTG tab(s) 0.4mg - 0.8 mg SL
- Nitrospray: single spray (0.4 mg) equivalent to single 1/150 SL tab; may repeat q3-5min, up to a maximum of 1.2 mg
- NTG 2" paste (to to chest wall; upper arm)
  - this will maintain NTG levels until IV NTG is started
- NTG IV start at 20 mic/min, increasing 5-10 mic/min increments q3-5min

Non-NTG Therapy of CHF / Pulmonary Edema

- Lasix 80-120 mg IV
- Morphine 4 mg IV (controversial)
  - both an anxiolytic/anti-anxiety and an analgesic
  - venodilation effect
    - reduces preload
  - arterial dilatation
    - reduces afterload (SVR) and increases cardiac output.
  - evidence that its use may increase rate of intubation
    - naloxone can reverse

CPAP and BiPAP Therapy of CHF / Pulmonary Edema

- Continuous positive airway pressure (CPAP)
  - CPAP/BiPAP, if O2 Sat still not 95% after NRB
  - decreased need for intubation rates
  - however, no significant difference in short-term mortality
- Bilevel positive airway pressure (BiPAP)
  - decreased intubation rate
  - may improve ventilation and VS more rapidly then CPAP
  - higher incidence of MI associated with BiPAP
- BiPAP and CPAP are contraindicated
  - in the absence of an intact airway, and
  - in patients with an altered mental status or who are uncooperative or frightened.

Still More Therapies for CHF / Pulmonary Edema

- Phlebotomy
  - removal of 500 mL of blood former mainstay of therapy
  - used to decrease preload.
- Albuterol (as per local protocol). [Not epi.]
- ALOC +/or marked fatigue: ET
- BNP : 2 mic / kg IV bolus

Inotrope Therapy for CHF / Pulmonary Edema

- Dobutamine
  - current inotropic agent of choice
- Digoxin
  - No role in the emergency management of CHF
  - Delayed absorption
  - Diminished efficacy in patients with increased sympathetic [epi] effect.
  - No benefit in patient presenting with A Fib., with RVR
- In hypotensive with CHF [cardiogenic shock], dopamine and dobutamine are used together

"Heart vs Lung" and Guide in the Acute CHF Treatment

BNP in CHF :

- New rapid, inexpensive test for CHF diagnosis and prognosis
- Test for distinguishing "heart vs lung"
  - High BNP levels help distinguish CHF from other causes of acute dyspnea
- At the "point of care" [like an accucheck …?]
- BNP levels :
  - Non-CHF dyspnea (pneumonia, COPD/RAD,…) : 40
  - CHF : 1000 + [1076 +/- 138 pg/ml]

"Heart vs Lung? " - Avoid Steroids

In "COPD vs CHF patient" -- avoid injectable steroids

- Steroids (Methylprednisolone), IV or PO,
  - worsen heart failure
    - due to systemic sodium retention and volume expansion,
  - hypokalemia, and
  - hypertension
- Inhaled steroids
  - lack of systemic side effects
  - may be an option in a confusing heart vs lung patient in future

BNP in Acute CHF

- High BNP level:
  - marker for LV dysfunction
- When LV is stressed and stretched
  - the myocyte (by activating a gene transcription) responds by producing a small protein [hormone]
- BNP neurohormone effects
  - of renin-angiotensin-aldosterone system (which is also activated by LV dysfunction)

BNP in Acute CHF

- Increased levels of BNP (and levels of other natriuretic peptides, ANP and CNP)
  - Cause diuresis, vasodilitation, and natriuresis
  - Reduce preload
  - Reduce afterload

IV BNP for Treating Acute CHF

IV BNP, nesiritide (Natrecor) :

- MOA:
  - Sodium excretion (natriuresis)
  - Diuresis
  - Vasodilation
- May be indicated for acute decompensated CHF
- Response to treatment can be assessed
- Dose : 2 mic / kg IV bolus; then
  0.01 mic / kg / min IV continuous drip

BNP in Acute CHF (5)

- CHF pathophysiology causes elevated BNP
- BNP blood sampling can accurately detects CHF
- IV BNP can be an effectively treat CHF (1)
- IV BNP is as effective as standard treatment (1)
- BNP levels can be used as a :
  - measure the response to therapy
  - predictive marker for limitation and for sudden cardiac death in CHF patients (2)

(1) Calucci *NEJM* 2000 334:246-253
(2) Berger, *Circulation*

Summary

We have discussed pulmonary edema and its :

- Clinical presentation: Hx, PE findings, and laboratory findings - including the point of care BNP.
- Management
  - Nitrates, in particular
  - Including the use of CPAP and BiPAP .