Diuretics for CHF

Is That Smart Medicine??

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Case

65 yo. man presents with dyspnea

- Developed over the past 6 hours
- History of hypertension, tobacco use
- Diaphoretic, normal mental status
- Afebrile, HR 110, BP 180/110, RR 26, pox 88%
- Lungs — crackles, JVD
Case

Kerley B Lines

Cephalization

Kerley B Lines
Case

- 100% NRB — pulse ox 93%
- Monitor, IV, ECG
  - Sinus tachycardia, no acute ST/T-wave abnormalities
Case

What is the typical treatment in the first 5-10 minutes??

a) Morphine
b) Furosemide
c) Nitroglycerin
d) ACE-Is?
e) Non-invasive ventilation
Case

What is the typical treatment in the first 5-10 minutes??

a) Morphine
b) Furosemide
c) Nitroglycerin → high dose!
d) ACE-Is?
e) Non-invasive ventilation
FUROSEMIDE

Where’s the debate??
FUROSEMIDE

Got Juice?
1. ACC/AHA 2007 Guidelines for the Management of Patients with Unstable Angina/Non-STEMI: Executive Summary

- Summary of literature and recommendations for management of non-STE ACS
OUTLINE

- Definition
- Pathophysiology
- Pharmacological management
  - Preload reduction
  - Afterload reduction
  - Inotropic support
- Noninvasive Positive Pressure Ventilation
- Prehospital Issues
- Summary
CARDIOGENIC PULMONARY EDEMA

Definition:

- Leakage of fluid from the pulmonary capillaries and venules into the alveolar space as a result of increased hydrostatic pressure
- Inability of the LV to effectively handle its pulmonary venous return
ANATOMY

Normal

lungs
PATHOLOGY

Pulmonary edema!!
PHYSIOLOGY

preload

afterload

lungs

LV function
PATHOPHYSIOLOGY

Pulmonary edema!!

 preload

 afterload

 LV function

 lungs
PATHOPHYSIOLOGY

1. increased preload
2. increased afterload
3. decreased LV function

Pulmonary edema!!
GOALS OF TREATMENT

1. decrease preload
2. decrease afterload
3. improve LV function
GOALS OF TREATMENT

Total body hypovolemia or euvolemia

Pulmonary edema

lungs

body
CARDIOGENIC PULMONARY EDEMA

- **Note:** up to 50% of patients with cardiogenic pulmonary edema are euvolemic!!

- Treatment should be based not necessarily on fluid *removal*, but on fluid *redistribution*.
CARDIOGENIC PULMONARY EDEMA

Causes

- Excessive venous return (preload)
- Excessive SVR (afterload)
- LV dysfunction
  - disorders of contractility
  - disorders of rate and rhythm
PRELOAD REDUCTION

- Traditional treatment
  - Morphine
  - Furosemide
  - Nitrates
Preload reduction

• Diuresis
  ▪ increased afterload causes decreased RBF
  ▪ delayed effect: 30 – 120 minutes

• Direct vasoactivity
  ▪ venodilation — little evidence
  ▪ does this correlate with decreased preload?

- Direct vascular effects of furosemide on the human forearm vascular bed and dorsal hand vein
  - local administration of furosemide resulted in dose-dependent venodilatation
- Does this correlate with decreased preload?

- Post-AMI CHF
- Furosemide administered to 15 patients
- Significant reductions in filling pressures
  
  *only in patients that had diuresis*

- IV furosemide administration in acute CHF
- Significant reductions (17%) in CO during first 90 minutes
- CO gradually returned to baseline with diuresis

- IV furosemide (1 mg/kg) administration in AMI patients with LV failure
- Initial adverse hemodynamic effects
  - decreases in CO and SV during initial 90 minutes
- Parameters returned to baseline over next 60 – 90 minutes

- Class III or IV CHF patients given IV furosemide
- Early adverse hemodynamic effects
  - 20 minutes after administration
    - significant increase in HR, SVR (afterload)
    - significant decrease in SV
- Gradual return to baseline with diuresis
Kraus, et al (Chest, 1990)

- Effects of IV furosemide on PCWP over 1 hour in patients receiving nitrates and/or captopril
  - Furosemide alone or furosemide plus nitrates
    - increase in PCWP over initial 15 minutes
    - then decrease PCWP with diuresis
  - If *premedicated* with nitrates plus captopril
    - immediate and sustained decrease PCWP
Kraus, et al (Chest, 1990)

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Biddle, et al (Am J Cardiol, 1979)

- Hemodynamic studies carried out in patients with post-MI LV failure
- “Despite a prompt diuresis, the reduction in lung water is delayed for at least several hours after the administration of furosemide...”
SUMMARY — FUROSEMIDE

- Decreases preload through diuresis
  - Delayed effect

- No consistent data regarding immediate direct preload reducing effect

- Initial adverse hemodynamic effects
  - Increased SVR
  - Decreased SV, CO

- Initial activation of renin-angiotensin system
Furosemide should be considered a *third-line medication* in the treatment of cardiogenic pulmonary edema!
So what’s the problem with just giving it anyway and waiting...?
PREHOSPITAL TREATMENT

- Differential diagnosis for severe dyspnea and hypoxia is vast
  - Common in older adult patients
    - CHF/pulmonary edema
    - pneumonia
    - asthma
    - COPD exacerbation
    - pulmonary embolus
PREHOSPITAL TREATMENT

- Difficult to accurately diagnose in the field
  - How reliable is prehospital diagnosis?
  - Is empiric treatment safe? With which drugs?

- Compared NTG, furosemide, morphine in patients with presumed pulmonary edema
- Best outcome with nitroglycerin
- Adverse effects in patients receiving furosemide
  - > 25% later required fluid repletion, some hypotensive
  - significant electrolyte abnormalities
Hoffman, et al (cont.)

- 23% of patients were misdiagnosed and didn’t have pulmonary edema, inappropriately treated
  - worse outcome in patients receiving furosemide and/or morphine
  - no adverse effects in patients receiving NTG alone

- Evaluated outcomes in 599 prehospital presumed decompensated CHF patients
  - 18% misdiagnosed, inappropriately treated
    - most had asthma, COPD, pneumonia, bronchitis
    - patients receiving NTG alone — 2.2% mortality
    - patients receiving morphine and/or furosemide (+ NTG) — 22% mortality
Jaronik, et al. (Prehosp Emerg Care, 2006)

- Evaluated 144 prehospital presumed decompensated CHF patients given furosemide

  - 42% had a final diagnosis that was not CHF, furosemide considered “inappropriate”
  - 17% diagnosed with sepsis, dehydration, or pneumonia (without CHF), furosemide “potentially harmful”
  - 7/9 deaths in furosemide group
CONCLUSION — PREHOSPITAL TREATMENT

Prehospital misdiagnosis is common.

If patients are misdiagnosed and inappropriately treated for pulmonary edema with furosemide, morbidity and mortality increase.
SUMMARY

- Up to 50% of patients with severe AHF are not fluid overloaded
  - These patients don’t need diuretics and may be harmed
- If a patient is misdiagnosed and receives diuretics, there can be significant harm
- Diuretics don’t work quickly in AHF anyway, so...
...why rush to give the diuretics??

My practice → no diuretics until...

- I’m sure that the patient has AHF
- I’m sure that the patient is total-body fluid overloaded (usually by history and exam)
- In the meantime, I focus on fluid redistribution rather than removal
REMEMBER…

- Up to 50% of patients with cardiogenic pulmonary edema are euvolemic!!
- Treatment should be based not necessarily on fluid removal, but on fluid redistribution.
Thanks! questions:
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