Acute Decompensated Heart Failure (ADHF)

Introduction

- Clinical diagnosis by worsening S&Ss of HF.
- Another name is AHF. If the 1st episode of ADHF = de novo HF.

Epidemiology

- Most common cause of hospitalization in pts \geq 65 yo (Circ 2013;127:e6).
- High mortality and morbidity (50% re-hospitalization at 6 months. (Am Heart J 2010;160:885).
- Heterogeneous groups of patients: ACS, HTN crisis, shock, RV failure, preserved EF (50%) etc.

Precipitating factor

• Medication noncompliance, diet noncompliance, ACS, infection, HTN, arrhythmia, worsening RF, NSAIDs, thyroid, anemia, PE, pregnant, iatrogenic etc.

Prognosis (ADHERE. JAMA 2005)

- Overall in-hospital mortality = 4%. 30% mortality at 1 year. 50% recurrent hospitalization at 6 months.
- If BUN ≥43, SBP ≤115 and Cr ≥2.75, in-hospital mortality rate of 20%.

Evaluation

\bullet Know patient's clinical hemodynamics status. "Warm - Wet - Cold - Dry"

<u>
Congestion</u>: Orthopnea, †JVP, rales, (+) HJR, ascites, edema, Valsalva square wave BP, PSM, S3.

<u>↓ Perfusion</u>: ↓Mentation, narrow pulse pressure (PPP $\leq 25\% \sim CI \leq 2.2$. JAMA 1989;261:884), pulsus alternans, hypotension, cool extremities, ↑Cr, ↑LFT, ↑Lactic acid. (Nohria A. JACC 2003;41:1797).

• Crepitation, cephalization on CXR have low sensitivity (<30%) in pt with history of chronic HF.

• BNP >100 has 90% Se, 76%Sp for diagnosis of ADHF in pt presented to ED with dyspnea (BNP. NEJM 2002). BNP should not be used in isolation from clinical. May compare to "dry BNP"

• Goal of therapy: 1. Improve symptoms; 2. Prevent and restore end-organ damages by shift hemodynamics to "Dry and warm".

Drug

Loop Diuretics

To Decrease Preload

Loop Diuretic

• Sigmoid dose-response curve. No response until threshold dose is reached. Minimal additional response after that.

• Once effective dose established, increased frequency of dosing for more urine output.

- Initial IV dose should be \geq home daily dose.
- Furosemide 80 oral ~ 40 iv = torsemide 40mg = Bumex 1 oral = 1 iv.

Diuretics resistant

- \uparrow Dose, \uparrow frequency, change to IV infusion, adding 2nd diuretic (thiazide, spironolactone).
- Ultrafiltration: Greater control but no greater weight loss compared to diuretics. More adverse effect (CARRESS-HF. NEJM 2012).

Bumetanide 1.0 mg 4 to 8 mg Furosemide 40 mg 160 to 200 mg 100 to 200 mg Torsemide 10 mg Thiazide Diuretics Chlorothiazide 1000 mg 500 mg Sequential Nephron Blockade Chlorothiazide 500 to 1000 mg (IV) once or twice plus loop diuretics once; multiple doses per day

CONGESTION

+

В

wet-warm

(N=222)

C

wet-cold

(N=91)

Maximum Single Dose

A

drv-warm

(N=123)

L

dry-cold

(N=16)

DEQUATE PERFUSION

Initial Dose

• "Renal dose dopamine": Not selectively 1 renal blood flow or prevent renal failure (ROSE. JAMA 2013).

• Tovaltan - Vasopressin receptor blocker: Greater weight loss and less symptoms at 1 day but no Δ CV death or rehospitalization at 10 months. (EVEREST. JAMA 2007).

• Serelaxin - Recombinant human relaxin-2: \downarrow dyspnea, \downarrow length of stay, \downarrow CV death at 180 days in both HFrEF and HFpEF with AHF (RELAX-AHF. Lancet 2013).

To improve perfusion (afterload reduction and/or increase contractility)

- IV vasodilator
- \uparrow afterload $\rightarrow \uparrow$ cardiac output $\rightarrow \downarrow$ PCWP
- \downarrow preload \rightarrow rapid symptom relief
- No side effect of inotrope/pressors eg. arrhythmia (AF, VT/VF), MI (from 1 MVO2)
- Should be avoid in hypotension, MS, AS

	Nitroglycerin	Nitropusside	Nesiritide
Mechanism	Nitric Oxide	Nitric Oxide	BNP
Onset of action	Mins	Mins	hours
Usual dose	10 - 200 mcg/min	0.1 - 5 mcg/kg/min	2 mcg/kg iv bolus then 0.01 – 0.03 mcg/kg/min
Effect on CPWP	Ļ	$\downarrow\downarrow$	$\downarrow\downarrow$
S/E	Headache (20%) Hypotension	Thiocyanate Hypotension	? worsening RF Hypotension
Expense	\$	\$\$	\$\$\$\$
Note	V > A dilatation Decrease preload Tachyphylaxis	V = A Very fast onset	? Diuresis effect Cannot measure BNP VMAC. JAMA 2002 ASCEND-HF. NEJM 2011

IV Inotrope

• \uparrow contractility \rightarrow \uparrow cardiac output \rightarrow \downarrow PCWP

- Choosing based on hemodynamic effects. No data consistently shows improved clinical outcome.
- Use only when needed (severe hypoperfusion with hypotension). Wean off as soon as possible.

• Bridge to definite treatment eg. revascularization, resolution of precipitating factors, optimization of preload and afterload stage.

	Dopamine	Dobutamine	Milrinone
Action	<u>α1 β1</u> β2 DA adrenergic agonist	β1 β2 (α1) adrenergic agonist	PDE-3 inh increase cAMP
Onset of action	mins	mins	hours
Usual dose	2-20 mcg/kg/min	2-20 mcg/kg/min	0.125 - 0.625 mcg/kg/min
Effect on afterload	 ↑↑	$\downarrow\downarrow$	↓↓↓
S/E	VT/VF AF/Afluter	VT/VF AF/Afluter may ↓ hospitalization ↑ QoL but ↑ mortality	Pt who was on beta blocker 10% hypotension 5% Aflutter/Afluter OPTIME-CHF

• Other inotropes that may be consider: Dopamine, Norepinephrine. Levosimendan - Calcium- sensitizing agents: Positive inotropic with vasodilator. Uncertain clinical efficacy and safety (compare to dobutamine)

Other management

- Treat precipitating causes.
- Na/fluid restriction. Carefully monitor I/Os, weight, electrolytes.
- O2 if hypoxia. NPPV (3CPO NEJM 2008).
- DVT prophylaxis. Opiate if needed.
- Chronic HF meds should be continued unless there are contraindications or hemodynamic instability.
- If hypotension, severe hypoperfusion, may consider PA cath and or arterial line.
- When inotrope is not enough consider OHT, mechanical circulatory support (MCS), or palliative care.
- Before discharge: Reverse precipitating cause, start/uptitrate chronic HF meds, education. Identified patient who may benefit from revascularization or devices.