HEART FAILURE

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Definitions

- HF: Progressive clinical syndrome in which the heart is unable to meet the metabolic and perfusion demands of the body
- Systolic HF: weakness in ventricular pumping/ contracting- most common
- Diastolic HF: weakness in ventricular filling. Also known as HF with preserved LVEF
- Other parts of heart muscle may cause HF, but most commonly ventricular

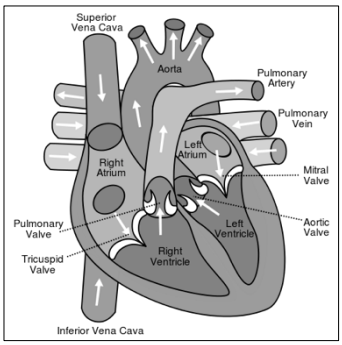
Definitions

- CHF → HF
- Acute Heart Failure (AHF): acute decompensation in signs/symptoms of HF
- Cardiomyopathy, LV dysfunction: not HF but may cause it

Epidemiology

- Risk doubles with every decade of life
- Major cause for hospitalization among elderly
- Main two contributors are AMIs and HTN
- 5-Year mortality rate around 50% for all pts with HF
- Around half of deaths from HF come as sudden cardiac death, indicating arrhythmias
- About 50% of HF pts have preserved LVEF (DHF)

Heart Anatomy



Etiology

- Any condition that affects cardiac tissue and its ability to contract or stretch can lead to HF
- Ventricular dysfunction can be L- or R-sided
  - L-side → pulm congestion, R-side → systemic congestion
  - Often spreads from one side to another
- SHF Vs. DHF
  - Coexist in up to 2/3 of patients
  - Similar mortality rates
  - Similar s/s

Etiology

- SHF- Reduced LVEF
  - AMI
  - Dilated cardiomyopathy- alcohol
  - Ventricular hypertrophy- 2/2 valves, pulmonary HTN, shunts, HTN
- DHF- Preserved LVEF
  - Myocardial stiffening 2/2 AMI, ventricular hypertrophy, or infiltrative myocardial disease (amyloidosis, sarcoidosis, fibrosis..)
  - Mitral or tricuspid valve stenosis
  - Pericardial disease

Pathophysiology

- The pathology is initiated with a loss in CO
- The heart initiates a number of compensatory mechanisms:
- 1) Tachycardia and Increased contractility
  - Initiated by SNS to increase CO
  - CO = SV X HR
  - Can lead to myocardial ischemia, arrhythmias, shortened diastolic filling time, decreased β1 receptor sensitivity

Pathophysiology

- 2) Ventricular hypertrophy
  - Initiated to build more muscle to increase CO
  - Mediated by RAAS and neuro-hormonal remodeling
  - Leads to fibrous, stiff, and weak ventricles (SHF, DHF)
  - Increased arrhythmia risk
  - Increased demand ischemia

Pathophysiology

- 3) Vasocnostriction
  - Initiated as reflex to ensure perfusion 2/2 low CO
  - Mediated by RAAS and SNS
  - Increases afterload
  - Increases cardiac ischemia due to extra effort
  - Decreases SV, feeding into compensatory mech.
- 4) Increased preload
  - Mediated through RAAS activation and Na/water retention

Pathophysiology

  - Increased intravascular volume leads to increased contractility and SV (Frank-Starling mechanism)
  - The weak heart can't keep up with increased volume, which backs up and leads to pulmonary and systemic congestion/edema.
- Current mode of managing HF focuses on targeting various elements of the *neurohormonal model*

Clinical Presentation & Diagnosis
Symptoms

- Congestion and hypoperfusion lead to the majority of symptoms
- Congestion
  - Pulmonary edema 2/2 failing L ventricle
    - SOB/Orthopnea/etc.
  - Systemic edema 2/2 failing R ventricle
    - Fluid retention/polyuria/nocturia/etc.
    - Peripheral edema and weight gain are most recognizable

Clinical Presentation & Diagnosis
Symptoms

- Hypoperfusion
  - ARF
  - Cold extremeties/poor capillary refill
  - AMS
- Other general symptoms may include:
  - Fatigue
  - Abdominal pain and bloating
  - Nausea

Clinical Presentation & Diagnosis
Signs

- Physical exam may reveal:
  - Pulmonary rales/edema
  - S_3 gallop
  - Pleural effusion
  - Tachycardia
  - Peripheral edema
  - Jugular venous distention (JVD)
  - Cyanosis of the digits
  - Cool extremeties
  - Cheyne-Stokes respiration
  - Polyuria

Clinical Presentation & Diagnosis
Labs

- BNP > 100 pg/mL
- EKG- LV hypertrophy, myocardial ischemia, arrhythmias
- SCr
- CXR
- Echocardiogram- the single most useful test
  - Valves, LVH, LVEF, structure abnormalities
- CBC
- Any other labs to assess for any co-morbidities/ risk factors, i.e. lipid panel, angiogram, A1C, etc.

Clinical Presentation & Diagnosis
History

- Medication Hx
  - Negative inotropic agents- antiarrhythmics, βBs, CCBs, itraconazole, terbinafine
  - Na/H_2O retention- NSAIDs, COX-2 inhibitors, glucocorticoids, androgens, estrogens, high-dose salicylates, thiazolidinediones, sodium-containing drugs
  - Cardiotoxic agents- doxorubicin, cyclophosphamide, etc.
- FH, PMH, SH
- Current complaints

Classification of HF

- NYHA Vs. ACC/AHA
  - NYHA is a functional classification- based on ability to function with minimal restriction- subjective
  - Pts can move back and forth between NYHA stages
  - ACC/AHA staging complements NYHA
  - Pts cannot move back in ACC/AHA stages
  - Both systems together enable clinicians to better assess risk factors, management, and prognosis

Classification of HF

NYHA CLASS	AHA/ACC STAGE	DESCRIPTION	EXAMPLE
N/A	A	Pts at high risk for heart failure but without structural heart disease or symptoms of heart failure.	HTN, CAD, DM
I	B	Pts with (structural) heart disease but without limitations of physical activity. Ordinary physical activity does not cause undue fatigue, dyspnea, or palpitation	Hx AMI, LVH, LV dysfunction
II	C	Pts with cardiac disease that results in slight limitations of physical activity. Ordinary physical activity results in fatigue, palpitations, dyspnea, or angina	LV dysfunction AND symptoms

Classification of HF

NYHA CLASS	AHA/ACC STAGE	DESCRIPTION	EXAMPLE
III	C	Pts with cardiac disease that results in marked limitation of physical activity. Although pts are comfortable at rest, less than ordinary activity will lead to symptoms	LV dysfunction AND symptoms
IV	C,D	Pts with cardiac disease that results in an inability to carry on physical activity w/o discomfort. Symptoms of HF are present at rest. With any physical activity, increased discomfort is experienced. Stage D refers to end-stage HF	Refractory HF, end-stage

Treatment Of Chronic Heart Failure
Goals of therapy

- Improve quality of life
- Minimize symptoms
- Minimize exacerbations and hospitalizations
- Slow progression of disease
- Prolong survival

Treatment Of Chronic Heart Failure
Approach to therapy

- Determine etiology and treat any underlying causes
- Classify HF stage
- Initiate non-pharmacologic therapy
- Initiate pharmacologic therapy if indicated
- Monitor therapy (IESC), re-assess stage, modify therapy as necessary

Treatment Of Chronic Heart Failure
Non-Pharmacologic Therapy

- Smoking and alcohol cessation
- Exercise tailored to pt- restrict when decompensated
- Na restriction < 2g/d
- General fluid restriction
  - < 2L/d if hyponatremia and/or persistent fluid O/L
- Daily weight measurements and logging:
  - Intervene if wt increase >1 kg in one day or 2 kg over 5 days

Treatment Of Chronic Heart Failure
Non-Pharmacologic Therapy

- Empower pt to self-care through counseling and providing tools for self-management
- Provide immunizations (annual Flu, pneumococcal)

Treatment Of Chronic Heart Failure
Pharmacologic Therapy*

- Stage A
  - Manage/reverse risk factors: HTN, DM, HLD, obesity, tobacco abuse, cardiotoxic agents
  - ACEI should be considered for patients with multiple CV risk factors (DM, atherosclerotic disease)

* Based on the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Circulation*. 2013:128

Treatment Of Chronic Heart Failure
Pharmacologic Therapy*

- Stage B
  - ACEI (EL-A) should be used in all pts with reduced EF to prevent symptomatic HF and *reduce mortality*
  - β-Blockers *with mortality benefit* should be used to reduce mortality in all pts with HF*r*EF (EL-C) and in patients with HF*r*EF AND Hx AMI/ACS (EL-B)
  - In all pts with Hx AMI/ACS statins should be used to prevent symptomatic HF and CV events (EL-A)

* Based on the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Circulation*. 2013:128

Treatment Of Chronic Heart Failure
Pharmacologic Therapy*

- Stage B (cont'd)
  - For all pts with structural heart defects including LVH: BP should be well controlled (EL-A)
  - NDHP CCBs may be harmful in asymptomatic pts with low LVEF

* Based on the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Circulation*. 2013:128

Treatment Of Chronic Heart Failure
ACEIs

- Can prevent HF in high risk pts (Stage A)
- Reduce afterload and preload, block neurohormonal remodeling, improve cardiac hemodynamics (SV, CO, MAP, SVR..)

Treatment Of Chronic Heart Failure
ACEIs

- Start at low doses, titrate slowly to target dose used in clinical trials or highest tolerated dose
  - Lisino: initial 2.5 mg QD, target 20-40/d
- Watch for cough, s/s angioedema, HoTN
- Check renal fcn and lytes within 1-2 wks of initiating or dose change
- ACEIs can be started in pts with Cr baseline up to 2.5-3 mg/dL
- Cr bumps of up to 0.5 mg/dL are tolerated

Treatment Of Chronic Heart Failure
ARBs

- ARBs can be treated like ACEIs when it comes to MOA, AEs, dose titration, monitoring, etc.
- Usual dosing
  - Losartan: 25-50 mg QD, target 50-100 mg QD

Treatment Of Chronic Heart Failure
Beta Blockers

- Slow/reverse neurohormonal remodeling, improve EF, reduce arrhythmias, reduce myocyte ischemia
- Benefit seen from βB in all pts with HF*r*EF, with or without CAD, with or without DM
- NOT A CLASS EFFECT! The benefit specific to bisoprolol, carvedilol, and *long-acting* metoprolol (succinate)
- Cautious use in pts with reactive airway disease or asymptomatic bradycardia

Treatment Of Chronic Heart Failure
Beta Blockers

- βB should be initiated at *very low* doses to avoid acute decompensation
- Titrate slowly if initial dose tolerated
- Closely monitor vital signs and symptoms after initiation and dose changes
- Do not increase dose until AEs with previous dose disappear

Treatment Of Chronic Heart Failure
Beta Blockers

- AEs- rarely a cause to permanently d/c βB
  - Fluid retention
  - Bradycardia
  - Hypotention
  - Fatigue
- Usual dosing:
  - Carvedilol: initiate 3.125 BID, titrate to 25 mg BID (50 BID for pts > 85kg)
  - Metoprolol succinate (XL): initiate 12.5-25 mg QD, titrate to 200 mg QD

Treatment Of Chronic Heart Failure
Pharmacologic Therapy*

- Stage C
  - Emphasize self-care: weight monitoring, Na restriction, med compliance, physical activity
  - Diuretics recommended to improve *symptoms* in pts with HF*r*EF with evidence for fluid retention
  - ACEIs recommended to improve *morbidity and mortality* in symptomatic pts with HF*r*EF (EL-A).
  - For those intolerant to ACEIs, ARBs as alternatives have same *morbidity and mortality* benefit
  - Use of βB *with mortality benefit* is recommended for all pts with HF*r*EF to reduce *morbidity and mortality*

* Based on the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Circulation*. 2013:128

Treatment Of Chronic Heart Failure
Diuretics

- Loops or thiazides are acceptable, but loops are much more potent diuretics
- Thiazides are acceptable if treating HTN if pt has *mild* fluid retention
- Should be given to all pts with fluid retention

Treatment Of Chronic Heart Failure
Diuretics

- Furosemide is most commonly used
- Combine with Na restriction
- Low dose initiated then titrated to goal wt loss <1k g/d, then tapered off or reduced to maintain dry wt (euvolemia)
- Ceiling effect can be reached (resistance)
  - Add thiazide (metolazone)
  - Increase frequency of dosing to BID or TID
  - IV administration

Treatment Of Chronic Heart Failure
Diuretics

- Patients can be taught to adjust dose per wt change
- Usual dosing
  - Furosemide: 20-160 mg/d, start QD dosing, higher dose required for resistance or advanced CKD
- AE: lytes (K, Mg), ARF, HoTN

Drug	Furosemide (IV)	Furosemide (PO)	Bumetanide (IV/PO)	Torsemide (PO)
Dose Equiv.	20 mg	40 mg	1	20

Treatment Of Chronic Heart Failure
Pharmacologic Therapy*

- Stage C (cont'd)
  - Aldosterone receptor antagonists are recommended in symptomatic pts with HF*r*EF to reduce *morbidity and mortality* (EL-A)
  - *Addition* of ARBs may be considered in pts with HF*r*EF who are already on ACEIs and a βB and in whom an aldosterone antagonist is not indicated or tolerated (EL-A)
  - Routine combined use of ACEI, ARB, and aldosterone antagonist is potentially harmful

* Based on the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Circulation*. 2013:128

Treatment Of Chronic Heart Failure
Aldosterone Antagonists

- Eplerenone and Spironolactone
- Benefit thought 2/2 blocking aldosterone pathway, which is elevated in HF pts
- Cr should be ≤ 2.5 mg/dL in men or ≤ 2.0 in women (or est. GFR >30 mL/min), AND potassium should be less than 5.0 mEq/L, before starting treatment

Treatment Of Chronic Heart Failure
Aldosterone Antagonists

- Initiate at a low dose, check K and Cr within 2-3d and again at 7d after initiation, then monthly for the first 3 mo, then every 3 mo thereafter. Adding ACEIs or ARBs should trigger a new cycle of monitoring as above
- Usual dosing
  - Spironolactone: initiate at 12.5-25 QD, titrate to 25 mg QD-BID
- Eplerenone lacks endocrine AEs

Treatment Of Chronic Heart Failure
Pharmacologic Therapy*

- Stage C (cont'd)
  - The combination of hydralazine and isosorbide dinitrate is recommended to *reduce mortality* for African Americans with symptomatic HF*r*EF remaining symptomatic despite βB and ACEI/ARB (EL-A)
  - Hydralazine/isosorbide dinitrate can be useful to reduce *morbidity or mortality* in pts with symptomatic HF*r*EF who cannot tolerate ACEI or ARB (EL-B)

* Based on the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Circulation*. 2013:128

Treatment Of Chronic Heart Failure
Hydralazine/Isosorbide Dinitrate

- Nitrates reduce preload and increase nitric oxide (NO), hydralazine reduces afterload
- Available in fixed dose combination (BiDil) but can also be given seperately
  - BiDil: ISDN 20 mg, Hydralazine 37.5 mg
- Usual dosing: initiate at 1 tab TID, titrate to 2 tabs TID
- Poor compliance issues
- AEs: related to vasodilation (dizziness, HA), GI complaints

Treatment Of Chronic Heart Failure
Pharmacologic Therapy*

- Stage C (cont'd)
  - Digoxin can *reduce hospitalizations* in pts with HF*r*EF
  - May be added to the initial regimen in pts presenting with severe symptoms, or may be added later after the standard therapy had been started and pt remained symptomatic

* Based on the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Circulation*. 2013:128

Treatment Of Chronic Heart Failure
Digoxin

- Increases inotropy and may block neurohormonal remodeling
- Avoid in pts with SA or AV block
- Initial dose: 0.125 mg/d, titrate to target serum level of 0.5-0.8 mg/dL
- Cleared renally, hypo-K and hypo-Mg can predispose to toxicity
- AEs: GI (N/V, anorexia), visual disturbances, confusion, bradycardia/arrhythmias
- Monitor HR, lytes, digoxin level if indicated, Cr

Treatment Of Chronic Heart Failure
Pharmacologic Therapy*

- Stage D
  - End-stage HF: refractory to all therapy
  - Fluid restriction is recommended (1.5-2 L/d)
  - May become candidate for LVAD and heart transplantation
  - Continuous IV infusion of inotropic agents is reasonable to bridge to transplantation
  - Focus on hospice care/comfort care

* Based on the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Circulation*. 2013:128

Treatment Of Chronic Heart Failure
Anticoagulation*

- Patients with CHF who have AFib and an additional risk factor for cardioembolic stroke (HTN, DM, previous stroke, TIA, age ≥ 75) should be placed on chronic anticoagulation (EL-A)
- Low dose ASA is recommended in HF pts with prior Hx of ischemic disease (stroke, CAD)

* Based on the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Circulation*. 2013:128

Treatment Of Chronic Heart Failure
Diastolic HF (HF*p*EF)

- Diagnosis based on HF symptoms without proof of reduced EF
- Similar morbidity to HF*r*EF, but with better survival
- Few studies specific to HF*p*EF

Treatment Of Chronic Heart Failure
Diastolic HF (HF*p*EF)

- ACC/AHA recommends:
  - Optimal treatment of HTN and CAD
  - Use diuretics to control congestion
  - The use of βB, ACEIs, and ARBs is reasonable to control HTN but *has not not been shown to provide any mortality benefit* if used without HTN