Heart Failure
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Objectives
- Review the trajectory of heart failure as a clinical syndrome
- Describe the methods of classification of heart failure
- Describe current recommendations for goal-directed medical therapy (GDMT) across a continuum

Definitions
- Complex clinical syndrome that can develop from any cardiac disorder that impairs the ability of the ventricle to either fill properly or eject optimally.
  - The heart cannot pump enough blood to meet the metabolic demands of the body
  - Syndrome: HF is manifested and recognized by combinations of “hallmark” symptoms/signs

Presentations
- Dyspnea and fatigue
  - Impacts exercise and activity intolerance
- Extracellular fluid retention
  - Causes peripheral edema and pulmonary congestion
  - Impact on sense of well-being and quality of life

Causes
- Clinical syndrome with multiple possible etiologies.
- Regardless of the cause – there is a typical pathological remodeling that occurs and over time the remodeling/compensatory changes lead to
  - Progressive cardiac enlargement
  - Decline in cardiac function
  - Neurohormonal model of HF (TBDL)
Classifications and (more) terminology

- Variability in documentation of HF treatment plans, new billing requirements, etc.
- Different classifications are used to best describe patient presentation, acuity, subjective/objective findings.
  - Acute or chronic? Systolic or diastolic HF? Right or left-sided HF? Disease progression? Heart Failure symptoms?

Acute vs. Chronic

- Acute decompensated heart failure
  - G: improvement of sx, hemodynamics, volume status, ↓injury to heart/kidneys, initiating life-saving therapies
- Chronic heart failure
  - G: reduction of mortality, improvement of sx, QOL, ↓hospital admissions s/t ADHF.
  - Difference relates to patient presentation and hemodynamic stability – treatment goals reflect that

Right vs. Left Sided HF

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<thead>
<tr>
<th>Right Sided Failure</th>
<th>Left Sided Forward Failure</th>
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<tr>
<td>Decreased pulmonary perfusion</td>
<td>Decreased pulmonary perfusion</td>
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<tr>
<td>Dyspnea</td>
<td>Dyspnea</td>
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<td>Hepatic</td>
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<td>Cyanosis</td>
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<td>Decreased diuretic</td>
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<td>- Edema, edema, oozing, oozing</td>
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<td>- Shortness of breath</td>
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<td>- Cough, cough, cough</td>
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<td>- Crackles</td>
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AHA/ACC Heart Failure Staging

The American Heart Association/American College of Cardiology staging system classifies heart failure as a progressive disorder.
Left ventricular dysfunction begins with an initial insult to the myocardium, and even without any further insults, LV dysfunction continues to progress.

NYHA Functional Classification for Heart Failure

- Most commonly used system to assess functional capacity.
- Rating scale – so some variability in classification.
- Based upon patient report of heart failure symptoms with varying degrees of activity.
- Patients can move between classes.
- Other tests: 6MWT, maximal exercise testing, and peak $O_2$ consumption.

Killip Classification

- Originated from a study in 1967 in a CCU unit in the US
- Post-MI patients were evaluated for risk of death 30 days after the coronary event based upon hemodynamics and signs/symptoms of heart failure/shock at initial presentation.
- You might find the picture below familiar.
**Systolic vs. Diastolic HF**

- Systolic or diastolic dysfunction
- Systolic dysfunction
  - Heart failure with reduced EF
  - Abnormality of ventricular emptying due to impaired contractility or greatly excessive afterload
- Diastolic dysfunction
  - Heart failure with preserved EF
  - Abnormality of ventricular relaxation during diastole/ventricular filling

**New Names for Systolic and Diastolic Dysfunction**

- New terminology from the AHA/ACC Guidelines for 2013
- HFrEF (HF with reduced ejection fraction)
  - Replaces systolic dysfunction/HF
  - EF < 35-40%
- HFpEF (HF with preserved ejection fraction)
  - Replaces diastolic dysfunction/HF
  - EF > 50%
- Learn the new names, but you’ll still hear the old names thrown around.

**Systolic Dysfunction: HFrEF**

- The affected ventricle has a ↓ capacity to eject blood due to impaired myocardial contraction or pressure overload.
- Loss of contractility from myocyte destruction, abnormal function or fibrosis
- Often the LV wall thins and the cavity dilates – causing an eccentric hypertrophy.
- EF < 40% defines systolic dysfunction
- SD is found in 2/3 of patients with HF, and they have low cardiac output.

**Diastolic Dysfunction: HFpEF**

- ↓ diastolic relaxation or ↑ stiffness of ventricular wall.
- Ventricular muscle thickens (concentric hypertrophy).
- Cavity size normal, or may become smaller
- Ejection isn’t impaired – ventricular relaxation and filling is.
- Associated with chronic HTN and LVH
- Symptoms often seen with exertion when HR is ↑
- 3 criteria for dx:
  1. Signs/symptoms of HF
  2. Normal or only slightly ↓ EF
  3. Increased diastolic filling pressure and abnormal relaxation of the LV

**Neurohormonal Responses in HF**

- Series of natural compensatory mechanisms that occur to help the body adjust to ↓ CO and to help preserve BP needed to perfuse vital organs
- Initially they help. Over time, they lead to clinical deterioration.
- SNS stimulation, activation of the RAAS
- ↑ levels of endothelin, vasopressin, and cytokines

**Neurohormonal Responses**

- Sympathetic Nervous System Stimulation
- Renin-Angiotensin-Aldosterone System
- Vasopressin and Endothelin
- Inflammatory Response
- Positive Neurohormonal Responses
  - Atrial and brain natriuretic peptides (ANP, BNP)
- Left ventricular remodeling
Patient Evaluation

- Assess patient stability
- Patient History
  - Risk factors and possible etiologies for HF
  - Functional status
  - Volume status
    - How can you assess patient volume status?

Management

### AHA/ACC Stages of Heart Failure (Crawford, 2009)

- **Stage A**: High risk for developing HF
  - No identifiable structural or functional abnormalities
  - No signs or symptoms of HF

- **Stage B**: Presence of structural heart disease strongly associated with development of HF
  - No signs or symptoms of HF
  - Advanced structural heart disease
  - Specialized interventions required
  - Advanced symptoms of HF at rest, chronic renal insufficiency

- **Stage C**: Part or present symptoms of HF associated with underlying structural heart disease

- **Stage D**: Advanced structural heart disease
  - No identified structural or functional abnormalities
  - No signs or symptoms of HF
  - Specialized interventions required

### Treatment Goals

- **GOALS**: Treat HTN, quit smoking, treat dyslipidemia, regular exercise, discourage ETOH, discourage illegal drug use, control metabolic syndrome

- **THERAPY**: ACEi, ARB, or BB in appropriate patients

- **GOALS**: Measures under Stages A and B; Dietary salt restrictions
  - **THERAPY**: Diuretics (fluid retention); ACEi, BB
  - In selected patients: aldosterone antagonist, ARB, digitalis, nitrates/hydralazine

- **GOALS**: Measures under Stages A, B, and C
  - **THERAPY**: In selected patients: aldosterone antagonist, ARB, digitalis, nitrates/hydralazine

- **GOALS**: Measures under Stages A, B, and C
  - **THERAPY**: Compassionate, end-of-life care, hospice

- **GOALS**: Measures under Stages A, B, and C
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### Case 1

- JW is a 48 year old woman with a hx of HTN, HLD and obesity who presents to clinic for yearly evaluation
  - PMH: HTN, obesity; FX: Mother – MI, CHF; Father – DM, HTN
  - SH: tobacco use

  - What are our goals? What drug therapy might be indicated?
Stage A

- High risk for heart disease
  - Primary prevention focus
    - Heart healthy lifestyle, prevention of coronary disease and LV structural abnormalities
  - Drugs: ACEi or ARB as appropriate (vascular disease or DM); statins as appropriate
  - Goals for tx of HTN and dyslipidemia*
  - Treatment of other disorders that inc. risk for HF
    - Obesity, DM, atrial fibrillation, cardiotoxics

Case 2

- MS is a 62 year old man with PMH of HTN, HLD, MI, bicuspid aortic valve who presents to clinic for follow-up.
  - PMH: HTN, HLD, MI (2012), mild AS (bicuspid AV) EF normal
  - What are our goals for therapy?
  - Drug choices?

Case 3

- JS is a 59 year old man with hx of MI, HTN, DM who presents to clinic with c/o palpitations and exertional dyspnea of 1 day duration.
  - PMH: MI (2013), HTN, DM
  - PE: HR irreg rate/rhythm, BBS = w/ scattered crackles in posterior bases, 2+ pretibial edema, Pulses 3+ equal bilaterally
    - VS: HR 122 (AF per 12-lead, no ST changes), BP 144/97, RR 18, T 98.8F
  - Goals? Strategy for tx? Treatment choices?
Stage C

• Includes recommendations for stages A/B
• Includes known SHD and HF s/sx
• Further divided into
  – HF with preserved EF
  – HF with reduced EF

HFpEF

• Goals
  – Control symptoms, improve QOL, prevent hospitalization and mortality
• Identify comorbidities
• Treatment
  – Diuresis to relieve congestion symptoms
  – Follow guidelines for management of identified comorbidities

HFrEF

• Goals
  – Control symptoms, prevent hospitalization and mortality, and patient education
• Drugs for routine use
  – Diuretics, ACEi/ARB, BB, aldosterone antagonists
• Drugs for selected use
  – Hydralazine/nitrates; ACEi/ARB; digitalis
• Other considerations
  – CRT, ICD, revascularization/valve replacement

Diuretics

• Class I rec’s for evidence (or hx of) fluid retention to improve symptoms
  – Balance to dose appropriately to achieve target effect without dehydration, AKI, etc.
  – Loop diuretics most common
    • Bumetanide/torsemide – increased oral bioavailability
    • Na+ restrictive diet
    • Electrolyte monitoring/replacement
    • Drug tweakage (adding thiazide, reducing doses, etc.)

ACE Inhibitors

• HFrEF and current/prior symptoms
• Watch for SBP < 80, creat > 3, bilat RAS, or K+ > 5.0
• Dose low and increase as tolerated
• Watch renal function and K+ levels
  – Angiotensin suppression/kinin production → cough experienced by 20% of patients
  – Rash and taste disturbances are also reported
Angiotensin Receptor Blockers

- Class I rec’s for use if intolerant to ACEi
  - Effective hemodynamic/neurohormonal/clinical effects. Reasonable alternative to ACEi.
- Small risk of angioedema in patients who react to ACEi
- Class III rec’s – warn of potential harm if combined use of ACEi, ARB and aldosterone antagonists in HFrEF

Beta Blockers

- Should be initiated as soon as HFrEF is diagnosed in all stable patients without contraindications
  - Metoprolol succinate; carvedilol; bisoprolol
  - Abrupt cessation should be avoided
  - Adverse rx: fluid retention/HF; fatigue; bradycardia; heart block; hypotension
  - Worsening HF can usually be managed by titrating other drugs so BB therapy can be continued

Aldosterone Antagonists

- HFrEF with NYHA Class II to IV with EF ≤ 35%
- NYHA II with prior CV hospitalization
- Following acute MI in patients with EF < 40%
  - Symptoms of HF or history of DM
- Watch renal fx and electrolytes
  - Creatinine < 2.5 (men); 2.0 (women)
  - Potassium < 5.0
  - Serial monitoring of these required, especially if ACEi/ARB is used

Hydralazine/Nitrates

- Addition of combination for African American patients with NYHA III-IV HFrEF on GDMT with ACEi/ARB
  - Research shows additional morbidity/mortality benefit
- IIa recommendations in HFrEF pts who cannot tolerate ACEi/ARB
  - Additional morbidity/mortality benefit

Digoxin

- IIa recommendations include use in HFrEF to decrease hospitalizations for HF.
  - Persistent symptoms of HF during GDMT
  - Added to initial therapy in patients with severe sx who have not yet responded to GDMT
    - Latest research suggests increased mortality when used in patients with newly diagnosed systolic HF
    - Loading doses not typically required. Low dosing recommended in > 70 yo, impaired renal function or low lean body mass

Anticoagulants

- Long-term anticoagulation in patients with chronic HF with permanent/persistent/paroxysmal AF and an additional risk factor of cardioembolic stroke
  - Age > 75, Hx HTN, DM, previous stroke, or TIA
    - CHA2DS2-VASc score
  - Reasonable tx without additional risk factors
  - Not beneficial in absence of a-fib
Strategies for Achieving Optimal GDMT

- Up-titrate in small increments, see patients/monitor lab results more frequently, monitor vitals closely before/during titration, alternate adjustments of different medication classes (ARB/ACEi; BB); monitor renal fx/electrolytes
- Reassure patients of transient med-related sx
- Discourage sudden med cessation; review doses of all medications when adjusting drug doses; consider temporary adjustments during noncardiac issues
- Patient/family education about GDMT

Device Therapy in HFpEF

- HFpEF – high risk for SCD due to ventricular arrhythmias. Current guidelines coordinate previously conflicting recommendations
- Primary prevention: nonischemic dilated CM or IHD at least 40 days after MI with EF of 35%; NYHA II or III receiving GDMT, expected to live > 1 year
- Special CRT recommendations*
Stage D

- Advanced HF with refractory symptoms
  - Repeat hospitalizations, progressive deterioration in renal fx, intolerance to GDMT, frequent ICD shocks, serum Na+ level < 133, worsening functional status (inability to perform ADLs), escalation of diuretics to high dose or need for addition of thiazide, signs of cardiac cachexia
  - Explore etiologies of worsening symptoms
  - Evaluate patient adherence

- Specialized treatment strategies
  - MCSD, procedures to remove fluid (aquapheresis [Iib], SCUF/CRRT), continuous IV inotropes, transplantation
  - Palliative care/hospice
    - Consider including palliative care early in any treatment plan at this point is important – discuss goals of care with patient and family

Hospitalized Patients with HF

- Specific subgroups based upon precipitant event
  - Accelerated HTN, acute cardiac ischemia, ADHF, shock, acute right-sided HF, decompensation after surgical procedures
  - Recommendations focus on investigation into the contributing causes of the decompensation that led to admission

- Classify patient with congestion or perfusion issue (think Killip table)
  - Warm-Wet: diuretics/vasodilators
  - Cool-Dry: Inotropic support
  - Cool-Wet: combination of inotropes/vasodilators/diuretics

- Use of BNP recommended in evaluation of acute HF and to r/o other dx as causes of symptoms

Transitions and Coordination of Care

- Big emphasis in new guidelines due to potential for fragmentation of care during a very fragile time
- Multidisciplinary care team approach essential
  - Evidence-based treatment plan with phone follow-up 3 days s/p discharge; visit within 1 week
  - Continued assessment of volume and end-organ lab indices
  - Palliative care; home health; rehabilitation
Other Considerations

• Guidelines also include nonpharmacological treatment considerations
  – Social support, sodium restriction, treatment of OSA, weight loss for obesity, and activity/rehabilitation
  – Surgical, transcatheter and percutaneous therapies are also discussed

Final Points

• Increasing number of patients living with heart disease
• Increased complexity of patient presentations
• Guidelines present HF management across a continuum and levels of care
• Adherence to GDMT essential to reduction of mortality and increase in quality of life

References


Thank you!