Medicine Team Notes

Heart Failure

429 Medicine Team

ABSTRACT

The doctor did not provide a power point presentation; these notes elaborate on the points he discussed during the lecture. Source: Kumar & Clark's Clinical Medicine, 7th e, and treatment: 427 Team Notes

DEFINITION:

- A complex syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the heart to function as a pump to support a physiological circulation
- Abnormality of cardiac function results in failure to provide adequate blood flow to meet the metabolic needs of the body's tissues and organs or in an excessive rise in cardiac filling pressures

TYPES:

1. *Left ventricular systolic dysfunction (LVSD)* **(Biventricular "congestive" Heat Failure is the most common)** caused by:
    a. Ischemic heart disease (most common)
    b. Valvular heart disease
    c. Hypertension
2. *Right ventricular systolic dysfunction (RVSD)* secondary to:
    a. Chronic LVSD
    b. Primary and secondary pulmonary hypertension
    c. Right ventricular infarction
    d. Arrhythmogenic right ventricular cardiomyopathy
    e. Adult congenital heart disease
3. *Diastolic heart failure:* is a syndrome consisting of symptoms and signs of heart failure with *preserved left ventricular ejection fraction (> 45-50%) and abnormal left ventricular relaxation*

↑ stiffness in ventricular wall & ↓ left ventricular compliance → impairment of diastolic ventricular filling → ↓ cardiac output

    a. Assessed by echocardiography
    b. More common in **elderly hypertensive patients** but may occur with primary cardiomyopathies (hypertrophic, restrictive, infiltrative)

CAUSES:

Classification either anatomical order, or by prevalence (most common)

PREVALENCE:

- Ischemic heart disease (35-40%)
- Dilated cardiomyopathy (30-34%)
- Hypertension (15-20%)
- Less common:
    - Infiltrative: amyloidosis
    - RV failure due to pulmonary disease, Valvular HD, congenital, arrhythmias, pericardial
    - Infections: Chagas disease

ANATOMICALLY:

1. Pericardium
    a. Pericarditis → dilates the heart
    b. Pericardial tamponade
    c. Constrictive pericarditis (due to TB = scarring leads to thickening & rigidity of pericardium; RARE nowadays)

> Usually w/myocarditis = acute myocarditis
>
> In the young (~20 yrs)
>
> Infection with Coxsackie B virus, group A streptococcus w/rheumatic fever etc
>
> Could lead to cardiogenic shock

2. Myocardium: cardiomyopathy (hypertrophic, dilated etc), congenital defect
3. Endocardium: Infective endocarditis
4. Valves: not very common
    a. Usually due to ischemic heart disease i.e. MI → muscle immobility → Chordae tendenii movement affected → valvular dysfunction → severe mitral regurgitation
    b. Aortic stenosis could develop with aging (calcification of valve), but would not cause HF alone
5. The conduction (electrical) system: arrhythmias
6. Vessels: coronary artery disease
7. Non-cardiac causes:
    a. Severe lung disease e.g. interstitial lung disease, leading to cor pulmonale
    b. Hyper-metabolic conditions (↑ demand) e.g. thyrotoxicosis
    c. Severe anemia (↑ demand)
    d. Pregnancy (↑ demand)
    e. Alcohol & drugs e.g. chemotherapy

CLINICAL PRESENTATION:

SYMPTOMS

- Exertional dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Fatigue

SIGNS

- Cardiomegaly
- Third and fourth heart sounds/murmurs
- Elevated JVP
- Tachycardia
- Hypotension = cardiogenic shock (*But initially there is compensation & vasoconstriction so ↑BP)*
- Bi-basal crackles
- Pleural effusion
- Ankle edema (pitting) ± Ascites
- Tender hepatomegaly

NYHA Class (Imp.)

- **I** No limitation of activities; They suffer no symptoms from ordinary activities
- **II** Slight, mild limitation of activity; They are comfortable with rest or with mild exertion
- **III** Marked limitation of activity; They are comfortable only at rest
- **IV** Confined to bed or chair; Any physical activity brings on discomfort and symptoms occur at rest

◆ **Framingham Criteria for Diagnosis of Heart Failure**

- **Major Criteria:**
  1. PND
  2. JVD
  3. Rales
  4. Cardiomegaly
  5. Acute Pulmonary Edema
  6. S_3 Gallop
  7. Positive hepatic Jugular reflex
  8. ↑ venous pressure > 16 cm H_2O

- **Minor Criteria**
  1. Lower limb edema,
  2. Night cough
  3. Dyspnea on exertion
  4. Hepatomegaly
  5. Pleural effusion
  6. Tachycardia 120 bpm
  7. Weight loss 4.5 kg over 5 days management

RECURRENCE:

Acute decompensation due to physiological stress (↑ demand → ischemia). Examples:

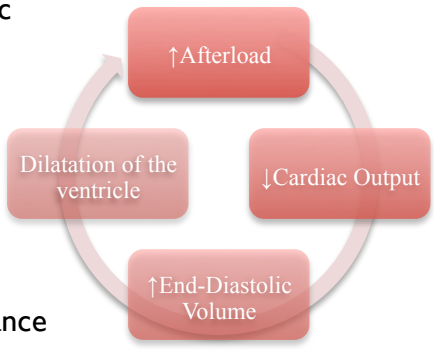
- Non-compliance with medications prescribed to treat HF (most common)
- Infections: pneumonia etc
- Uncontrolled hypertension
- Sepsis, septic shock

PATHOPHYSIOLOGY:

Activation of the renin-angiotensin-aldosterone system = Decompensation

↓ CO → ↓ renal perfusion → activates rennin-angiotensin system → salt & water retention → ↑↑↑ venous pressure

- Factors involved are venous return, outflow resistance, contractility of the myocardium, and salt and water retention
  - Preload (increased volume): Increased diastolic volume stretches the myocardial fibers → depression of ventricular function → ejection fraction is reduced → Tachycardia & symptoms & signs appear: dyspnea, edema, ascites etc (due to a compensatory increase in systemic venous pressure) → advanced disease: depressed CO
  - Afterload (increased pressure): load or resistance against which the ventricle contracts



TREATMENT:

Main pathology: Renin-angiotensin system (RAS)

To improve survival: target the rennin-angiotensin-aldosterone system

Treatment that does not target RAS only improves symptoms and reduces admission rate

DRUGS

1. **Diuretics:**
    a. Loop diuretics e.g. furosomide, bumetanide.
        - MOA: Inhibit Na reabsorption in the ascending limb of the loop of Henle.
        - They are potent.
        - Side Effects: Marked renal k loss, promote hyperuricemia, pre-renal azotemia, hypokalemia, Skin rash, ototoxicity
        - Used for severe heart failure → loop diuretics: Lasix, furosomide, bumex, or torsemide

    b. Thiazides e.g. bendroflumethiazide.
        - MOA: Inhibit Na reabsorption in the distal renal tubule.
        - Mild diuretics except metolazone (which causes excess dieresis and used in severe & resistant heart failure)
        - S/E: Hypokalemia, hyperglycemia & hyperuricemia.

    c. K-Sparing Diuretics
        - Spironolactone (aldosterone inhibitor), triamterene & amiloride (acts on distal tubules to ↓ K secretion)
        - *Recent evidence suggests that it may improve survival in CHF patients due to the effect on renin-angiotensin-aldosterone system with subsequent effect on myocardial remodeling and fibrosis*
        - MOA: Increase Na secretion on the distal nephron and inhibit K secretion.

2. ACE Inhibitors e.g. captopril, enalapril, lisinopril
    a. *They block the R-A-A system by inhibiting the conversion of angiotensin I to angiotensin II → vasodilation*
    ↓ Na retention, and ↓ Water retention.
    b. *↓ Bradykinin degradation → ↑ its* level *→ ↑ PG secretion & nitric oxide*
    c. Ace Inhibitors were found to improve survival in CHF patients;
        - Delay onset & progression of HF in pts with asymptomatic LV dysfunction
        - ↓ cardiac remodeling
    d. MOA:
        - Inhibit angiotensin II

- Increase cardiac output by decreasing preload and afterload
- Decrease vascular resistance and PCWP (Pulmonary Capillary Wedge Pressure)

e. Common Side Effects

- First dose hypotension particularly in patients receiving diuretics.
- So, the dose of diuretics, 24 hr before first dose of ACEI, should be started with low dose followed by gradual increase every 1-2 weeks
- Hyperkalemia

f. Other S/E: angioedema, persistent cough (↑ *bradykinin)*, renal insufficiency , and rash

g. Contraindicated in patients with bilateral renal artery stenosis.

h. Studies Of LVD (SOLVD): Enalapril

- Decrease all cause mortality 16%
- Decrease mortality from HF 22%

i. S/E: Gynaecomastia, nausea and abdominal pain.

3. Angiotensin II Receptor Antagonist e.g. losartan, valsartan

a. MOA: They block binding of angiotensin 2 with type 1 receptors.

b. Do not produce cough, and have comparable effect to ACE I

c. Can be used in certain conditions when ACE I are contraindicated (angioneurotic edema, cough)

d. Contraindicated in patients with bilateral renal artery stenosis.

4. B-Blockers e.g. metoprolol, bisoprolol, carvedilol.

a. Has been traditionally contraindicated in pts with CHF

b. Now they are the main stay in treatment on CHF & may be the only medication that shows substantial improvement in LV function

c. In addition to improved LV function multiple studies show improved survival

d. The only contraindication is severe decompensated CHF

5. **Digoxin:** (No mortality benefit)

a. The role of digitalis has declined somewhat because of safety concern

b. Recent studies have shown that digitalis does not affect mortality in CHF patients but causes significant:
   - Reduction in hospitalization
   - Reduction in symptoms of HF

c. MOA:

- Positive inotropic effect by ↑ intracellular Ca & enhancing actin-myosin cross bride formation (binds to the Na-K ATPase → inhibits Na pump → ↑ intracellular Na → ↑ Na-Ca exchange)
- Vagotonic effect
- Arrhythmogenic effect

d. Digitalis Toxicity

- Narrow therapeutic to toxic ratio
- Non cardiac manifestations (Anorexia, nausea, vomiting, headache, Xanthopsia sotoma, disorientation)
- Cardiac manifestations
  - » Sinus bradycardia and arrest
  - » A/V block (usually 2nd degree)
  - » Atrial tachycardia with A/V Block
  - » Development of junctional rhythm in patients with atrial fibrillation
  - » PVC's (Premature Ventricular Complexes) , Ventricular Tachycardia/ Ventricular fibrillation (bi-directional VT).
- Treatment of the toxicity:
  - » Hold the medications
  - » Observation
  - » In case of A/V block or severe bradycardia → atropine followed by temporary PM if needed
  - » In life threatening arrhythmia → digoxin-specific fab (Fragment-antigen-binding) antibodies
  - » Lidocaine and phenytoin could be used – try to avoid D/C cardioversion in non life threatening arrhythmia

6. ***Vasodilators***

a. **Reduction of afterload** by arteriolar vasodilatation (hydralazin) → reduce LVEDP (Left ventricular end diastolic pressure), O_2 consumption, improve myocardial perfusion, ↑ stroke volume and COP

b. **Reduction of preload** By venous dilation (Nitrate) → ↓ the venous return →↓ the load on both ventricles.

c. Usually the maximum benefit is achieved by using agents with both actions

7. ***Positive inotropic agents***

a. These are the drugs that improve myocardial contractility (β adrenergic agonists, dopaminergic agents, phosphodiesterase inhibitors)

b. Dopamine, dobutamine, milrinone, amrinone

c. Several studies showed ↑ mortality with oral inotropic agents

d. So the only use for them now is in acute sittings as cardiogenic shock

8. ***Anticoagulation (coumadine)***

    a. Atrial fibrillation

    b. History of embolic episodes

    c. Left ventricular apical thrombus

9. ***Antiarrhythmics***

    a. Most common cause of Sudden Cardiac Death in these patients is ventricular tachyarrhythmia
    b. Patients with history of sustained ventricular tachycardia or SCD → ICD (Implantable Cardioverter Defibrillator) implant
    c. Patients with non sustained ventricular tachycardia
        - Correction of electrolytes and acid base imbalance
        - In patients with ischemic cardiomyopathy → ICD implant is the option after rule out acute ischemia as the cause
        - In patients with non ischemic cardiomyopathy management is ICD implantation

- ***New Methods***
    - **Implantable ventricular assist devices**
    - **Biventricular pacing** (only in patient with LBBB & CHF)
    - **Artificial Heart**

SURGERY: CARDIAC TRANSPLANTATION.

- It has become more widely used since the advances in immunosuppressive treatment
- Has 90% 1-year survival after surgery
- 75% alive after 5 years
- Death usually due to: Operative mortality, organ rejection

PROGNOSIS

- Annual mortality rate depends on patients symptoms and LV function
- 5% in patients with mild symptoms and mild ↓ in LV function
- 30% to 50% in patient with advances LV dysfunction and severe symptoms
- 40% – 50% of death is due to SCD