Congestive Heart Failure

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Outline

Congestive Heart Failure

- **Epidemiology**
- **Concepts in Pathology/Prevention**
- **Drug therapy**

Epidemiology

- 5 million patients nationwide
- 550,000 newly dx'd each year
- 12 to 15 million office visits/year
- 6.5 million hospital days/year
- 10 patients per 1000 population
- Condition of the elderly: 80 percent of those hospitalized with HF are over 65 years old
- Most common DRG
- Over 28 billion in cost USA

Pathophysiology of Heart failure

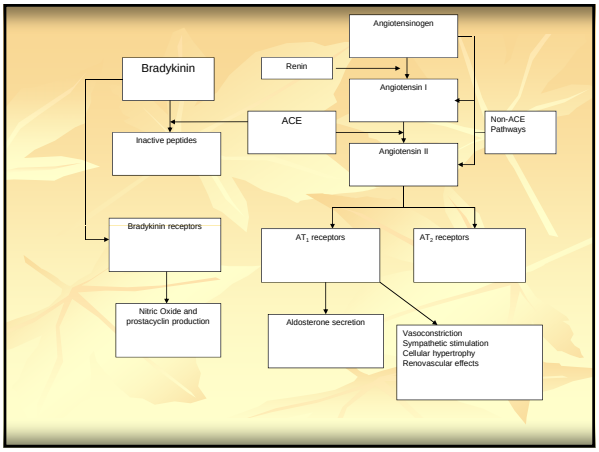
- **Etiology**
  - **Volume overload (Valve Regurgitation)**
  - **Pressure overload (HTN)**
  - **Loss of myocytes (AMI)**
  - **Infections (viral, rickettsia, bacterial, fungal, etc)**

Pathophysiology of Heart failure

- **A variety of changes take place that, in the short term, help maintain cardiac output in the face of reduced pump function**
  - **Chamber Dilation**
  - **Cardiac Hypertrophy**
  - **Sympathetic Nervous System (SNS) Discharge**
  - **Renin-Angiotensin-Aldosterone System (RAAS) Activation**

Pathophysiology of Heart Failure

- **SNS activation occurs due to reduced blood pressure**
  - **Contractility is increased initially**
  - **Sympathetic overstimulation results in cardiac remodeling and makes the heart prone to developing arrhythmias**



Pathophysiology of Heart Failure

- **Activation of the RAAS occurs in heart failure because of:**
  - **Reduced cardiac output**
  - **SNS activation**
  - **Reduced renal perfusion**

Pathophysiology of Heart Failure

- **Initially the system helps maintain cardiac output by**
  - **Promoting Na^+ and water retention**
  - **Increasing thirst**
  - **Activating the Sympathetic Nervous System**
  - **Stimulating vasopressin release**
  - **Constricting blood vessels**

Pathophysiology of Heart Failure

- **Renin Angiotensin Aaldosteron System eventually promotes myocardial dysfunction by:**
  - **Increasing preload and afterload**
  - **Ang-II and aldosterone promote cardiac remodeling**

Pathophysiology of Heart Failure

- **Natriuretic Peptide System**
  - **Consists of three types of peptides**
    - **ANP – secreted from atria in response to increased wall tension**
    - **BNP – secreted by the ventricle in response to increased wall tension**
    - **CNP – secreted by blood vessels and acts locally to promote vasodilation**
  - **ANP and BNP are physiologic antagonists to Ang II.**

Drugs Tx That Does Not Decrease Mortality Long Term in CHF

Most Diuretics
- Lasix (furosemide)
- Demadex (torsemide)
- Bumex (bumetanide)
- Dyazide, Maxzide (HCTZ)
- Chlorthalidone

Digoxin (does decrease readmit)
- Lanoxin
- Digitek

Inotropes
- Dobutrex (dobutamine)
- Primacor (milrinone)
- Inocor (inamrinone)
- Dopamine

Calcium channel blockers
- Norvasc (amlodipine)
- Cardizem, Cartia (diltiazem)
- Calan, Isoptin, Covera (verapamil)

Alpha blockers
- Minipress (prazosin)
- Hytrin (terazosin)
- Cardura (doxazosin)

Treatment That Does Decrease Mortality Long Term in CHF

Beta blockers
- Toprol XL (metoprolol)
- Coreg (carvedilol)
- Zebeta (bisoprolol)

ACEIs
- Vasotec (enalapril)
- Capoten (captopril)
- Zestril (lisinopril)
- Prinivil (lisinopril)
- Accupril (quinapril)

Aldosterone Antagonists
- Aldactone (spironolactone)
- Inspra (eplenerone)

Hydralazine / Nitro Combo
- Apresoline (hydralazine)
- Imdur (isosorbide)
- Ismo (isosorbide)

Beta Blocker Mechanisms

- Not clearly understood
- Protects against cardiotoxic effects of catecholamines (norepinephrine)
- Up regulation of Beta-1 receptors to improve myocardial response
- Decreases HR, Increases coronary blood flow, improves myocardial perfusion
- Corrects abnormal calcium deposits
- Antioxidant
- Protects against circulating autoantibodies
- Reverses/Prevents remodeling and programmed cellular death
- Increases C.I. / ejection fraction
- Decreases Pulmonary Capillary Wedge Pressure

Beta Blocker Benefits

- **Effective in Mild to Severe CHF**
- **Improves:**
  - **New York Heart Association Class of CHF**
  - **Cardiac Output/Index**
  - **Left ventricular ejection fraction**
  - **HR, exercise tolerance, quality of life**
- **Decreases:**
  - **Mortality 20-65%**
  - **Hospitalization 23-32%**
  - **Progression of CHF**
  - **Need for Heart Transplant**

Beta Blocker Key Issues

- **Start Low & Titrate Upward Over Weeks**
- **May feel tired for up to 6 weeks while titrating to final dose**
- **Takes 3 full months of therapy to begin seeing the positive benefits**
- **Using a combination beta/alpha blocker may decrease the negative effects early in therapy by decreasing afterload (Coreg)**
- **Inform patients that this is a long-term treatment strategy to truly increase their life span**

ACE Inhibitors

- **ACEIs and ARBs**
  - **Captopril, enalapril, ramipril, lisinopril, quinapril and fosinopril are FDA approved for treatment of CHF.**
  - **Mechanism:**
    - **Reduce preload and afterload**
    - **Prevent Ang II and aldosterone mediated cardiac remodeling**
    - **ACEIs block bradykinin breakdown, which causes vasodilation**
  - **Recommended for all stable CHF patients**
  - **Start with a low dose and titrate**
  - **ARBs are not yet FDA approved for CHF treatment**

ACE Inhibitor Benefits

- **Decreases:**
  - **Overall Mortality by 50 %**
  - **Re-Hospitalization Rate**
  - **Myocardial Stress via Decreased Afterload**
  - **Remodeling of the heart**
  - **Ischemic episodes**
  - **Thrombogenic / Fibrinolytic effects**
  - **Net sodium loss when combined with diuretic**
  - **Exercise tolerability**
  - **Survival by 50 %**

Hydralazine + Nitrate

- **Reduces CHF related mortality compared to placebo but to a lesser degree than ACEIs.**
- **Mechanism**
  - ✓ **Reduce preload and afterload, relieving cardiac stress.**
  - ✓ **Increase renal blood flow**
- **Used in patients intolerant to or in combination with ACEIs**
- **Start at a low dose and titrate to avoid SEs such as hypotension and headaches**

Diuretic Benefits

- **Minimize Sodium and Water Reabsorption**
- **Decrease Intravascular Fluid**
- **Lessens symptomatic effects of CHF**
  - **Pulmonary edema**
  - **Peripheral edema**
- **Assists with the action of ACE-Inhibitors**

Aldosterone Antagonists

- **Spironolactone and Eplerenone**
  - **Mechanism**
    - **Block aldosterone mediated cardiac remodeling**
    - **Promote Na+ and H2O excretion**
    - **Should these drugs be used with ACEIs?**
  - **Eplerenone should be used in patients intolerant of the metabolic side effects of spironolactone**
    - **gynecomastia**

Spironolactone Benefits

- **Potassium sparring diuretic (Aldactone)**
- **Mechanism**
  - **Blocks aldosterone receptors at level of the kidney to decreases intravascular fluid load**
  - **Block aldosterone mediated cardiac remodeling**
  - **Promote Na+ and H2O excretion**
  - **Anti-Fibrotic (decreases myocardial fibrosis)**
  - **Toxic free oxygen radical scavengers**
  - **Blocks some of the vasoconstrictive effects of aldosterone**
  - **Should these drugs be used with ACEIs?**
- **Decreases mortality 30 % and decreases hospitalization 35 %**

Digoxin Benefits

- **Decreases overall re-hospitalizations**
- **Improves force of contraction**
- **Decreases Symptoms, Increases Exercise Tolerance, Increases Quality of Life**
- **Low dose for > 70 yrs (0.125 mg daily)**
- **Higher dose for < 70 yrs (0.25 mg dialy)**

Nesiritide

- **Recombinant hBNP**
- **Used for patients with decompensated CHF and dyspnea**
- **Mechanism**
  - **Reduces preload and afterload**
  - **Promotes Na+ and H2O excretion**
  - **Reduces PCWP and relieves dyspnea**
- **Should only be used for 48 consecutive hours.**

DONE

Thanks