HEAT FAILURE

BY PROF JOSHI

DEFINITION

- Abnormality in cardiac structure or function resulting in the inability of the heart to fill with or eject blood at a rate commensurate with the requirements of the metabolizing tissues

- It is a syndrome characterized by dyspnea. Fatigue and fluid retention

- It is a progressive disorder of LV remodeling, usually resulting from an index event that culminates in a clinical syndrome characterized by impaired cardiac function and circulatory congestion

FORMS OF HEART FAILURE

- Systolic: inability of the ventricles to contract normally and expel sufficient blood

- Diastolic: HF in patients with preserved systolic function; EF > 50%
  - Due to increased resistance to ventricular inflow -> reduced ventricular diastolic capacity or reduced ventricular relaxation
  - Mostly abnormal or both contraction and relaxation

HF PATHOPHYSIOLOGY

- Risk factors
- Myocardial injury (MI, HTN, VHD, Cardiomyopathy)
- Initial fall in LV performance – increase wall stress
- Activation of RAAS and SNS
- Fibrosis, apoptosis, hypertrophy, cellular and molecular alterations
  - Hemodynamic alterations, salt and water retention -> HF symptoms: dyspnea, edema, fatigue
  - Re-modelling and progressive worsening of LVF -> Morbidity and mortality

COMPENSATORY MECHANISMS

- Increase preload – Frank-Starling ventricular dilatation and volume expansion
- Increase afterload – PV vasoconstriction
- Hypertrophy – preserve wall stress – Laplace law
- Salt and water retention – preload
- Increased HR and myocardial contractility
- Via neuro-hormonal activation – RAAS. AVP

ETIOLOGY

- Categories Six Major: failure related to:
  - Myocardial abnormalities – myocyte loss etc.
  - External work overload
  - Valvular abnormalities
  - Abnormal cardiac rhythm
  - Pericardial abnormalities
  - Congenital abnormalities

ADHF EPIDEMIOLOGY KENYA

- Period prevalence hospitalized medical
  - KNH 1998 3.3%; 2008 4-6%
  - Muranga rural 2011 DH 5%
- Age: median age KNH 44 year; MDH60 years
- Prognosis mortality
  - In-hospital 11%
  - Post-discharge 25-37% at 6 months
  - Re-admission 38%; average 1.3 per patient
  - M&M 49% (40.6 – 57.8)

SYMPTOMATOLOGY

- Cardiac
  - Exertional dyspnea
  - PND; orthopnea
  - Dyspnea at rest
  - Palpitations
  - Leg swelling
  - Abdominal swelling
  - RUQ pain
- Non-cardiac
  - Fatigue
  - Anorexia
  - Weight-loss cachexia

SIGNS

- Systemic venous congestion
  - Pedal edema
  - Tender hepatomegaly
  - Elevated JVP
- Pulmonary venous congestion
  - Basal rales
- Hypotension and tachycardia
- Precordial findings depend on cause

PRINCIPLES OF MANAGEMENT

- Diagnosis:
  - Presence of HF
  - Underlying cardiac condition
  - Precipitating conditions
  - Comorbidity

CRITERIA 1 & 2 should be fulfilled in all cases

1. Symptoms of HF at rest or on exertion
2. Objective evidence of cardiac dysfunction (at rest)

IN CASES WHERE THE DIAGNOSIS IS IN DOUBT

1. Response to treatment directed towards HF

MODIFIED FRAMINGHAM CLINICAL CRITERIA FOR THE DIAGNOSIS OF HEART FAILURE

- Major
  - PND
  - Orthopnea
  - Elevated JVP
  - Pulmonary rales
  - S3
  - Cardiomegaly on CXR
  - Pulmonary edema on CXR
  - Weight loss
- Minor
  - Bilateral leg edema
  - Nocturnal cough
  - Dyspnea on exertion
  - Hepatomegaly
  - Pleural effusion
  - Tachycardia

ALGORITHM FOR THE DIAGNOSIS OF HF

- Suspected HF because of symptoms and signs
- Assess presence of cardiac diseases by ECG, X-ray or Natriuretic peptides
- If test is abnormal -> echocardiography
- If abnormal – assess etiology, HF, precipitating factors and type degree of cardiac dysfunction
  - Additional diagnostic tests where appropriate e.g. coronary angiography
  - Choose therapy

INVESTIGATION

- ECG – normal or high negative predictive value
- CXR to r/o dyspnea
- 2D echocardiography
  - LV dysfunction (systolic/diastolic)
  - Etiology
- Biochemistry and hematology
- Natriuretic peptides (NT-BNP)
- Others:
  - Stress testing, Holter, catheterization, RNA, MRI

MANAGEMENT AIMS

- Prevention
  - Prevention and/or controlling of diseases leading to cardiac dysfunction and HF
  - Prevention of progression to HF once cardiac dysfunction is established
- Morbidity
  - Maintenance or improvement in quality of life
- Mortality
  - Increased longevity

MANAGEMEN TSTRATEGIES

- Non-pharmacological intervention and lifestyle modification
  - Physical activity
  - Dietary modification
  - Vaccination – influenza, pneumococcal
  - Education and counselling

CONT.

- Symptomatic therapy
  - Diuretic
    - Less signs and symptoms; no mortality benefit; use loop diuretic along with a thiazide (plus a RAAS blocker); monitor renal function
  - Digoxin
    - Useful in rate control in AF, indicated in: Symptomatic patients after maximal doses in medical treatment and frequent hospitalizations; rate control in AF; large RCTs – no mortality benefit; DIG trial

CONT.

- Disease modifying therapy
  - Neuro-hormonal interventions:
    - ACE-inhibitors: first line agents, C/I in renal disease, angioedema, persistent cough
    - Beta-blockers: nebivolol, metoprolol, bisoprolol, carvedilol; provide significant mortality and long-term symptomatic benefit in all grades of HF and post MI-LVSD; start beta blocker in patient free of decompensation.
    - Aldosterone antagonists: spironolactone, eplerenone; reduced morbidity and mortality in patients with moderat – severe HF; combined with standard ACE-I + BB therapy; watch out for hyperkalemia and tender gynecomastia (spironolactone)
    - Hydralazine/Nitrate
    - AT-II receptor antagonists

CONT.

- Other pharmacological agents
- Device therapy
- Surgical options

DRUGS TO AVOID IN HF

- NSAIDs – cause fluid retention
- Rate limiting CCBs – Diltiazem and Verapamil
- Class I anti-arrhythmics
- Steroids
- TCAs

- **TYPED BY DR. E. NAILA**