CONGESTIVE CARDIAC FAILURE

Def: Congestive cardiac failure is a clinical syndrome resulting from a structural or fxnal cardiac disorder with impaired ability of the ventricles to fill or eject blood at a rate commensurate to the body's metabolic demands.

Epidemiology

Heart failure is frequently due to coronary artery disease. The prevalence rises from about 1% at 50-59yrs to between 5-10% at 80-89yrs.
In the UK, most of the patients with heart failure are > 65yrs. Congestive cardiac failure carries a poor prognosis with about 50% of patients with LV dysfxn dying within 2 yrs of diagnosis.
Many of the patients with CCF die from:-

- Sudden cardiac dysarrhythmias
- Myocardial dysfxn

Aetiology of CCF

- Pump Failure
  - Muscle dysfxn; IHD, Cardiomyopathy, poisons.
  - Restricted filling; mitral stenosis, pericarditis, cardiac tamponade.
  - Heart rate abnormality
- Excess preload
  - Regurgitant valves
  - High output states; anaemia, thyrotoxicosis, thiamine deficiency.
- Chronic Excess after Load
  - Long standing HTN
- Others
  - infections
  - electrolyte imbalance

Precipitants of heart failure

1. Anaemia
2. Infections including pneumonia and infective endocarditis
3. Uncontrolled HTN
4. Uncontrolled hyperglyceamia
5. Thyrotoxicosis –patients with heart failure must have the thyroid gland examined
6. Excessive exercise
7. Drugs – NSAIDS, steroids, Ca CBs
8. Pulmonary embolism
9. Patients already on medication (for CCF) and are non- compliant
10. Substance abuse
11. A new MI
12. Pregnancy
13. Arrhythmias
14. Electrolyte imbalance of whatever cause

Pathophysiology

The pathophysiologic changes are usually due to overwhelmed compensatory mechanisms for the heart to meet the body's metabolic demands. The pathology results from:-

i) Haemodynamic changes

- Decreased output due to systolic dysfxn and decreased filling due to diastolic dysfxn
- The ejection fraction; a measure of EDV is decreased in systolic dysfxn as occurs in decreased ventricular contractility like MI, myocarditis, dilated cardiomyopathy.
- Systolic dysfxn also occurs in ventricular outflow obstruction (pressure overload) as in HTN,AS, PS, or it can occur in ventricular inflow obstruction as in MS, TS or in ventricular volume overload like in MR, AR, VSD & ASD.
- Diastolic dysfxn results from impaired relaxation. In normal physiology, the heart's filling is passive, mostly from the normal atria. A second filling; atrial kick also brings residual blood into the heart.
- The early/atrial kick ratio is normally greater than one (1) or 2 but in diastolic dysfxn, the E.A ratio (measured using echo) is <1. In diastolic dysfxn, the ejection fraction is normal and CO may be initially normal but with time, LV end diastolic pressure is shifted to the left leading to CCF.
- Diastolic dysfxn can occur in:-
    - Constrictive pericarditis
    - Restrictive cardiomyopathy
    - LVH
    - Cardiac tamponade

ii) Neurohormonal changes

A fall in cardiac output usually activates counter regulatory neurohormonal mechanisms that in normal physiological circumstances would support cardiac fxn.

Stimulation of the RAAS leads to vasoconstriction, salt and water retention, and sympathetic activation mediated by angiotensin II, which is a potent constrictor of arterioles both in the kidney and systemic circulation. This may initially maintain cardiac output through increased myocardial contractility, HR and peripheral vasoconstriction. But prolonged sympathetic stimulation leads to cardiac myocyte apoptosis, hypertrophy and focal myocardial necrosis.

Salt and water retention is promoted by the release of aldosterone, endothelin, severe heart failure and ADH. Natriuretic peptides act as physiological antagonists to the fluid conserving effect of aldosterone.

iii) Cellular changes and remodelling

After myocardial infarction, cardiac contractility is impaired and neurohormonal activation may lead to hypertrophy of non –infarcted segments, with thinning, dilatation and expansion of the infarcted segment (remodelling). This may lead to further deterioration in ventricular fxn and worsening heart failure.

Remodelling leads to changes in calcium handling, adrenergic receptors, contractile apparatus and myocyte structure.

NB: Mediators of cardiac remodelling

i) Norepinephrine and epinephrine (toxic to the heart muscles)
ii) Aldosterone
iii) ADH

iv) Cytokines – These are secreted by macrophages, lymphocytes, monocytes which release IL and TNF that are important in the cycle of myocyte hypertrophy and cell death.
IL – 1 may accelerate myocyte hypertrophy, endothelin is a potent vasconstrictor and excessive endothelin release may be responsible for pulmonary arterial HTN and LVF. It's also associated with myocyte growth and deposition of matrix.

Clinical syndromes of heart failure

1. Left ventricular failure
2. Right ventricular failure
3. Systolic heart failure – inadequate cardiac or forward failure.
4. Diastolic heart failure (backward failure)
5. High output failure – associated with high cardiac output (A-V shunt, beriberi, severe anaemia or thyrotoxicosis) and elevation in pulmonary venous pressure.
6. Biventricular heart failure (Lt + Rt ventricular failure)
7. Acute and chronic heart failure

Presentation

S + S are not sensitive and specific and include:

- Dyspnoea, fatigue due to fluid retention.
- Pulmonary or peripheral oedema due to fluid retention. Patient could even have anarsaca.
- Orthopnoea, paroxysmal nocturnal dyspnoea or nocturnal cough with sputum production.
- Wheeze (cardiac asthma)
- Weight loss and muscle wasting (cardiac cachexia)
- In RVF – patient has peripheral oedema or anarsaca, abdominal distension due to ascites, breathlessness, nausea, anorexia, facial engorgement, pulsation in the neck.

Framingham clinical criteria for the diagnosis of heart failure:

Major criteria

1. Paroxysmal nocturnal dyspnoea (PND) –Depression of resp. centre makes patient wake up at night once PaO_2 levels fall. PND is also called cardiac asthma.
2. Orthopnoea – redistribution of retained fluid leads to increase in venous return to the heart.
   NB: The above 2 features occur with fluid retention.
3. Elevated JVP
4. Rales or crackles
5. Cardiomegally on CXR
6. S_3 gallop
7. Plain pulmonary oedema on CXR
8. Weight loss of > 4.5kg in 5 days in response to treatment for heart failure.

Minor criteria

1. Bilateral leg oedema
2. Nocturnal cough

3. Dyspnoea on ordinary exertion
4. Hepatomegally
5. Pleural effusion
6. Tachycardia >120/min
7. Weight loss > 4.5kg in 5 days in response to treatment.

Diagnosis of heart failure

2Major criteria or

1Major and 2 minor

Diagnosis of heart failure is usually made from:-

1. History

i) Precipitants must be looked for in the history

ii) Look for the underlying cause

- Identify the cause and if reversible correct it and the patient will be fine.
- Congenital valvular lesions
  - Symptoms of heart failure from childhood.
  - Recurrent admissions from childhood
  - Failure to thrive
  - Slow in educational progression
- Rheumatic fever – may be history of tonsillitis
- History of DM and other risk factors for coronary heart disease; smoking, obesity, alcohol.
- Recent delivery – think of cardiomyopathy
- Drugs – ARVs and their SE
- Chemotherapy –doxorubicin
- Hiv infxn

iii) Enquire on activity and symptoms for **New York heart association classification of heart failure**

2. Physical examination

Patient may be ill –looking depending on severity, dyspnoic, tachypnoic, cyanosed, pale, jaundice, leg oedema.

Vital signs –Temp, PR, RR, BP

Hands – pallor, cyanosis, splitter h'ges, finger clubbing, Osler's nodes, nicotine staining.

Pulse – Rate, rhythm, volume, character compare both Rt and Lt radial pulse and take all other pulses (all of them).

BP – Low systolic Bp or high systolic Bp

- Wide or narrow pulse pressure

Neck – distended neck veins JVP

Examine for thyroid gland enlargement

Chest – Abnormal chest (shape), scars, distended veins

- Hyperactive precordium
- May be displaced apex beat; heaving apex, tapping apex (ms)

- Thrill, Lt parasternal heave
- Palpable P_2

Auscultation – normal S_1, S_2

- S_3 gallop rhythm in systolic failure
- S_4 gallop rhythm in diastolic failure
- + murmurs

R/S – crackles + pleural rub

P/A – Abdo. distension

- Hepatic pulsation – tricuspid regurgitation on left heart failure
- Hepatomegally
- Ascites, thrill, shifting dullness
- Hepatojugular reflux

3. Investigation

i) FBC – Hb, WBC

ii) W/E – Electrolyte imbalance

iii) Serum creatinine

iv) LFTs – Acutely congestive – hepatic failure

- Chronically congestive – cardiac cirrhosis

v) CXR – for Framingham classification to rule out underlying pathology; cardiomyopathy, ventricular hypertrophy, pulmonary oedema, pericardial effusion or pleural effusion.

- Kerley ABC lines –linear opacities seen on CXR in patients with pulmonary oedema.
- Kerley A lines – longer, at least 2cm, unbranching, crossing diagonally from periphery to hilar in the inner ½ of lungs. Are due to distension of anastomotic channels between periphery & central channels.
- Kerley B lines – about 1-2cm long, generally horizontal and meet the pleura at right angles to fat aspect of lungs.
- Typically seen as a ladder from below at costal angle.
- **DDX** – Pulmonary oedema
  - -CCF especially, LVF & MS
  - -Lymphangitis carcinomatosis
  - -Pulmonary fibrosis
  - -Parasitic infestation
  - -Heavy metal particles
  - -Haemosiderosis
- Kerley C lines –Non – specific reticular patterns. Are the least seen

4. ECG – To detect arrhythmias
5. Echo – is the best way to measure E.A ratio and for proper management.
6. MRI – Gold standard for assessing ventricular volume
7. BNP – Cardiac myocyte necrosis
   - -Is an expensive test
   - -A normal plasma level excludes heart failure

Stages of heart failure (structural)
Stage A – A patient who has risk factors for heart failure e.g DM, smoking etc, but has no structural heart disease or symptoms
Stage B- A patient with a structural heart dz e.g cardiomyopathy, valvular heart disease but is asymptomatic.
Stage C – patients with prior or current symptoms of heart failure.
Stage D – refractory or end stage heart failure.

Classification of severity for heart failure
The New York heart association classification
This usually indicates the class or degree of heart failure, associated symptoms and activity limitation.

Class / degree	**Symptoms and activity limitation**
Class I – None	No symptoms from ordinary activity
Class II – Mild HF	Comfortable at rest or during mild exertion. Dyspnoea on ordinary activity
Class III – Mod. HF	Symptomatic with any activity
Class IV – Severe HF	Symptoms at rest – confined to bed or chair

Management of heart failure
Aims
- To reduce symptoms
- To reduce morbidity
- To improve the quality of life

A. Quick assessment of patient
- Resuscitate patient with decompensated HF (patient with rapidly developing cardiogenic pulmonary oedema).
- Prop up patient
- Give oxygen while checking O_2 saturation
- I.V frusemide (slowly) is more effective than oral; larger doses are required in renal failure.
- Opiates –Sedates patient, decrease anxiety and helps patient meet metabolic needs.
- Nitrates especially if systolic BP ≥ 100mmHg

B. Non pharmacological management
1. Correct all precipitants

2. Lifestyle modification

- Comply with medications
- Salt restriction (2g/day)
- Alcohol ceasation
- Stop smoking
- Exercise training for ambulatory patients
- Limit fluid intake (input /output chart) to avoid hyponatraemia.

3. Look for underlying cause and treat or modify.

- HTN – optimize anti – HTN regime
- DM – control sugars
- Manage any coronary syndromes
- Arrhythmias – anticoagulate

Pharmacotherapy

- Manage symptoms and improve quality of life.

I) Asymptomatic LV systolic dysfxn

- ACE –I
- Health education
- Risk factors
- Rx precipitants

II) Symptomatic LV systolic failure

1. Start with loop diuretics

- Frusemide 40mg – 80mg preferably as OD dose, all at once rather than divided doses.
- Frusemide gives symptomatic relief of fluid retention.
- Monitor U/E, weight loss 1kg/day. Frusemide is a rapid response drug, if patient not losing weight, change to thiazide diuretic preferably I.V.

2. ACE – I – Consider in all patients with LV systolic dysfxn
   ACE –I commonly used

- Captoprill
- Lisinopril
- Enalapril

ACE –I – Improves heart failure symptoms

- Prolongs life
- Reduces mortality

Principles

- Response is immediate
- Once patient is losing fluid, no worsening of symptoms.
- Start with a low dose slowly
  Enalapril 10-20mg BD
  Captopril 6.25mg TID max 50mg
  Lisinopril 5mg OD – max 40 mg OD
  Over 1-2 wks – you can double the dose to the max dose depending on response.
  Monitor –cough – usually due to increased bradykinin levels
  If patient develops uncontrollable cough, change to ARB
  U/E – Hyperkalemia, azotemia. Candesartan, valsartan (ARBs) tested and found to be good but do not give in patients with MI.

ARBs usually reserved for patients intolerant of ACE-I.

3. B – Blockers – only given when patient is generally stable and the BP is good.

CI – Asthma
- COPD
- Hypotension
- 2nd or 3rd degree heart block, bradycardia & sinus syndrome.

BB commonly used:
- Carvedilol – beta and alpha blocker, anti –oxidant
- Metoprolol (selective B_1)
- Bisoprolol
- Nevibolol

BB – Improve LV ejection fraction & survival in patients with symptomatic LV dysfxn.
Always start at a low dose and titrate to target max doses e.g.
Carvedilol 3.125mg BD – 25 – 50mg BD
Bisoprolol 1.25mg OD – decreased 5-10mg OD
Metoprolol 12.5mg OD – max 25mg
NB: 1st one month, patient may get worsening of symptoms because of sympathetic effect which is blocked.
Long term effect – down regulation alters over time.
The effects are usually long term, not short term.

4. ARB – Can be used as substitute to ACE –I or as an adl on therapy in HF.
5. Aldosterone blockers / antagonists
- Spironolactone – indicated if low ejection fraction <40%
- Dose 25 -50mg daily
- It improves endothelial dysfxn (nitric oxide bioavailability) and prevents remodelling
- Decreases mortality by 30%
- Is K^+ sparing
- Eplerenone 25 -50mg OD – New York heart association class III.

6. Digoxin – for purely heart failure, start with low dose digoxin 0.125mg OD.
- It reduces rate of hospitalization
- Offers symptomatic relief benefits
- Good at controlling fibrillation
- No mortality benefits

NB: No significant improvement in survival rates.

7. Vasodilators
- Nitrates together with hydrallazine (combination);
- **Indications** – failure to control symptoms with all above drugs.
- Hydrallazine 25mg tid plus
- Isosorbide nitrate 40mg tid

The combination of hydrallazine & nitrate has more benefits in blacks than whites.
- Provides an alternative in patients with ACE –I intolerance or those who may require additional therapy for BP.
- Reduces mortality in symptomatic patients (mortality benefit)
- Reduces rate of hospitalization

- Improves quality of life

D) Management of refractory heart failure

- Implantable cardioverter defibrillator (ICD.

Indications

- After cardiac arrest
- Ventricular tachycardia that is defibrillating
- Lt ventricular assists device indications include;
- Very slow ejection fraction < 30%
- Severe myocardial infarction
- If nothing is working then consider heart transplant.

Diastolic heart failure

Management – manage underlying cause

- Use drugs that manage fluid retention (diuretics)