HEART FAILURE

Introduction:

- ***Definition***: It is a clinical syndrome resulting from structural or functional cardiac disorders that impair the ability of ventricles to fill or eject blood (to meet the metabolic needs of the body) [Hinkle & Cheever, 2014]
- Fluid buildup in the heart from myocardium that can’t provide sufficient cardiac output
- Usually occurs in a damaged left ventricle but may occur in right ventricle, either primarily or secondary to left-sided heart failure

- Heart failure (HF) was referred to congestive heart/Cardiac failure (CHF or CCF) due to many patients present with pulmonary or peripheral congestion with oedema
- Currently, HF is recognized as clinical syndrome characterized by signs and symptoms of fluid overload or inadequate tissue perfusion
- Fluid overload and decreased tissue perfusion occurs when heart cannot generate sufficient cardiac output(CO) to meet body’s demand for oxygen and nutrients
- In heart failure, impaired contraction of the heart (systolic dysfunction) or filling of the heart (diastolic dysfunction) may cause pulmonary or systemic congestion

Pathophysiology

Given according to the cause:

- Left-sided verses right-sided failure
- Systolic or diastolic failure
- Low-output or high output failure
- Acute or chronic failure
- Forward or backward failure

Pathophysiology

- **Left-sided Heart Failure:**

✓Pumping ability of the left ventricle fails and cardiac output falls

✓Blood backs up in the left atrium and lungs, causing pulmonary congestion and decreased cardiac output.

✓Causes include: hypertension, myocardial infarction of left ventricle, or valvular heart disease

- **Right-sided Heart Failure: (Cor pulmonale)**

✓Ineffective contractile function of the right ventricle leads to blood backing up into the right atrium, in the vena cava and the peripheral circulation (venous system), which results in peripheral edema and engorgement of the kidneys and other organs.

✓Causes include: severe pulmonary disease and myocardial infarction of right ventricle.

Systolic or diastolic failure

Systolic heart failure:

- Due to ventricle failure to contract adequately to eject enough blood through aorta into arterial system. Causes decreased ventricular blood ejection
- Caused by loss of myocardial cells in ischemia, myocardial infarction (contractile dysfunction), cardiomyopathy or inflammations, increased afterload (hypertension), mechanical abnormalities (e.g. valvular disease)
- manifested in effects of low CO e.g. weakness, fatigue, exercise intolerance
- Over time, it causes left ventricle dilatation and hypertrophy

Diastolic heart failure:

- Occurs when heart is unable to completely relax (stiffness and non-compliant heart muscle) in diastole, thus disrupts normal filling
- Passive diastole filling decreases thereby decreasing stroke volume and cardiac output, but increases atrial contraction to preload
- Diastolic function occurs from chronic hypertension (most common), decreased ventricular compliance due to hypertrophy, and impaired heart muscle contraction
- Manifested by dyspnoea, tachypnoea, respiratory crackles in if left ventricle affected and pulmonary hypertension. Neck vein distension, liver enlargement, anorexia, nausea, if right ventricle is affected

Low-output VS high-output failure:

➢***Low-output failure***

- Develops when there is a decrease in the biventricular output resulting from: coronary heart disease, hypertension, cardiomyopathy, and other primary cardiac disorders.

➢***High-output failure (increased/normal cardiac output)***

- Hypermetabolic states increases CO to maintain blood flow and O2 to tissues
- This activates compensatory mechanisms to increase CO thus further increasing O2 demand
- Though high CO, the heart is unable to meet increased demands of the tissues and thus fails
- Causes include: septicemia, anaemia, hyperthyroidism or AV shunting

Acute VS chronic failure:

- ***Acute failure***: it’s life-threatening and occurs abruptly resulting from myocardial injury e.g. massive myocardial infarction manifested by sudden decrease in cardiac function and signs of decreased CO. Manifests as pulmonary oedema and congestion
- ***Chronic failure***: progressive (gradual) decline or deterioration of heart muscle, which allows compensatory mechanisms to come into play. It causes decreased cardiac output.

✓Causes include: cardiomyopathies, valvular disease, or coronary heart disease (CHD)

Pathophysiology of Compensatory mechanism leading to heart failure

- There are three primary compensatory mechanisms are:

1. **The Frank-Starling mechanism**: that is, the greater the stretch of the cardiac muscle, the greater the force of contraction. This increases contractile force thereby increasing CO. Complications include: *increased myocardial demand; limited by overstretching.*

pathophysiology

2. Neuroendocrine response including activation of SNS and RAAS

- Decreased CO induces SNS and catecholamine release, thereby increasing HR, BP, contractility, vascular resistance and venous return.
- **Complications include**: tachycardia, decreased filling time, low CO, increased vascular resistance, increased myocardial work and O2 demand

pathophysiology

- Decreased CO and renal perfusion induce renin-angiotensin system. This causes vasoconstriction and increased BP. Complications include: increased myocardial work, renal vasoconstriction, and decreased renal perfusion
- ACE converts angiotensin I to angiotensin II, which stimulates aldosterone release from renal cortex resulting in salt and water retention by kidneys, and increased vascular volume overload and vasoconstriction, increasing BP and afterload. Complications include: increased preload and afterload; pulmonary congestion; increased stress on ventricular wall (increased cardiac work-load).

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- ADH is released from neurohypophysis, which inhibits water excretion; while atrial natriuretic factor that is released from the heart increases Na excretion and diuresis. Complications include; fluid retention and increased preload and afterload
- Blood flow is redistributed to vital organs (heart and brain) thereby decreasing perfusion of other organ systems, skin and muscles. Complications include: renal failure, anaerobic metabolism, and lactic acidosis.

pathophysiology

3. Ventricular hypertrophy:

- Increased cardiac workload causes myocardial muscle to hypertrophy and ventricles to dilate.
- This in turn leads to increased contractile force to maintain CO.
- This effect complicates into: increased myocardial O2 demand and cellular enlargement
- The enlarged myocardial cells become dysfunctional and die early (apoptosis), thus leading to low CO.

causes

- Mitral stenosis secondary to rheumatic heart disease, constrictive pericarditis, or atrial fibrillation
- Mitral and aortic insufficiency
- Arrhythmias
- Hypertension
- Atherosclerosis with myocardial infarction (MI)
- Myocarditis
- Ventricular and atrial septal defects

causes

- Endocarditis
- Pregnancy
- Thyrotoxicosis
- Pulmonary embolism
- Infections
- Anaemia
- Emotional
- Increased sodium or water intake
- Renal failure

Incidence

- Affects less than 5% in ages between 55 – 64 years
- Affects about 10% of people older than 65 years

common characteristics

- Reduced cardiac output
- Shortness of breath
- Peripheral oedema
- Dyspnoea on exertion

Classification heart disease by New York Heart Association

Class I:

No limitation of physical activity

Ordinary activity does not cause fatigue, dyspnoea, palpitation or anginal pain

Class II:

Slight limitation of physical activity

No symptoms at rest

Ordinary physical activity causes fatigue, dyspnoea, palpitations or anginal pain

Classification cont.

Class III:
Marked limitation of physical activity but usually comfortable at rest
Ordinary physical activity causes fatigue, dyspnoea, palpitations, or anginal pain
Class IV:
Inability to carry on any physical activity without any discomfort
Cardiac insufficiency symptoms or angina may be present
Increased discomfort for any activity undertaken

complications

- Pulmonary oedema
- Organ failure, especially the brain and kidneys
- Myocardial infarction
- Pleural effusion
- Left ventricular thrombus

Complications cont.

- Hepatomegaly (right ventricular failure)
- Splenomegaly (in right ventricular failure)
- Ascites (in right ventricular failure)
- Dysarrhythmias- due to enlargement of chambers of the heart
- Cardiomyogenic shock

Diagnostic assessments

History of:

- A disorder or condition that can precipitate heart failure
- Dyspnoea or paroxysmal nocturnal dyspnoea
- Peripheral oedema
- Fatigue
- Weakness
- Insomnia

History of:

- Anorexia
- Nausea
- Sense of abdominal fullness, due to right-sided heart failure
- Substance abuse e.g. alcohol, drugs, tobacco.

Physical exam findings

- Cough that produces pink, frothy sputum
- Cyanosis of the lips and nail beds
- Orthopnoea
- Pale, cool, clammy skin
- Diaphoresis
- Jugular vein distension
- Ascites resulting poor venous return from abdomen and liver congestion
- Tachycardia
- Pulsus alternans- weak pulse alternating with strong one

Physical findings cont.

- Hepatomegaly and, possibly, splenomegaly
- Decreased pulse pressure
- S3 and S4 heart sounds
- Moist, bibasilar crackles, rhonchi, and expiratory wheezing
- Decreased pulse oximetry
- Peripheral oedema
- Decreased urinary output

Lab test results:

- B-type natriuretic peptide immunoassay is elevated
- Liver function tests e.g. ALT, AST, LDH, serum bilirubin, and total protein and albumin levels are evaluated to show effect of heart failure on the liver
- Thyroid function tests e.g. TSH, TH levels done because both hypothyroidism and hyperthyroidism can be primary or contributory factor for heart failure
- In acute heart failure, ABGs done to evaluate gas exchange in lung tissues

- Serum electrolytes measured to evaluate fluid and electrolyte status
- Urinalysis, BUN and serum creatinine done to evaluate renal function

Imaging results

- Chest x-ray shows increased pulmonary markings, interstitial oedema, or pleural effusion and cardiomegaly
- Radionuclide imaging to evaluate ventricular function and size

Diagnostic procedures

- ECG reflects heart strain or enlargement or ischemia; atrial enlargement, tachycardia, extrasystole, or atrial fibrillations
- Pulmonary artery pressure monitoring typically shows elevated pulmonary artery wedge pressures, left ventricular end-diastolic pressure in left-sided heart failure, and elevated right atrial or central venous pressure in right-sided heart failure

Management

General management:

- Antiembolism stockings to promote venous return
- Elevation of lower extremities to relieve oedema
- Sodium restricted diet to prevent fluid retention
- Fluid restriction to prevent fluid overload
- Calorie restriction if indicated to prevent obessity
- Low-fat fat diet to prevent atherosclerosis
- Walking programs to reduce weight to optimum level
- Activity as tolerated to keep fit and prevent dependance

Medications

- **Oxygen therapy** as necessary
- **Diuretics**, such as frusemide, bumetanide, torsemide, metolazone and hydrochlorothiazide
- **Inotropic drugs** such as digoxin (digitalis), dobutamine, and dopamine to reduce heart rate and improve cardiac output
- **Vasodilators** such as nitrates (nitroglycerin), sodium nitroprusside, isosorbide, and nesiritide- causes dilatation of arteries and veins
- **Angiotensin-converting enzyme inhibitors**, such as captopril, enalapril, and lisinopril

- **Angiotensin-receptor blockers**, such as losartan, valsartan, and irbesartan to prevent action of angiotensin II and produce vasoconstriction and increased salt and water excretion. Full effect experienced after 3-6 weeks.
- **Calcium channel blockers**, such as amiodipine, felodipine, nefedipine, verapamil to block movement of extracellular Ca++ into cells, causing vasodilation and decreased HR, conractility and systemic vascular resistance

- **Potassium –sparing diuretics** e.g. amiloride and spilonolactone (Aldactone) for exchange of K+ and Na+ in distal renal tubules and reduce excretion of K+, H+, Ca++ and Mg++
- **Beta-adrenergic blockers**, such as atenolol, metoprolol, propranolol (inderal) and carvedilol
- **Anticoagulants**, such as warfarin
- **Morphine**- decreases preload and afterload in acute decompesated heart failure and pulmonary oedema, dilates pulmonary and systemic blood vessels.

Surgical intervention

- For valvular dysfunction with recurrent acute heart failure, surgical replacement
- Heart transplant – transfer of a heart from one person (especially for one who has suffered massive brain dead) to another
- Ventricular assist device
- Stent placement

Nursing management

Nursing Diagnoses

- Decreased cardiac output related to altered contractility, altered preload and altered stroke volume as evidenced by jugular vein distension, orthopnoea, S3, S4
- Impaired gas exchange related to increased preload and alveolar capillary membrane changes AEB dyspnoea, tachypnoea, restlessness, verbalisation , “am short of breath”

- Excess fluid volume related to increased venous pressure and decreased renal perfusion secondary to HF AEB edema, rapid weight gain, oliguria.
- Activity intolerance related to fatigue secondary to cardiac insufficiency and pulmonary congestion
- Deficient knowledge: low sodium diet related to lack of information about disease process

Expected client outcomes

- The client will:

✓ Maintain haemodynamic stability

✓ Maintain cardiac output

✓ Carry out activities of daily living without excess fatigue or decreased energy

✓ Maintain adequate ventilation

✓ Maintain adequate fluid balance

Nursing interventions

- Place patient in Fowler’s position
- Give supplemental oxygen
- Provide continuous cardiac monitoring during acute and advanced stages
- Assist the patient with range-of-motion exercises
- Apply antiembolism stockings
- Check for calf pain and tenderness
- Administer prescribed drugs
- Provide emotional support

Monitor closely for the following:

- Daily weight for peripheral oedema and other signs and symptoms of fluid overload
- Cardiac rhythm
- Intake and output
- Response to treatment
- Vital signs: TPR and BP and report to physician any deviation
- Mental status
- Peripheral oedema

monitoring

- Auscultate for abnormal heart beat and breath sounds, report any changes immediately
- Blood urea and nitrogen and serum creatinine, potassium, sodium, and magnesium levels
- Prothrombin time

Patient Teachings

- You must the following patient teachings:

✓Heart failure disorder, diagnosis and treatment

✓Signs and symptoms of worsening heart failure

✓When to notify the physician

✓The importance of follow up-care

✓The need to avoid high-sodium foods

✓The need to avoid fatigue

✓Instructions about fluid restrictions

Patient teaching

- ✓The need to take weight every morning, at the same time, before eating, and after urinating, keeping record of this weight, and reporting weight gain of 1.5 to 2.5 kg in a week
- ✓The importance of smoking cessation, if appropriate
- ✓Diet education to include low sodium and low cholesterol diet intake
- ✓Weight reduction as needed for optimal wt management
- ✓Medication administration, dosage, possible adverse effects, and monitoring needs

Discharge planning

- Encourage client to attend for follow-ups
- Refer the client to a smoking-cessation program, if appropriate