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ANGINA PECTORIS

Angina is chest pain resulting from myocardial ischemia caused by inadequate myocardial blood and oxygen supply.

Angina is central chest tightness or heaviness due to myocardial ischemia. The pain is brought about by emotional upset, physical exertion and is relieved by rest. It may radiate to arms, neck, jaw and teeth.

Cause:

- AtheromatousEpicardiac Coronary Obstruction,
- Anaemia,
- Tight Aortic Stenosis,
- Tachy Arrhythmia,
- Arteritis And Small Vessel Disease
- Cold whether
- Heavy meals.

It is of 4 types.

Patterns of angina:

1. Stable angina

a. Also called exertional angina

b. Occurs with activities that involve exertionor emotional stress; relieved with rest ornitroglycerin

c. Usually has a stable pattern of onset, duration, severity, and relieving factors

2. Unstable angina

a. Also called preinfarction angina

b. Occurs with an unpredictable degree of exertion or emotion and increases in occurrence,duration, and severity over time

c. Pain may not be relieved with nitroglycerin.

3. Variant angina

a. Also called Prinzmetal's or vasospasticangina

b. Results from coronary artery spasm

c. May occur at rest

d. Attacks may be associated with ST-segmentelevation noted on the ECG.

4. Intractable angina is a chronic, incapacitatingangina unresponsive to interventions.

5. Preinfarction angina

a. Associated with acute coronaryinsufficiency

b. Lasts longer than 15 minutes

c. Symptom of worsening cardiac ischemia

d. Characterized by chest pain that occurs daysto weeks before an MI.

Clinical Manifestation:

1. Pain

a. Pain can develop slowly or quickly.b. Pain usually is described as mild ormoderate.

c. Substernal, crushing, squeezing pain mayoccur.

d. Pain may radiate to the shoulders, arms,jaw, neck, or back.

e. Pain intensity is unaffected by inspirationand expiration.

f. Pain usually lasts less than 5 minutes; however, pain can last up to 15 to 20 minutes.

g. Pain is relieved by nitroglycerin or rest.

2. Dyspnea

3. Pallor

4. Sweating

5. Palpitations and tachycardia

6. Dizziness and syncope

7. Hypertension

8. Digestive disturbances

Diagnostic studies

1. Electrocardiography: Readings are normal during rest, with ST depression or T-wave inversionduring an episode of pain.

2. Stress testing: Chest pain or changes in the ECGor vital signs during testing may indicateischemia.

3. Cardiac enzyme and troponin levels: Findingsare normal in angina.

4. Cardiac catheterization: Catheterization provides a definitive diagnosis by providing information about the patency of the coronaryarteries.

Interventions:

1. Immediate management

a. Assess pain, institute pain relief measures.

b. Administer oxygen by nasal cannula as prescribed.

c. Assess vital signs and provide continuous cardiac monitoring and nitroglycerin as prescribed to dilate the coronary arteries, reduce the oxygen requirements of the myocardium, and relieve the chest pain.

d. Ensure that bed rest is maintained, place the client in semi-Fowler's position, and stay with the client.

e. Obtain a 12-lead ECG.

f. Establish an IV access route.

OTHER MANAGEMENT:

- GTN spray/SL nitrates every ½ hr till symptom relief or hypotension.
- Aspirin 75-150 mg daily (reduces mortality by 34%).
- Isosorbidcmononitrate 10-30 mg PO bid or slow release (e.g. Imdur-60 mg/24 hr); alternatively nitrate skin patch.
- Aetinolol- 25-50 mg daily (unless there is contraindication e.g., COPD, asthma, variant angina, LVF).
- CCB – diltiazem 90-180 mg bid.

- K- cannel activator – nicorandil 10-30 mg bid
- Treat associated conditions like diabetes, hypertension and dyslipidemia, obesity.
- Referral to cardiac center if no pain relief, new angina of sudden onset, recurrent angina, past MI/CABG, unstable angina, pain duration exceeds 30 minutes.
- Risk stratification by stress ECG and further planning (CABG vs balloon angioplasty).
- Behavioral modification – stop smoking, weight loss if obese, discontinuation of HRT, low fat high fiber diet, meditation – yoga etc.

CONGESTIVE HEART FAILURE

Introduction:

Heart failure is the inability of the heart to maintain adequate cardiac output to meet the metabolic needs of the body because of impaired pumping ability.

Diminished cardiac output results in inadequate peripheral tissue perfusion.

Congestion of the lungs and periphery may occur; the client can develop acute pulmonary edema

Definition :

Cardiac failure is a state where heart fails to pump enough blood to meet metabolic demand. LVF or RVF may occur independently or together, the latter called congestive heart failure.

Classification:

1. Acute heart failure occurs suddenly.

2. Chronic heart failure develops over time; however, a client with chronic heart failure can develop an acute episode.

Types of heart failure:

1. Right ventricular failure, left ventricular failure

a. Because the 2 ventricles of the heart represent 2 separate pumping systems, it is possible for

1 to fail alone for a short period.

b. Most heart failure begins with left ventricular failure and progresses to failure of both ventricles.

c. Acute pulmonary edema, a medical emergency, results from left ventricular failure.

d. If pulmonary edema is not treated, death will occur from suffocation because the client literally drowns in his or her own fluids.

2. Forward failure, backward failure

a. In forward failure, an inadequate output of the affected ventricle causes decreased perfusion to vital organs.

b. In backward failure, blood backs up behind the affected ventricle, causing increased pressure in the atrium behind the affected ventricle.

3. Low output, high output

a. In low-output failure, not enough cardiac output is available to meet the demands of the body.

b. High-output failure occurs when a condition causes the heart to work harder to meet the demands of the body.

4. Systolic failure, diastolic failure

a. Systolic failure leads to problems with contraction and ejection of blood.

b. Diastolic failure leads to problems with the heart relaxing and filling with blood.

Compensatory mechanisms

1. Compensatory mechanisms act to restore cardiac output to near-normal levels.

2. Initially, these mechanisms increase cardiac output; however, they eventually have a damaging effect on pump action.

3. Compensatory mechanisms contribute to an increase in myocardial oxygen consumption; when this occurs, myocardial reserve is exhausted and clinical manifestations of heart failure develop.

4. Compensatory mechanisms include increased heart rate, improved stroke volume, arterial vasoconstriction, sodium and water retention, and myocardial hypertrophy.

SYMPTOMS:

RHF:

- Dependent edema (legs and sacrum)
- Jugular venous distention

- Abdominal distention
- Hepatomegaly
- Splenomegaly
- Anorexia and nausea
- Weight gain
- Nocturnal diuresis
- Swelling of the fingers and hands
- Increased BP (from fluid volume excess) or decreased BP(from pump failure).

LHF:

- Signs of pulmonary congestion
- Dyspnea
- Tachypnea
- Crackles in the lungs
- Dry, hacking cough
- Paroxysmal nocturnal dyspnea
- Increased BP (from fluid volume excess) or decreased BP (from pump failure)

INVESTIGATIONS:

- Raised BNP and ANP, low ejection fraction (BNP> 100 ng/L has highest diagnostic value, even better than EF).
- CXR- cardiomegaly, upper lobar venous diversion, Kerly lines, peribronchical cuffing, fluid in fussures, batwing appearance.
- Echo – chamber dilatation and dysfunction.

MANAGEMENT:

1. *Acute heart failure*
    1. Place the client in a high Fowler's position.
    2. Administer oxygen.
    3. Assess the client quickly, including assessing lung sounds.
    4. Ensure that an intravenous (IV) access device is in place.
    5. Prepare for the administration of a diuretic and morphinesulfate.
    6. Insert a Foley catheter as prescribed.
    7. Prepare for intubation and ventilator support, if required.

8. Document the event, actions taken, and the client's response

2. *Chronic heart failure*
    - Treat exacerbating factors (anemia, infection, thyroid disease and hypertension).
    - Salt restriction, activity restriction, smoking cessation
    - Frusemide 40 mg PO or aldactone 25 mg PO, if edema refractory – metolazone 5-20 mg PO daily.
    - ACE inhibitor (enalapril 5 mg). Avoid if hyperkalemia, renal failure, hyponatremia, hypovolemia, pregnancy lactation, COPD etc. Be aware of side effects like dry cough, renal impairment, hyperkalemia, proteimuria and leukopenia. If side effects severe substitute by receptor antagonist – losartan 25-100 mg/day.
    - Betablocker – low dose like bisoprololcarvedilol.
    - Digoxin – 0.125-0.25 mg daily; monitor K+ level.
    - Vasodilators – long acting nitrates to reduce preload; e.g., isosorbidemononitrate up to 60 mg daily.

COR PULMONALE

Corpulmonale is the right heart failure caused by chronic pulmonary hypertension. *Causes* include:

1. Chronic lung disease (COPD, bronchiectasis, pulmonary fibrosis, chronic asthma).
2. Pulmonary vascular disease (pulmonary vasculitis, sickle cell disease, parasitic lung disease)
3. Thoracic cage abnormality (kyphosis, scoliosis)
4. Neuromuscular disease (MND, polio, myasthenia gravis)
5. Hypoventilation (sleep apnoea, large adenoids). Pulmonary infection aggravates/precipitates corpulmonale.

DIAGNOSIS:

- Symptoms : dyspnoea, fatigue, syncope
- Signs: ↑ JVP, cyanosis, early diastolic murmur (Graham Steel murmur), tender pulsating hepatomegaly, and edema.
- Investigations : ↑ haematocrit due to polycythemia,
- ABG – hypoxia; CXR – large RA, RV.
- ECG

MANAGEMENT:

- Treatment of underlying cause, infection if any.
- Treatment of cardiac failure with diuretics, ACE intibitor and ± digoxin
- Venesection if haematocrit>55 %
- Treatment of respiratory failure - O_2 therapy if PaO_2 < 8KPa. Start with 24% O_2, increase depending on ABG. In COPD longterm O_2 therapy (LTOT) 15 hours/day may increase survival.
- Pulmonary vasodilators to reduce pulmonary hypertension (eg. Sildenafil, nitric oxide)
- Consider heart lung transplantation in young patients.
- Prognosis: 50% death within 5 years.

CARDIAC TAMPONADE

A pericardial effusion occurs when the space between the parietal and visceral layers of the pericardium fills with fluid.Pericardial effusion places the client at risk for cardiac tamponade, an accumulation of fluid in the pericardial cavity.

Tamponade restricts ventricular filling, and cardiac output drops.

Signs and symptoms:

1.Pulsusparadoxus

2. Increased CVP

3. Jugular venous distention with clear lungs

4. Distant, muffled heart sounds

5. Decreased cardiac output

6. Narrowing pulse pressure

DIAGNOSIS:

- Tachycardia, low blood pressure, raised JVP, Kussmaul's sign, muffled S1 and S2.
- Beck's triad – falling BP, raised NVP, small quiet heart
- CXR – Big globular heart (if > 250 ml).

- ECG – low voltage QRS ± electrical alternans.
- ECHO – Diastolic collapse of RA and RV.

TREATMENT:

- Treatment of underlying cause – tuberculosis, hypothyroidism, MI, uremia, collagen vascular disease.
- Pericardiocentesis under Echo guidance
- Pericardiectomy if constriction is severe.