Cardiac Diseases

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Heart Failure

- Heart failure generally is referred to as *congestive heart failure* (CHF). CHF is the common end point for many forms of cardiac disease and typically is a progressive condition that carries an extremely poor prognosis.
- Most cases of heart failure are due to
  1. Systolic dysfunction—inadequate myocardial contractile function, characteristically a consequence of ischemic heart disease or hypertension.
  2. Diastolic dysfunction—inability of the heart to adequately relax and fill, such as in massive left ventricular hypertrophy, myocardial fibrosis, amyloid deposition, or constrictive pericarditis.
### Causes of congestive heart failure

#### Right heart failure:
1. LHF
2. Primary disease of pulmonary vasculature
3. Pulmonary or tricuspid valve disease
4. Cardiomyopathies & myocarditis
5. Primary lung diseases

#### Left heart failure:
1. Ischemic heart disease
2. Hypertension
3. Aortic & mitral valve disease
4. Myocardial disease
Manifestations of heart failure

**LHF**
1. Dyspnea: exertional, orthopnia, paroxysmal nocturnal dyspnea due to congestion & edema of the lungs
2. Large heart, S3, systolic murmur; due to dilatation of heart & displacement of papillary muscle of mitral valve
3. Atrial fibrillation: due to chronic dilatation of left atrium
4. Renal failure: due to renal hypoxia
5. Hypoxic/ischemic encephalopathy: due to CNS hypoxia

**RHF**
1. Peripheral edema
2. Effusion in body cavities (ascitis, pleural effusion)
3. Dusky face, engorged veins of the neck
4. Chronic liver congestion & congestive splenomegaly
Ischemic heart diseases (IHD)

- Ischemic heart disease (IHD) is a broad term encompassing several closely related syndromes caused by myocardial ischemia—an imbalance between cardiac blood supply (perfusion) and myocardial oxygen and nutritional requirements.
- It accounts for 80% of cardiac death and nearly 1/3rd of all deaths in USA.
- Most common underlying cause is atherosclerosis of coronary arteries.
- **Clinical syndrome of IHD includes:**
  - Angina pectoris
  - Myocardial infarction
  - Sudden cardiac death
  - Chronic IHD & CHF
Pathogenesis of ischemic heart disease
Pathogenesis of ischemic heart disease

- IHD is primarily a consequence of inadequate coronary perfusion relative to myocardial demand. This imbalance occurs as a consequence of the combination of preexisting (“fixed”) atherosclerotic occlusion of coronary arteries and new, superimposed thrombosis and/or vasospasm.

- Fixed obstructions that occlude less than 70% of a coronary vessel lumen typically are asymptomatic, even with exertion. In comparison, lesions that occlude more than 70% of a vessel lumen—resulting in so-called critical stenosis—generally cause symptoms.
Other that play in IHD

1. Acute change in plaque morphology:
   ◦ Fissuring, hemorrhage & rupture
     • embolization of debris into distal coronary artery
     • Increase platelet aggregation
     • increase risk of thrombosis
   ◦ Platelet aggregation causes
     • mechanical occlusion
     • vasospasm
     • coronary artery thrombosis

2. Non atherosclerotic lesion of coronary artery
   ◦ Emboli from vegetation on valves
   ◦ Vasculitis
   ◦ Severe hypotension
   ◦ Spasm of coronary artery

3. Increase myocardial demands
   ◦ Hypertrophy
   ◦ Cardiomyopathies
Angina Pectoris

A clinical syndrome characterized by paroxysmal attacks of chest pain usually situated retrosternal, radiating to the left shoulder.

1. **Classical AP**
   - precipitated by exertion & emotion
   - relieved by rest & nitroglycerine
   - caused by critical stenosis by atherosclerosis

2. **Vasopastic angina**
   - can occur in rest less frequently related to effort
   - respond to nitroglycerine
   - St segment elevation in ECG
   - caused by CA spasm over atherosclerosis
3. **Unstable angina (preinfarction angina)**

- More frequent
- Provoked by less effort or emotion
- Lasts longer
- Nitroglycerine is required more & less effective
- Carries a bad prognostic implication
- Caused by acute plaque lesion with superimposed partial thrombosis
Acute Myocardial Infarction

- An area of ischemic necrosis
- Acute MI is the most common cause of death in West
  1.5 million of MI / yr in USA with ½ million deaths.
  Large number of which don’t reach hospital
- 2 types of MI
  1. Transmural MI: full thickness infarction caused by severe atheroma with acute plaque changes leading to complete occlusion.
  2. Subendocardial MI: limited to inner 1/3rd or ½ of wall thickness caused by global ischemia due to atheroma with increased demand often super-imposed on chronic non-critical coronary stenosis
This cross section through the heart demonstrates the left ventricle on the left. A large recent myocardial infarction extending from the anterior portion into the septum. The center is tan with surrounding hyperemia. The infarction is "transmural" in that it extends through the full thickness of the wall.
Morphology of MI

- Size of MI depends on segment of coronary artery blocked & collateral circulation
- The location of MI depends on site of occlusion
  - LAD (40-50%):
  - RCA (30-40%):
  - LCA (15-20%)
Morphology of MI

Gross pathology:

- MI fewer than 12 hr old are usually unapparent on gross examination.
- 12-24 hr............slight pallor area
- 4-7 days............central pallor with hyperemic border
- 10 days.............yellow soft shrunken center with purple border
- 7-10 weeks........firm gray fibrous tissue
Histology:
- Necrosis start after 20-40 min of ischemia
- Coagulative necrosis become apparent microscopically by about 4-12 hrs
- 1-2 hr..............few wavy fibers at the margin of infarct
- 4-12 hr.............early coagulative necrosis, edema, occasional polymorph
- 12-24 hr...........established coag. necrosis, heavy polymorph
- 4-7 days...........macrophages appear, disintegration of muscle fibers & phagocytosis, granulation tissue at border
- 7-10 days.........granulation tissue maximum
- 7-10 weeks........fibrosis
## Myocardial Infarction

<table>
<thead>
<tr>
<th>Time</th>
<th>Histology</th>
<th>Grossly</th>
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<tbody>
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Acute M.I (1-2) days: dark red contraction band necrosis extending along myocardial fibers with coagulative necrosis
Intermediate M.I with 1-2 weeks age: Note that there are remaining normal myocardial fibers at the top. Below these fibers are many macrophages along with numerous capillaries and little collagenization (granulation tissue)
There is pale white collagen within the interstitium between myocardial fibers. This represents an area of remote infarction.
Infarction modification after reperfusion

- Reperfusion sufficiently early (within the first 15-20 min) may prevent all necrosis.
- If delay after 15-20 min might limit the size of infarction & induced some changes includes:
  - Hemorrhage into ischemic area due to vascular injury.
  - Contraction band necrosis in fibers at periphery of infarct caused by hypercontraction of myofibrils in dying cells which induced by influx of calcium from plasma into the cells after the damage of cell membrane by free radicals formed by reperfusion.
Reperfusion injury
Clinical features of MI

- Patient has rapid weak pulse, dyspnea, and is sweating profusely
- Diagnosis depends on
  - Typical symptoms
  - Biochemical evidence
  - ECG changes
  - Other diagnostic modalities are mainly radiological

Laboratory Diagnosis of MI

- Cardiac enzymes & proteins
  - **Creatinine kinase-MB**: rise within 2-4 hr, peak at 18 hr & disappear after 48 hr
  - **Ld1**: rises within 24 hr, peaks at 72 hr & persist for 1 week
Troponines (I & T): most specific, appear similar to CK but remain 7-10 days

C-reactive protein serves as a marker to predict the risk of MI in patients with angina and the risk of new infarcts in patients who recover from infarcts

ECG changes: q wave, ST elevation & T inversion.

Consequences and complications of MI

- Half of the deaths occur within one hour of onset of symptoms

- Factors associated with poor prognosis:
  - Advanced age
  - Female gender
  - Diabetes mellitus
  - Previous MI
Complication of MI

- After infarction about 25% of patients experience sudden death due to fetal arrhythmias.
- If patient survive the acute event, 80-90% develop complications:
  1. Arrhythmias (75-95%)
  2. CHF (60%)
  3. Cardiogenic shock.
  4. Rarely papillary muscle infarction with or without rupture leading to mitral valve dysfunction.
  5. Mural thrombosis with thromboembolic phenomena (15-40%)
  6. Ventricular rupture (1-5%)
  7. Venticular aneurysm
  8. Fibrinous or hemorrhagic pericarditis
In cross section, the point of rupture of the myocardium is shown with the arrow. Rupture occurs through the already thin infarcted ventricular wall 3-5 days following M.I.
Left ventricular aneurysm with mural thrombus inside it, as a complication of old MI
Rheumatic Fever & Rheumatic Heart Disease
Etiology & Pathogenesis of RF

RF is an immune reaction secondary to streptococcus infection

Antibodies against strept. antigen cross react with tissue antigens including cardiac antigens

Major Criteria:
1. Pancarditis
2. Migratory polyarthritis
3. Subcutaneous nodules
4. Sydenham’s chorea
5. Erythema marginatum of the skin

Minor Criteria:
1. Fever
2. Arthralgia
3. Elevated ASOT
4. Elevated acute phase reactant C protein
5. Prolongation of PR intervals

RF is an acute recurrent immunological mediated multi systemic inflammatory disease mainly affecting children following pharyngitis with group A beta hemolytic streptococci & characterized by Jones Criteria
Morphology
1/ Acute attack
The pathognomic lesion is disseminated focal inflammatory foci known as Aschoff bodies

Aschoff body:
- focus of fibrinoid necrosis surr. by a collagen, lymphocytes, macrophages, & few plasma cells plus modified histiocytes known as Antischow cells & multinucleated cells known as Aschoff giant cells.
- Typically seen in myocardium esp. around blood vessels
2/ Chronic attack

- Fibrosis & deformities of the heart esp. the valves
- Mitral valve most commonly affected followed by aortic valve, tricuspid valve & lastly pulmonary valve
Chronic Rheumatic Heart Disease

- More likely to occur in:
  1. First attack in early childhood
  2. Severe initial attack of RF
  3. Recurrent attacks

- Mitral valve alone most commonly involved in 65-70% of cases, then mitral & aortic valves in combination in 25%, less affection is aortic valve alone

- Pulmonary & tricuspid valves are rarely affected
Chronic Rheumatic Heart Disease

Mitral Valve:
1- Fibrous thickening & calcification of leaflets
2- Fusion of commissaries result in fish mouth or button hole deformity & stenosis
3- Chorda tendeni are thickened, fused & shortened
4- Mitral stenosis or combined stenosis & incompetence
5- More common in female

Aortic Valve:
1- Similar to MV with prominent nodular calcification in the sinuses-bind the leaflets
2- Aortic stenosis or combined stenosis & incompetence
3- More common in male
Aneurysms:

It is a localized dilatation of the vessel or heart wall. We have two types:

1. True
2. False.

Causes, include:

1. Atherosclerosis.
2. Congenital.
3. Trauma.
4. Infection (syphilis).
5. Inflammation.

Morphologically: there are 2 types

1. Saccular.
2. Fusiform.
Veins and Lymphatics:

**Varicose Veins**

Abnormally dilated, tortuous veins produced by prolonged increased intraluminal pressure and loss of vessel wall support. The superficial veins of the legs are the main sites of involvement. People at risk are:

1. Occupations with long period of standings.
2. People older than 50 years.
3. Obese individuals.
4. Women with multiple pregnancies.
5. Familial tendency (wall weakness).

**Morphology** the dilated veins are tortous, elongated and scarred, with thrombosis.

**Clinical course**

Leg edema with stasis dermatitis. No embolism. Apart from legs, varices occur also at the lower end of esophagus and ano-rectum (hemorrhoids).
Varicose veins of the leg (arrow). (Courtesy of Dr. Magruder C. Donaldson, Brigham and Women’s Hospital, Boston, Massachusetts.)
Hypertension

- Hypertension is a common disorder affecting 25% of the population; it is a major risk factor for atherosclerosis, congestive heart failure, and renal failure.

- Essential hypertension represents 95% of cases and is a complex, multifactorial disorder, involving both environmental influences and genetic polymorphisms that may influence sodium resorption, aldosterone pathways, and the renin–angiotensin system.
Pathogenesis

Most of the cases of hypertension (95%) are idiopathic (essential hypertension). This form is compatible with long life unless a myocardial infarction, stroke, or another complication supervenes.

Most of the remaining cases (secondary hypertension) are due to primary renal disease, renal artery narrowing (renovascular hypertension), or adrenal disorders.
Pathogenesis
Types and causes of hypertension

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<th>Essential Hypertension</th>
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<tbody>
<tr>
<td>Accounts for 90% to 95% of all cases</td>
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<tr>
<th>Secondary Hypertension</th>
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<tr>
<td>Renal</td>
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<td>Acute glomerulonephritis</td>
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<td>Chronic renal disease</td>
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<td>Polycystic disease</td>
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<tr>
<td>Renal artery stenosis</td>
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<td>Renal vasculitis</td>
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<td>Renin-producing tumors</td>
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<th>Endocrine</th>
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<tr>
<td>Adrenocortical hyperfunction (Cushing syndrome, primary</td>
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<td>aldosteronism, congenital adrenal hyperplasia, licorice</td>
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<td>ingestion)</td>
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<td>Exogenous hormones (glucocorticoids, estrogen [including</td>
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<td>pregnancy-induced and oral contraceptives], sympathomimetics</td>
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<td>and tyramine-containing foods, monoamine oxidase</td>
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<td>inhibitors)</td>
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<tr>
<td>Pheochromocytoma</td>
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<td>Acromegaly</td>
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<td>Hypothyroidism (myxedema)</td>
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<td>Pregnancy-induced (pre-eclampsia)</td>
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<td>Coarctation of aorta</td>
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<tr>
<td>Polyarteritis nodosa</td>
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<td>Increased intravascular volume</td>
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<tr>
<td>Increased cardiac output</td>
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<td>Rigidity of the aorta</td>
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<td>Increased intracranial pressure</td>
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<tr>
<td>Sleep apnea</td>
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<td>Acute stress, including surgery</td>
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Mechanism of essential hypertension:

1. **Reduced renal sodium excretion** This is a common etiologic factor in most forms of hypertension. Decreased sodium excretion causes an obligatory increase in fluid volume and increased cardiac output, thereby elevating blood pressure.

2. **Increased vascular resistance** may stem from vasoconstriction or structural changes in vessel walls.

3. **Genetic factors**

4. **Environmental factors** such as stress, obesity, smoking, physical inactivity.
Morphology

Two forms of small blood vessel disease are hypertension-related: hyaline arteriolosclerosis and hyperplastic arteriolosclerosis

1. **Hyaline arteriolosclerosis** is associated with benign hypertension. It is marked by homogeneous, pink hyaline thickening of the arteriolar walls, with loss of underlying structural detail, and luminal narrowing.

2. **Hyperplastic arteriolosclerosis** is more typical of severe hypertension. Vessels exhibit “onionskin,” concentric, laminated thickening of arteriolar walls and luminal narrowing. The laminations consist of smooth muscle cells and thickened, reduplicated basement membrane.
Hypotension

- Hypotension is low blood pressure.
- Hypovolemia is the most common cause of hypotension, which result from vomiting, diarrhea, and bleeding.
- Other causes include: drugs, large MI, and CHF.