Cases

1- Rheumatic fever
2- Endocarditis
3- Myocardial infarction
4- Hypertension
5- Atherosclerosis
6- Aneurysm
Case 1

- 3 weeks after a bout of acute pharyngitis, a 12 year old child presents with fleeting arthritis affecting big joints, subcutaneous nodules over her elbows together with cardiac affection which appeared as friction rub at auscultation

1- Most probable diagnosis of the case is...
2- Pathogenesis of the disease
3- Morphological changes ( gross/microscopic)
4- Complications
Pathogenesis of the disease

1. Untreated strep sore throat
   - Anti-strep antibodies
     - Cross reaction with
       - Endocardium & joint tissues
         - Pericardium
         - Myocardium
         - Endocarditis
           - Deposition of protein M
           - Effects of Streptolysins O & S
           - Scarring of heart valves
           - Polyarthritis

2. Rheumatic Heart Disease (RHD)
   - Stenosis & Regurgitation

Unit lesion of the disease

1. Structure is called....
2. It is found in....
3. It is a type of .......
   Granuloma
4. It is formed of....
5. Its fate is .......

Granuloma is a type of lesion found in certain diseases, characterized by the formation of a granuloma, which is a collection of immune cells and tissue within the body. Its fate may vary depending on the underlying disease process.
Complications (chronic valve lesions)

Aortic stenosis

Stenosis means........
Effects of aortic stenosis

Aortic incompetence

Incompetence means......
Effects of aortic incompetence..
Complications (chronic valve lesions)

- Mitral stenosis...
  - Hemodynamics.....
  - Effects on the lungs....
  - Cor-pulmonal
Vegetation

- Vegetation of rheumatic fever
  1- Site......
  2- Size......
  3- Adherence to the valve
  ...........
  4- Fate......
Other vegetations

1- **SLE**.... It is called.....
   Site..........  
   Adherence......

2- **SBE**....
   Organism.....
   Valve......
   Site.....
   Size......
   Adherence.....
   Effects......
Case 2

- A 34 year old intravenous drug abuser presents recently with fever and a new cardiac murmur. Blood culture is positive for staph aureus. 2 Days later the patient dies and on autopsy the cardiac valve showed large, yellow irregular vegetation with destruction of the valve

- Most probable diagnosis of the case...
- Describe the course and prognosis of the case.
Acute bacterial Endocarditis

- Organism.....

- Vegetation
  - Site.....
  - Size......
  - Adherence.....
  - Effects......

- Heart..
Acute bacterial Endocarditis

Causative organism ... Staph. Aureus (50% of cases)
- Valves affected.... Mostly mitral and aortic.
  Right side valve affection in drug addicts.
- Lab. Diagnosis.....Blood culture
Case 3

A 55-year-old man presents to the emergency room with chest crushing pain radiating to his left arm and dyspnea for two hours. For several months prior to presentation he has had a few episodes of mild chest pain which developed while climbing stairs. The pain resolved within several minutes after resting. The patient smokes and has been told that his cholesterol is “on the high side”. On exam, the patient is obese. Serum myoglobin and troponin levels are measured:

Myoglobin 106 (0-75ng/mL)
Troponin 1.37 (<0.1ng/mL)
Case 3

1- What is your possible diagnosis?
2- what are the risk factors in this patient?
3- what are the underlying pathogenesis?
4- What are the possible complications of the case?
Ischaemic heart diseases
Acute ischemia (myocardial infarction)

**Causes and risk factors:**

**A-** Complicated atheroma of coronary vessel. (Thrombosis, rupture of the plaque or hemorrhage into the plaque).

**B-** Unusual causes include:

1- Embolism due to vegetations of bacterial endocarditis of aortic valve.
2- Polyarteritis nodosa
3- Occlusion of coronary ostia in dissecting aneurysms and syphilis
4- Coronary spasm
Thrombosis of the coronary artery
Thrombosis on top of atheroma of the coronary artery
Myocardial infarction
Before 6 hours of the occurrence of the infarction, no gross or microscopic changes are detected on the myocardium.
Infarct area is pale opaque and dry. It is slightly swollen.

- Coagulative necrosis of cardiac muscle.
- Infarct area is yellow, surrounded by a zone of hyperemia.
- Removal of necrotic debris
- Granulation tissue invades the infarct area from outside inwards.

- Infiltration by neutrophils then by macrophages which engulf necrotic debris.
- Starting invasion of the infarct by young granulation tissue from the periphery to the center.
+ Serofibrinous inflammation of pericardium.
- Increasing pallor of the infarct due to progressive fibrosis till a well developed grey-white scar is formed.

- Progressive maturation of granulation tissue with collagenization
Myocardial infarction
Fully formed recent infarction

- Infarct area is swollen, yellow, soft and friable.
- It is surrounded by a zone of hyperemia
- It involves most of the myocardial thickness
Complications of myocardial infarction

1- Arrhythmia.....most common cause of death in the first few hours following infarction

2- Myocardial (pump) failure...can lead to congestive heart failure and/or cardiogenic shock
3- Myocardial Rupture

- Occurs within 4-7 days after Infarction (due to neutrophilic liquifactive enzymes).

- Can lead to **cardiac tamponade** and compression of the heart by hemorrhage into the pericardial space.
Myocardial Infarction Complications

4- Myocardial aneurysm with thrombosis inside.

5- Rupture of papillary muscle.....mitral incompetence.
Myocardial Infarction Complications

6- Chronic heart failure.
7- Acute pericarditis.
8- Prolonged confinement to bed +wk cardiac action....leg thrombosis &pulmonary embolism.
Case 4

During a routine medical check up, a 52 year old man was surprised to find his blood pressure to be 155/95 mm. Hg, he has no other previous medical problems and is taking no current medication

1- What is the pathogenesis of this type of hypertension??

2- Discuss the pathological changes that occur in the heart, blood vessels, kidneys and brain as consequence of this disease
Pathogenesis of hypertension
BP = C.O × TPR
I- Benign Hypertension
Heart changes

- With increasing pressure, the left ventricular myocardium undergoes hypertrophy.

- With hypertension, the severity of atheromatous lesions are usually more severe.

- Coronary blood flow may be insufficient leading to ischemic heart diseases
I- Benign Hypertension
(Left ventricular hypertrophy)

Left ventricular failure is a common complication of hypertension
I- Benign Hypertension
Small Blood vessel changes

- Benign arteriolosclerosis
- Benign arteriosclerosis
- Atheroma is severe

hyalinosis and elastosis
I- Benign Hypertension

Hyalinosis
Both changes lead to narrowing of the blood vessels and ischemia of the affected parts and also increased peripheral resistance.

Elastosis
I- Benign hypertension
Renal changes
I- Benign hypertension
Renal changes

1- Hyalinosis and elastosis of afferent and efferent arterioles ..... Ischemic necrosis of glomeruli and related nephron ..... Fibrosis ..... contraction.

2- Compensatory hypertrophy of other nephrons.
3- Interstitial fibrosis & few lymphocytes
1+2+3 are called primary contracted kidney.
Primary contracted kidney

- Kidneys are small in size. Finely irregular outer surface
- Capsule is adherent
- C/S:-
  - Less demarcation between cortex and medulla.
  - Thick arteries at the base of the pyramids
Case 6

- A pulsating abdominal mass is paplated in a 45 y.o male suffering from progressive abdominal pain for the previous 3 months. CT scan revealed a 6 cm. long dilated segment of the abdominal aorta.

1- Localized dilatation of the wall of a blood vessel is called........
2- What are the types, causes and vessels affected by this lesion??
3- What are the possible complications of this lesion??
Aneurysm

- It is localized dilatation of the wall of an artery due to weakening of the wall and/or increase of blood pressure.

- It may be either true in which the wall is part of arterial wall or false in which the wall is formed of fibrous tissue.
1- False aneurysm

- In which the wall is formed of fibrous tissue

1- Simple pulsating aneurysm (pulsating hematoma).

2- Arteriovenous fistula...due to traumatic injury joining two adjacent vessels.
2- True aneurysm

***According to its cause they can be classified

A- Weak wall

1- Congenital weakening of the wall
   a. Berry aneurysm in the circle of Wills at the base of the brain.
   b. Medionecrosis (Marfan's syndrome)...dissecting aortic aneurysm.

2- Inflammatory weakening of the wall:-
   a. Mycotic aneurysm of subacute bacterial endocarditis, polyarteritis nodosa, TB.
   b. Syphilitic aortic aneurysm of thoracic aorta.

3- Degenerative.. Atheroma and hypertensive changes.
2- True aneurysm

B- Increased intravascular pressure
1- Aortic aneurysm
   a. Atheromatous.....abdominal aorta.
   b. Syphilitic...thoracic aorta.
   c. Dissecting....blood passes inside the wall of the aorta. It may be due to medionecrosis of Marfan's syndrome or blood passes in a crack of atheromatous plaque.
Aortic aneurysm Atherosclerotic

- Atherosclerotic aortic aneurysm affecting the abdominal aorta.

- This aneurysm is complicated by a thrombus.

- Other complications include rupture and pressure on other structures.
Aortic aneurysm
Dissecting

Dissecting aortic aneurysm can result from either
1- Crack in an athermanous plaque. Or
2- Congenital medial necrosis in Marfan’s syndrome or Erdheim’s medial necrosis.
II- Cerebral aneurysm

- a. Berry aneurysm (CONGENITAL)
  - Small, multiple, saccular.
  - Occur in the circle of Willis at sites of medial weakness at the bifurcations.
  - They are the most common cause of subarachnoid hemorrhage.

- b. Mycotic aneurysm (SBE).
- c. Hypertensive microaneurysm (benign hypertension)
- d. Atheromatous
Berry aneurysm
Cerebral aneurysm
Complications of aneurysms

1) Rupture leading to fatal hemorrhage
2) Pressure on the surrounding structures.
3) Thrombosis and embolization