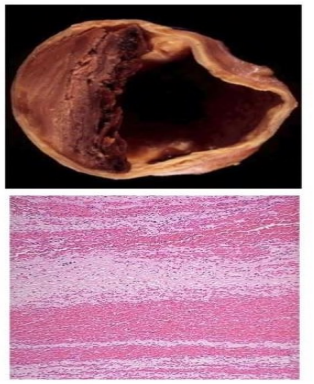
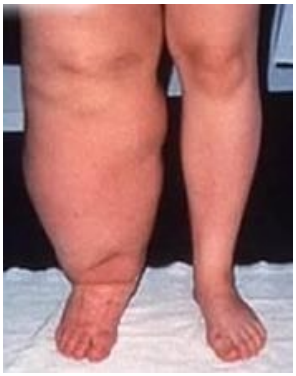
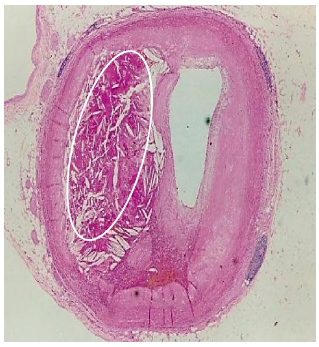
CVS Pathology lab

Done by : leen Farouq

Pictures	What are mentioned by the dr	Theory information from sheets
Lines of Zhan 	▪ Antemortem blood clot (form during life) ▪ Seen in thrombosis	▪ represent a blood clot which contains pale platelets and fibrin layers alternating with darker erythrocyte rich layers. And this alteration in the layers will give the characteristic color differences in those lines. ▪ seen both grossly and microscopically.
Lymphedema 	▪ Secondary lymphedema ▪ causes : Malignant tumors ,Surgical procedures removing lymph nodes , Post-irradiation, Fibrosis , Filariasis , that leads to inflammation of the associated Post inflammatory thrombosis and scarring	▪ more common than primary lymphedema
Atherosclerosis 	▪ hallmark of atherosclerosis is atheromatous plaque (atheroma) ▪ two components of atheroma : ✔Fibrous cap: composed of **proteins** (collagen, elastin, proteoglycans), certain types of **cells** (smooth muscle cells, macrophages, lymphocytes, foam cells) and **neovascularisation** (formation of new blood vessels for perfusion). ✔Necrotic centre: contains cholesterol crystals (LDL) with some debris, calcium and foam cells	▪ the needle-like whitish areas within the atheroma represent the necrotic centre.

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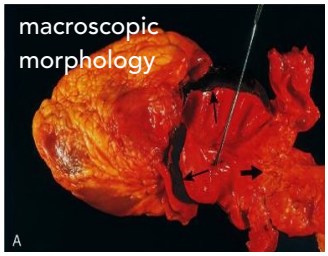
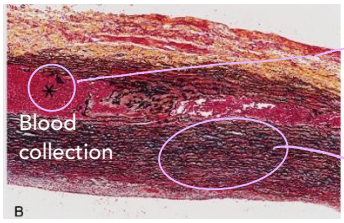
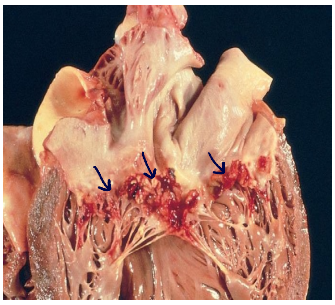
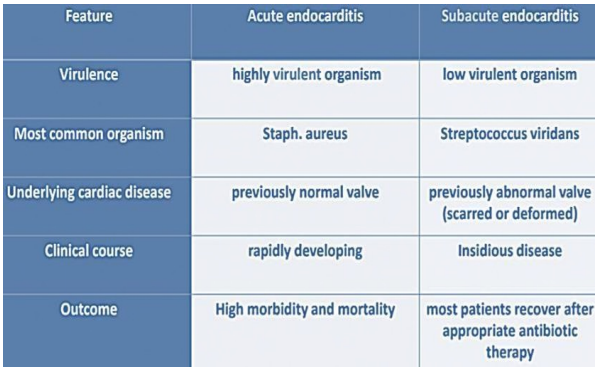
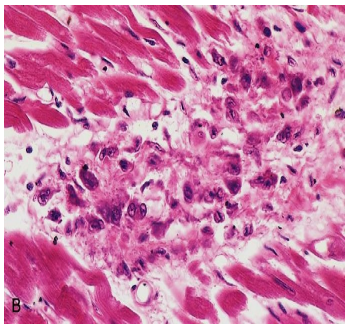
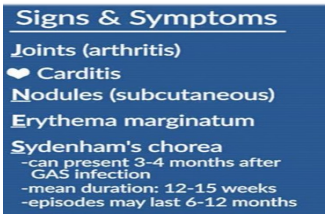
Aortic dissection  	▪ The major risk factor of aortic dissection is ***hypertension*** ▪ elastic fiber are lost in this location & blood filling the dissected portion ▪ elastic fibers in black lines inside the media	▪ <u>Complications are :</u> › massive hemorrhage › cardiac tamponade ▪ All parts of aorta can be affected ▪ Blood pressure difference between Rt & Lt arms ▪ Hypotension , shock ▪ <u>Aortic dissections types:</u> 1. Type A dissections: › More common, More dangerous › Proximal to the takeoff of major aortic branches › Involve either ascending aorta only or both ascending and descending aorta 2. type B dissections › Occur distal to the takeoff of major aortic branches › Does not involve ascending aorta › usually beginning distal to the subclavian artery
Infective endocarditis , vegetations 	▪ vegetations produced by the infection	 ▪ vegetations send pieces of the thrombus leading to the formation of emboli ▪ the most commonly affected valves are the aortic and the mitral valves. However, it depends on the exact clinical picture
Acute rheumatic fever; Aschoff bodies, 	▪ Aschoff bodies: granulomatous structures consisting of T lymphocytes, plasma cells, and activated macrophages	▪ involvement in all 3 layers of the heart (pancarditis) ▪ endocardium covers the four cardiac valves, thus, itsinvolvement might lead to the formation of valve vegetations (thrombi overlying the valves). j💜nes 

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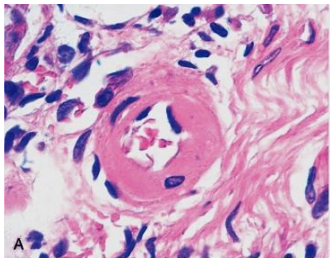
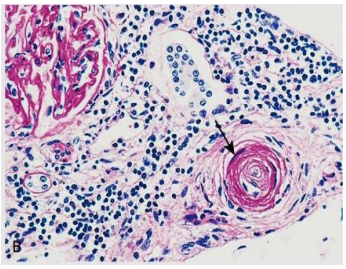
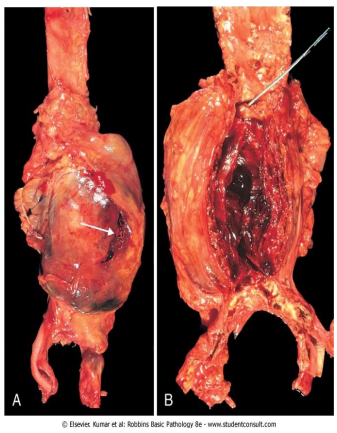
Arteriolosclerosis: hyaline 		▪ Associated with Benign hypertension ▪ called Hyaline; because of the homogeneous pink hyaline thickening of arteriolar walls. ▪ will lead to luminal narrowing due to the leakage of plasma components across injured endothelial cells into vessel walls & and increased ECM production by smooth muscle cells in response to chronic hemodynamic stress ▪ Most significant in kidneys ---> nephrosclerosis (glomerular scarring) and with time this leads to chronic renal failure. • Other causes of hyaline arteriolosclerosis (without the presence of hypertension): - 1- elderly patients (normo-tensive) 2- diabetes mellitus
Arteriolosclerosis: hyperplastic 	▪ associated with severe (malignant) hypertension ▪ The hallmark of this condition is the " Onion skin "	▪ this is a result of concentric laminated thickening of arteriolar walls, which leads to luminal narrowing ▪ multiple layering
Aneurysms  © Elsevier. Kumar et al: Robbins Basic Pathology 8e - www.studentconsult.com	▪ below renal arteries and above bifurcation of aorta into common iliac arteries ▪ A: shows a RUPTURE in the wall of the aorta ▪ B: shows a large MURAL THROMBUS in the lumen of the aorta	▪ can be saccular or fusiform ▪ atherosclerosis is the main cause of AAA; —> evidence of atherosclerotic plaques, advanced lesions of atherosclerosis and thinning of media . ▪ Symptoms of AAA: ➢ Deep abdominal pain or discomfort or back pain ➢ Pulsating feeling in the abdomen ▪ Clinical consequences of AAA: Rupture, Obstruction of downstream vessel, Embolism→mural thrombus, Abdominal mass, Compression on adjacent structures

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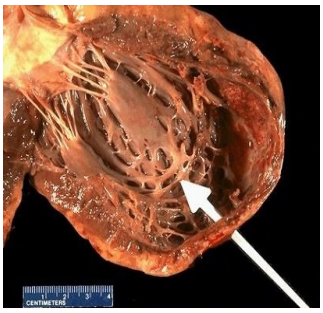
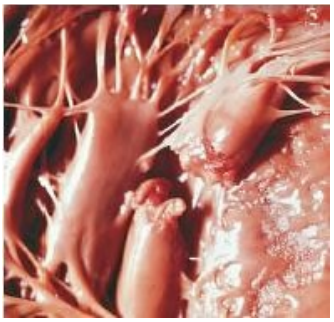
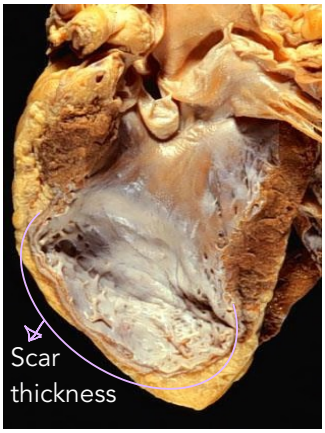
Complications of acute myocardial infarction		
Myocardial rupture 	▪ Rupture of the ventricular free wall which leads to hemopericardium and cardiac tamponade (usually fatal).	
Myocardial rupture 	▪ ***Rupture of Papillary muscles*** that surround the valves, which leads to valve abnormalities like severe regurgitation [if it happened in the mitral valve]	
Ventricular aneurysm 	▪ A late complication; needs several weeks (at least 6 weeks) to happen because it needs to have scar tissue to develop. ▪ white weak thin scar that cannot have the same contractile capacity of the viable myocardium	▪ **Potential complications of ventricular aneurysms include:** 1-Development of mural thrombus inside the affected chamber. 2-Development of arrhythmias, because this scar has abnormal conduction process inside it. 3-Heart failure.