Patho - Test 2

Week 4 - AS

Disorders of Arterial Circulation

1. Hyperlipidemia - an increase in blood cholesterol as chol. and tri. increase, so does HD & stroke

2. Atherosclerosis - plaque buildup in arteries

3. Occlusions/Obstructions - complete or partial bloackage in b.v (veins or arteries)

5 Lipoproteins

1.Chylomicrons 2. VLDL 3. IDL

4. **LDL** - too much fat --> liver saturated --> too much in blood --> excess LDL binds to endothelial cells --> macrophages bind to LDL --> m.p oxidize LDL (key contributor to AS)

5. **HDL** - "good" & synthesized by liver - transports chol back to liver from periphery

CAD RISK FACTORS

*smoking * HTN * family hx * HDL less than 40mg/dL * diabetes

Atherosclerosis

Accumulation of lipid-laden macrophages forming a lesion called plaque

Leading cause: CAD, stroke, peripheral arterial disease

RISK FACTORS AS

increasing age, male, genetic disorders of metabolism, family hx of CAD OR smoking, obesity, HTN, HYLIP, diabetes

Patho - Test 2 (cont)

Pathogensis of AS

1. Endothelial Injury - smoking, LDL, immune mechanisms & mechanical stress from HTN cause this with adhesion of monocytes and platelets

2. Migration of inflammatory cells - endothelial cells bind to monocytes and platelets that start AS lesions - monoctyes adhere to endothelium and stay in intima, transform into macrophages and engulf LDLs

3. Lipid Accumulation & SM proliferation - macrophages engulf LDL (protects but contributes to AS) & activated macrophages release toxic o2 that oxidizes LDL. Oxidized LDL ingested by macrophages result in FOAM CELLS

4.Gradual development of plaque - consists of smooth muscle cells aggregation, macrophages, ECM, lipids. Superficial fibrous cap = SMC's and dense ECM

Plaque Structure

shoulder consists of macrophages, SMC's & lymphocytes

Central core = lipid laden foam cells and fatty debris

Rupture or erosion of unstable fibrous cap can lead to hemorrhage into plaque or thrombotic occlusion in vessel lumen

STABLE thick fibrous cap, partially blocked vessels, no clot formation or emboli

UNSTABLE thin fibrous cap, completely block artery, can rupture = thrombus or embolus

Week 6 - Cerebrovascular Disease

TERMS

Tissue Perfusion: process of blood to a cap. bed in tissue "pour over or through" - blood flow

Aneurysm abnormal bulging of arterial wall, worsens over time as blood pushes against it, eventually bursting

Ischemia: low flow of blood to tissues and causes damage to target tissues (via obstruction or hemorrhage)

Embolism blockage forms clot and moves through circ.

Stroke: acute focal neurological deficit from vascular impairment of cerebral blood flow (> tissue perf & ischemia --> neurological deficits)

2 types **ISCHEMIC** (caused by thrombosis/emboli) & **HEMORRHAGIC** (subarachnoid anuerysmal hemorrhage)

Ischemic Stroke

risk factors: HTN, smoking, diabetes, carotis stenosis, sickle cell diease, hyperlipidemia, atrial fibrillation

5 stoke subtypes: 1. large artery AS disease, 2. small vessel or pen. artery, 3. cardiogenic embolism 4. cryptogenic stroke 5. unusual causes

Week 6 - Cerebrovascular Disease (cont)

Penumbra central core of dead/dying cells surrounded by ischemic band of cells called "-penumbra" HALO.

cells inside penumbra experience: impaired metabolic activity, eletrical failure, structural intg. cells maintained

Survival is dependant on return of circ.

will remain viable for several hrs due to collateral arteries supplying the zone

LARGE vessel (thrombotic) Stroke

thrombi most common cause of ischemic stroke in AS vessels (common sites: internal carotid, veterbral arteries, junctions at basilar and vertebral, arterial bifurcations)

Affects - cerebral cortex as APHASIA & neglect as VISUAL & UNILATERAL

SMALL vessel (lacunar infarct) Stroke

small infarcts located deep in brain result from occlusion of smaller penetrating branches of larger cerebral arteries

- healing lucunar ifarcts leave behind lacunae (small cavities from AS)

Affects - hemiplegia, dysarthia (weakness of hands), MRI to diagnose

TIA - "ministroke* where blood flow reverses before infarction occurs (1 hr symptoms), zone of penumbra, caused by AS, warning

Week 6 - Cerebrovascular Disease (cont)

Cardiogenic Embolic Stroke
caused by moving blood clot that travels from its origin to brain, frequently in middle cerebral artery, originate from heart, SUDDEN

Homonymous Hemianopsia - food on left side not seen

Diagnosis of Acute Stroke
CT, MRIs, catheter based conventional arteriography, sonography
Treatment GOALS: saving tissue, preventing secondary stroke, min. long-term disability --- reperfusion tech. like tPA, catheter-directed clot distruption, aug. of CPP
Post MGMT highest risk 1 week after stroke or TIA, anti-platelet agents, warfarin

HEMORRHAGIC STROKE
- often fatal - rupture of b.v - hemorrhage in brain tissue - compression in brain tissue by expanding hematoma and tissue edema in brain
most common is aneurysmal subarachnoid hemorrhage (SAH)
Risk Factors** - age, HTN, aneurysm, trauma, tumors, blood coag. disorders, drugs etc

Manifestations 1. vomiting/headache 2. contralateral hemiplegia (hemorrhage into basal ganglia) 3. Edema exert pressure = coma & death
(monro-kellie hypothosis)

Week 6 - Cerebrovascular Disease (cont)

SAH: arise from congenital defect in medial layers of involved vessels - rupture of aneurysm casues bleeding into SA space leads to increased ICP

Manifestation of SAH BEFORE: asymptomatic, history of headaches, chronic headache
AFTER: sudden headache, LOC, vomiting, blurred vision, sensory deficits, HTN, cerebral edema
Diagnosis: clinical, CT scan, vascular imaging, lumbar puncture

VASOSPASM: involves focal narrowing of cerebral arteries - decreasing neurological status due to blood loss to area, 3-10 days after rupture
treatment - vasoactive drugs, IV fluid, risk of re-bleeding, balloon dilation, meds (nimodipine)

Patho - Test 2

Week 4 - HTN
o most common health problem
o leading risk factor for CV disorders (creates AS, increases workload on heart in left ventricular hypertrophy)
o more men
BP = CO x SVR

Complications
high BP marked with progressive target organ damage (180/120)
w/ severe headache/cerebral edema
Treatment partial reduction in bp to safer level

Patho - Test 2 (cont)

Special Pops
Pregnancy - preclampsia-eclampsia
Children/adolescents - lifestyle or secondary HTN (kidney issues)
Older adults - stiffening of large arteries

Week 4 - Cardiac Conditions
CAD: Heart disease cause by impaired coronary blood flow (AS most common cause)
Pathogenesis of CAD
no symptoms until advances b/c collateral flow
lesions usually located in LAD and RCA

CAD - 2 types
1. ACS - acute plaque disruption (unstable angina to MI) & presence of ST segment elevation present to confirm, T wave inversion, abnorm. Q wave
Diagnosis of ACS troponin I & troponin T (PRIMARY - rise 3 hr post MI and last 7-10 days), myoglobin, CKMB
2. Chronic ischemic heart diease - AS or vasospatic obstruction of coronary artery (ie, stable angina)
Stable Plaque = stable angina
Unstable Plaque = pl. disruption, platelet agg, thrombus, unstable angina & MI
Pathophysiology of MI Occlusion -->contractibility stops depriving myocardial cells --> LA accumulates and fibres irritated --> angina --> lead to MI

Patho - Test 2 (cont)

Chronic Stable Angina
Angina Pectoris"sudden attack of angina due to transient myoc. ischemia
PRIMARY MANIFESTATION IS **PAIN**

MI
STEMI: ischemic death of myocardial tissue occurs when a ruptured plaque blocks a major artery completely. - ST elevation.
Unstable Angina/NSTEMI: caused by a block in a minor artery or a partial obstruction in a major artery. More severe prolonged angina
STEMI - Referfusion: Reestablish blood flow w/fibrinolytic therapy **BENEFITS** - prevent necrosis, improve mycar. perfusion (recovery called stunned)
Treatment of AMI firbinolytic therapy, PCI & CABG

Week 7 - Respiratory Conditions Part 1

PULMONARY EDEMA
cap fluit move to alveoli, hgb leaves = cyanosis, coughing, crackles, tachycardia, cool skin
treatment - non pharm: o2 and assistance with breathing --- pharm: diuretics, ACE inhibitors

Week 7 - Respiratory Conditions Part 1 (cont)

PNEUMOTHORAX
presence of air in pleural space, causes partial or full collapse
1. Spontaneous rupture of bleb on surface of lung, allows air from airways to enter pleural space, higher alveolar pressure and air flows into space, collapse
primary:healthy, belbs at top, smoking
secondary:pt with lung disease, can be life threatening
2. Traumatic caused by penetrating or non-penetrating chest injuries (fracture ribs)
3. Tension life-threatening condition where injury allows air to enter but not leave, opposite side compression, shift in mediastinum, compression in vena cava, decrease in venous return to heart and decrease CO
diagnosis - clinical and xray CT
treatment small pnuemothroaces, o2, need apsiration

HEMOTHORAX presence of blood in plueral space - all same as penuemothorax

Week 7 - Respiratory Conditions Part 1 (cont)

ASTHMA
1. Expose to allergin - mast cells release inflammatory mediators (symp. 10-20 min) 2. Infiiltration of WBCs - release of cytokins (increased mucous) 3. Bronchospasm - caused by stim. of PS receptors, mucosal edema
late phase 1. Inflammation and increased airway responsiveness (4-8 hrs after exposure) 2. Release of inflamm. mediators from mast cells (induce migration and basophil activation) 3. Epithelial injury and increase vascular permeability (edema) 4. Bronchospasm
treatment SABA or LABA, brochidilators (b2 agonists, anticholinergic agents), steroids, aerochamber more effective

Week 7 - Respiratory Conditions Part 1 (cont)

COPD (emphysema & chronic brochitis)
-chronic obstruction of lung airflow that interferes with normal breathing and not reversible
- not cureable -
emphysema: enlargement of air spaces and destruction of lung tissues, a1 deficiency
COB: obstruction of small airways, chronic irritation (smoking)
patho increased mucous cells, mucous hypersecretion, hypertrophy in glands in trachea/bronchi, imflammation, fibrosis bronchiolar wall, increase goblet cells, viral and bacterial infections
clinical fts insidious onset, cough in am, dyspnea, SOB
manifestations wheezes and crackles, tripod position, pursed lip breathing, hypoxemia, cyanosis

Emphysema Patho
increased neutrophils in alveoli secrete trypsin, and imbalance of tryspin and a1 decreases protection, elasrase triggers breakdown of elastin, which damages alveoli
smoke --> inflammation --> acti. neutrophils --> inactiv. of antiproteases --> increase elastase activity --> tissue destruction

Week 7 - Respiratory Conditions Part 1 (cont)

PINK PUFFER - usually emphysema, increases resp to maintain o2, dyspnea, lip breathing **BLUE BLOATERS** - usually bronchitis, cannot increase resp enough to maintain o2, cyanosis, cor pulmonale

Week 5 - Heart Failure (CHF)

Heart Failure any structural or functional disorder of the heart w/ low CO &/or pulmonary or systemic congestion
Common Causes: CAD, HTN, dilated cardiomyopathy, valvular HD

Heart as a pump
Preload - blood in ventricles at end of diastole right before ven. contract, blood pressure in l. vent. before contraction
Afterload - force of contracting heart muscles to eject blood, resistance in systole, **A**fterload created by **A**rteries
Contractibility - ability to contract, increases CO, ATP & Ca+

Week 5 - Heart Failure (CHF) (cont)

Systolic dysfunction decreased contracting --> decrease EF (less than 40%)
Manifestations - increase in preload --> blood accumlates in atria and pulmonary venous system --> pulmonary congestions
Diastolic dysfunction - inability for l. ven. to fill during diastole. leads to increased pressure in left atrium --> pulmonary congestion & decrease lung compliance --> CO is decreased bc decreased filling

Frank Starling Mechanism
- end result is increased SV - preload (end diastolic volume) increases - cardiac muscle fibres stretch & trigger stronger contraction - increases CV at lower HR

Manifestations of CHF
1. impaired pumping 2. decreased renal blood flow 3. sympathetic nervous system
symptoms fluid retention, dyspnea, fatigue, cyanosis, malnutrition, arrhythmias

Week 5 - Heart Failure (CHF) (cont)

Acute pulmonary edema
cap. fluid moved into alveoli
SEVERE pulmonary edema due to elevated left ventricular filling pressures *decrease o2 supply to brain, confusion, dyspnea, frothy pink sputum, crackles*
Treatment non-pharm: exercise, Na+ & water restrictions, weight mgmt
pharm: diuretics, digoxin, ACE inhibitors, beta-blockers
others - o2, cardiac re-synchronization, ventricular assist devices

Week 8 - Respiratory Conditions Part 2

Pneumonia
TYPICAL: bacterial infection, inflammation and exudation of fluid into alveoli ATYPICAL: involves alveolar septum and interstitium of lung, ourulent sputum, leukicytosis
patho 1. aspiration 2. release of bacterial endotoxin 3. inflammatory response (neutrophils, fibrinous exudate, RBCs) 4. red hepatization and consol. of lung parenchyma -- leukocyte infilration -- 5. gray hepatization and depo. of fibrin, phagocytolsis of alveoli 6. resolution of infection (macrophages engulf neutro, fibrin and bacteria

Week 8 - Respiratory Conditions Part 2 (cont)

Acute Bacterial (typical) Pneumonia
1. Pneumococcal pne. or streptococcus pne. - attaches and colonizes to mucosa, delays phagocytosis, acts as antigen
onset malaise, shaking, chills, fever
initial stage cough, watery sputum, fine crackles
progressive cough with purulent blood tinged sputum, lung pain with breathing
elderly = less likely to have temps (may only have loss of apetite or bad mental status)
2. Pneumococcal Pne. - vehicle transmission, impairs gas exhange, 2-10 days after infection, dirrhea
manifestations malaise, weakness, lethargy, fever, dry cough

atypical - lack of lung consolidation and alveolar exudate, less sputum, elevation of WBCS mycoplasma pne. common bacterial agent in children

Week 8 - Respiratory Conditions Part 2 (cont)

TB - slender rod-shaped bacilli that do not form spores
myobacterium waxy cell wall and responsible for: slow growth - ability to trigger immune response - rest. for destruction/- antibiotics/lab stains
patho inhaled droplet pass down bronchial tree and land in alveoli, bacilli are phag. by alveolar macro but resist killing, intitiate cell mediated immune response that contains infection, bacilli multiply, infect macrophaes, degrade myobacteria and present antigens on helper t lymphs.
INITIAL TB INFECTION - macro begin cell mediated response, results in granulomatous lesion (GHON FOCUS) containing macro, t cells and inactive TB bacteria
patho cont t helper cells stim. macro to increase and kill mycobacteria, when released they damage lung tissue, cytotoxic t cells and macro constitute the cell mediate response that takes 3-6 weeks to become effective

Ghon Focus: area where organisms ends up in lungs turn gray granuloma (typically in upper seg. of lower lobes and lower seg. of upper lobes
Ghon Complex: undergoes soft necrosis, caseous granuloma form along lymph channels, later shrinks, becomes fibrous and calcified, visible with chest xray

Week 8 - Respiratory Conditions Part 2 (cont)

primary TB - previous unexposed pt (inhale) - insidious symptoms: fever, weight loss, fatigue, night sweats abrupt onset: high fever, pleuritis and lympthadenitis

secondary TB: reinfection from inhaled droplets or reactivation with dry cough, low grade fever, productive blood tinged sputum

treatment of TB - eliminating bacilli in infected pt, preventing spread, antibioltics (INH and rifampin)

Week 5 - Peripheral Vascular Disease

PAD

systemic AS distal to aorta w/ claudication, atrophic changes/t-hinning of skin, weak pedal pulse, ischemia pain, gangrene, ulcers on toes, ankles

Diagnostics

- inspection of limbs, palp, pulses, ankle-brachial index, US, MRI, CT, angiography

Treatment

- protection of area, walking to point of claudication, avoidance of injury, antiplatelets, surgery

Week 5 - Peripheral Vascular Disease (cont)

PVD

- manifested by venous HTN, causes reflux in veins, prolonged standing increases pressure and dilated vessel wall

Manifestations - tissue congestion, edema, necrosis of fat deposits, brown pigmentation, advanced (stasis dermatisis, ulcers ankles uneven)

Treatments - compression therapy, dressings, bandages

DVT or thrombophlebitis

- presences of thrombus in vein w/ imflammatory response (calf) risk factors: VIRCHOWS TRIAD (blood stasis, hyperactivity of blood coag. vessel wall injury) & increase risk with bad cardiac function

- usually asymptomatic (if not, pain, swelling etc) U/S, tx is prevention, warfarin, IVC filter, complications - pulmonary embolism