Class 3 Notes for BioChem Labwork

Lipids

1. Diet and liver
2. Cholesterol – steroids, cell membrane, signaling molecule, bile salts
3. Triglycerides – primary energy storage molecule in fat cell (adipocytes) and muscle cells
4. (Phospholipids – makes most cell membrane, glycolipids – signaling molecules)
5. Lipoproteins – complex molecule that contains proteins and transport triglycerides and cholesterol in blood.
    a. Chylomicrons – transports dietary TGs and cholesterol from intestine to liver
    b. VLDL – transports TGs and some cholesterol from liver to tissues
    c. LDL – cholesterol – transports and acts as donor of cholesterol. LDL can deposit cholesterol in blood vessel wall
    d. HDL – cholesterol receiver/remover from circulation and blood vessel walls and delivers it to the liver.
    e. IDL

Lipid panel

1. Total cholesterol – measure cholesterol in all the lipoproteins particles
    a. Less than 200 mg/dL
    b. 200-239 mg/d: borderline
    c. 240-279 mg/dL: high
    d. >280 mg/dL: very high
2. LDL – "bad cholesterol" – deposits excess cholesterol in blood vessel wall and contribute to development of atherosclerosis → CAD (coronary artery disease) and stroke
    a. 75% of cholesterol is bound to LDL
    b. Optimal: < 100 mg/dL
    c. 100-129 mg/dL near optimal
    d. 130-159 mg/dL borderline
    e. 160-189 mg/dL high
    f. >190 mg/dL very high

3. HDL – “good cholesterol” – removes excess cholesterol and carries it to liver.
    a. Male 40 and women >50
    b. <35 mg/dL = increased risk for CAD (CHD/IHD)
4. Triglycerides
    a. Total triglycerides – measure TGs in all lipoproteins
    b. VLDL
    c. Shows ability to store fat
    d. Identify the risk of CAD(CHD)

High total cholesterol, high LDL, low HDL, high triglycerides

Patient has low HDL – increased risk of atherosclerosis – CAD

5. Other test
    a. Apoproteins (apolipoproteins)– Apo A, Apo B, Apo C, Apo E (Part of LDL, HDL)
        - In lipid transport, apolipoproteins function as structural components of lipoprotein particles, ligands for cell-surface receptors and lipid transport proteins, and cofactors for enzymes (apolipoprotein C-II for lipoprotein lipase and apolipoprotein A-I (apoA1) for lecithin-cholesterol acyltransferase)
        - Apo B-100 (VLDL, LDL)
        - Apo A-I, Apo A-II (HDL)
        - Elevated ApoB and decreased ApoA1 are associated with increased risk of cardiovascular disease.
        - ApoC1 level increases in neuropathic pain and fibromyalgia patients which suggest it plays an important role in occurrence of these conditions.
        - ApoC3 is a risk factor of cardiovascular disease. Accumulation of plasma TRLs caused by elevated apoC-III leading to hypertriglyceridaemia.
        - ApoD level increases in nervous system with many neurologic disorders inclusive of Alzheimer's disease, schizophrenia, and stroke.
        - ApoE has been implicated in dementia and Alzheimer's disease.

- Apo(a) is a component of lipoprotein(a) (Lp(a)) and elevated plasma Lp(a) level is a heritable, independent, and possibly causal risk factor for Atherosclerotic Cardiovascular Disease (ASCVD). The cholesterol-rich apoB-containing lipoproteins are also participate in the pathogenesis of ASCVD

b. Enzymes
   - LCAT – esterifies free cholesterol for transport within HDL

c. Cholesterol to HDL ratio
   - Your total-cholesterol-to-HDL ratio can be figured out by dividing your total cholesterol number by your HDL cholesterol number
   - Most healthcare providers want the ratio to be below 5:1.
   - A ratio below 3.5:1 is considered very good.

6. Dyslipidemias
   a. Primary – gene mutation – overproduction of LDL, or underproduction of HDL
   b. Secondary
      - The most important secondary cause is – sedentary lifestyle with excessive dietary intake of total calories, saturation fat, cholesterol, and trans fats
      - Diabetes – high levels of LDL and low levels of HDL
      - Alcohol overuse
      - Chronic kidney disease
      - Hypothyroidism
      - Drugs: thiazides, beta-blocker, glucocorticoids
      - Secondary cause of low HDL – cigarette smoking, anabolic steroids, HIV infection, nephrotic syndrome
7. Dyslipidemias are mostly silent (no signs or symptoms) until causes coronary artery disease (CAD), strokes, peripheral artery disease (PAD)
   a. High triglycerides – can cause acute pancreatitis, hepatosplenomegaly, paresthesia, dyspnea, confusion
   b. LDL – yellow plaques on the eyelids – Xanthelasma (familial hypercholesterolemia. Xanthomas at the Achilles tendon, elbow tendons, knee tendons

Cardiac panel (profile) = cTnI, CK/CK-MB, Myoglobin

1. Cardiac troponin, Cardiac Troponin I: cTnI
   a. Highest known sensitivity. Best biomarker for finding heart attack (Myocardial infarction)
   b. Increased = Myocardial infarction (sensitivity 50% at 4 hours, 97% at 6 hours; specificity 95%), cardiac trauma, cardiac surgery, myocardial damage following PTCA, and other cardiac interventions, nonischemic dilated cardiomyopathy, prolonged supraventricular tachycardia, acute dissection of the ascending aorta. Slight elevations noted in patients with recent aggravated unstable angina, muscular disorders, CNS disorders, HIV infection, chronic renal failure, cirrhosis, sepsis, lung diseases, and endocrine disorders.
2. Creatinine kinase (CK)
   a. Elevated CK-MB = Myocardial infarction, cardiac trauma, certain muscular dystrophies, and polymyositis. Slight persistent elevation reported in a few patients on hemodialysis. CK-MB - rises 4-6 hours after MI, back to norm in one to two days.
   b. Elevated CK = Myocardial infarction (MI), myocarditis, muscle trauma, rhabdomyolysis, muscular dystrophy, polymyositis, severe muscular exertion, malignant hyperthermia, hypothyroidism, cerebral infarction, surgery, Reye syndrome, tetanus, generalized convulsions, alcoholism, IM injections, DC countershock. Drugs: clofibrate, HMG-CoA reductase inhibitors. During an MI, serum CK level rises rapidly (within 3–5 hours); elevation persists for 2–3 days post-MI. Total CK is not specific enough for use in diagnosis of MI, but a normal total CK has a high negative predictive value
3. Myoglobin – can be used in addition to troponin. Myoglobin is the first enzyme that increases, but it returns to normal levels within the first 24 hours after the onset of symptoms.

Blood gas panels

1. **pH 7.4 (7.35-7.45)** – measure acid-base balance in the blood
   a. pH goes down = acidosis
   b. pH goes up = alkalosis
2. Partial pressure of oxygen (PaO2): 75 to 100 mmHg
3. **Partial pressure of carbon dioxide = PaCO2 (Respiratory): 34-45 mmHg**
4. **Bicarbonate = HCO3 (Metabolic): 22-26 mEq/L**
5. Oxygen saturation = O2 Sat (94-100%)

Reasons to orders ABG's - Lung Failure, Kidney Failure, Shock, Trauma, Uncontrolled diabetes, Asthma, Chronic Obstructive Pulmonary Disease (COPD), Hemorrhage, Drug Overdose, Metabolic Disease, Chemical Poisoning

Metabolic acidosis, Metabolic alkalosis, Respiratory acidosis, Respiratory alkalosis

1. Metabolic acidosis – primary reduction in HCO3
   a. GI loss of HCO3
   b. Increased production of acid and acid ingestion
   c. Ketoacidosis – Diabetes, fasting
   d. Lactic acidosis – CO poisoning, iron
   e. Renal failure
2. Metabolic alkalosis – primary increase in bicarbonate (HCO3)
   a. Less acid – decreased hydrogen
   b. Vomiting – loss of hydrogen
   c. Increased bicarbonate
   d. Hypokalemia (low potassium)
   e. Diuretics
3. Respiratory alkalosis – primary decrease in PaCO2
   a. pH increased, PaCO2 decreased, normal HCO3
   b. Hyperventilation
   c. Pulmonary disorders
4. Compensated respiratory alkalosis
   a. pH increased, PaCO2 decreased, HCO3 decreased

5. Respiratory acidosis – primary increase in PaCO2
    a. pH decreased, PaCO2 increased, normal HCO3
    b. COPD
    c. Respiratory failure
6. Compensated respiratory acidosis
    a. pH decreased, PaCO2 increased, HCO3 increased

ABG: pH 7.34, Pa CO2 – 51 mmHg (high)

1. What is present in this patient – acidosis
2. Elevated Pa CO2 – points towards primary respiratory acidosis

ABG pH 7.46. Pa CO2 – 30 mmHg (low), PaO2 normal, HCO3 normal, Sat O2 normal

1. What is present in this patient – alkalosis
2. Low Pa CO2 – points towards respiratory alkalosis

Metabolic panel – basic or comprehensive. Basic metabolic panel is 7 to 8 tests and Comprehensive metabolic panel is 14 tests (SMA12+2)

BMP = sodium (Na+), potassium (K+), chloride (Cl−), bicarbonate (HCO3−) or CO2, blood urea nitrogen (BUN), creatinine, glucose

CMP, 14 tests (SMA12+2):

1. Total protein – how well kidney and liver are working
2. Albumin
3. Glucose
    a. Diabetes, hypoglycemia
    b. (Can order more tests – fasting blood sugar, oral glucose tolerance test, Hemoglobin A1c)
4. BUN (blood urea nitrogen)
    a. Increased in: Renal failure (acute or chronic), urinary tract obstruction, dehydration, shock, burns, CHF, GI bleeding. Nephrotoxic drugs (eg, gentamicin). Diet high in protein, heart failure
    b. Decreased in: Hepatic failure, nephrotic syndrome, cachexia (low-protein and high-carbohydrate diets).

5. Creatinine
    a. High – kidney disease, muscle damage, dehydration, pregnancy
    b. Low – low muscle mass
6. Bilirubin
7. ALT
8. AST
9. ALP

Electrolyte levels and the balance among them are tightly regulated by the body. Both individual values and ratios among the values are significant; abnormalities among either can indicate problems such as an electrolyte disturbance, acid-base imbalance, or kidney dysfunction.

10. Sodium
    a. Hyponatremia – decreased intake, increased body water, hyperglycemia, CHF (Congestive heart failure), renal failure, cirrhosis
    b. Hypernatremia -increase intake, sea water, diabetes insipidus, sweating
11. Potassium
    a. Hypokalemia – starvation, anorexia, diuretics, burns, vomiting, diarrhea. Low potassium intake, prolonged vomiting or diarrhea, renal tubular acidosis types I and II, hyperaldosteronism, Cushing syndrome, osmotic diuresis (eg, hyperglycemia), alkalosis, trauma (transient), subarachnoid hemorrhage
    b. Hyperkalemia – renal failure, infection, hypoaldosteronism, dehydration, acidosis, hemolysis, severe tissue damage, rhabdomyolysis, Addison disease, renal tubular acidosis type IV (hyporeninemic hypoaldosteronism), exercise (transient) and drugs
12. Calcium
    a. Hypocalcemia – vitamin D def, pancreatitis, hypoparathyroidism, alkalosis, malabsorption, medication (aspirin, laxatives, diuretics, estrogens, oral contraceptives)
    b. Hypercalcemia – vitamin D intoxication, metastatic bone tumors, prolonged immobilization, lymphoma, hyperthyroidism, hyperparathyroidism
13. Chloride

a. Hyperchloremia. It may be due to: Addison disease, Carbonic anhydrase inhibitors (used to treat glaucoma), Diarrhea, Ethylene glycol poisoning, Ketoacidosis, Kidney disease, Lactic acidosis, Metabolic acidosis, Methanol poisoning, Renal tubular acidosis, Respiratory alkalosis (compensated), Salicylate toxicity (such as aspirin overdose)
b. Hypochloremia. It may be due to: Bartter syndrome, Burns, Congestive heart failure, Cushing syndrome, Dehydration, Excessive sweating, Hyperaldosteronism, Metabolic alkalosis, Respiratory acidosis (compensated), Syndrome of inappropriate diuretic hormone secretion (SIADH), Vomiting

14. Carbon dioxide
    a. Increased in: Respiratory acidosis: decreased alveolar ventilation (eg, COPD, respiratory depressants), neuromuscular diseases (eg, myasthenia gravis).
    b. Decreased in: Respiratory alkalosis: hyperventilation (eg, anxiety), sepsis, liver disease, fever, early salicylate poisoning, and excessive artificial ventilation.