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The Crux

BIMONTHLY FORUM FOR THE LABORATARIANS

CONTENTS

Editorial

One of the most important life style disorders is related to our diet, level of physical activity and the status of cardiac blood vessels (mainly coronary arteries). The high levels of cholesterol (mainly LDLc) that get precipitated within the coronary and other arteries as atherosclerosis lead to narrowing of the vessels with the consequent reduction in the level of blood flow within them leading to ischaemia and eventually myocardial infarction. Larger infarctions that seriously hamper cardiac functioning lead to cardiac arrest and eventually prove fatal. Many medications are available these days to lower blood lipid levels that can prevent or at least delay the onset of atherosclerosis and IHD. Before one starts with any of the lipid lowering molecules, one must get a lipidogram done. What level of lipids is good for a person cannot be accurately ascertained. Many individuals with very much normal lipid levels also suffer from IHD. What is dyslipidemia then? Well, dyslipidemia; term is applied to lipid levels for which treatment has proven beneficial. Proof of benefit is strongest for lowering elevated low-density lipoprotein (LDL) levels. I request you to flip this page and turn over and go through the **DISEASE DIAGNOSIS** segment of this issue that amply discusses Dyslipidemias (both hyper and hypo).

INTERPRETATION portion outlines a very commonly used leucocytic marker called as CD marker (Cluster Of Differentiation). The **cluster of differentiation** (**cluster of designation**) (often abbreviated as **CD**) is a protocol used for the identification and investigation of cell surface molecules present on leukocytes. CD molecules can act in numerous ways, often acting as receptors or ligands (the molecule that activates a receptor) important to the cell. CD4 and CD8 are important in HIV/AIDS too. The article simply defines their utility vis-à-vis various leucocytes.

TROUBLE SHOOTING section explains Infection control tips on hand washing. What appears to be a routine simple exercise must be performed to perfection in order to be of utility. Also given are a list of Universal Standard Precautions (USP). If we have to work for others, first we have to be healthy. USP is followed by a small protocol on “How to handle a blood spill”. It paves way to precautions while handling sharps and needles. Waste management, venipuncture, fumigation and managing exposure to potentially infective fluids are not forgotten at all. All in all “MOST IMPORTANT ASPECT IN THE LIFE OF LABORATARIANS” is protection of self as well as others we serve!

BOUQUET is there in all its hues and colours. Take a peep inside.

Crux has completed 6 years of its existence. Trust it has been a fruitful exercise. Here’s wishing happy reading to all our readers.

May the coming NEW YEAR bring with it PLENTY OF PEACE, PLEASURES AND PROSPERITY to our universal TULIP family.

ISO 9001

Tulip Group

DISEASE DIAGNOSIS

DYSLIPIDEMIA

Dyslipidemia is elevation of plasma cholesterol, triglycerides (TGs), or both, or a low high density lipoprotein level that contributes to the development of atherosclerosis. Causes may be primary (genetic) or secondary. Diagnosis is by measuring plasma levels of total cholesterol, TGs, and individual lipoproteins. Treatment is dietary changes, exercise, and lipid-lowering drugs.

There is no natural cutoff between normal and abnormal lipid levels because lipid measurements are continuous. A linear relation probably exists between lipid levels and cardiovascular risk, so many people with "normal" cholesterol levels benefit from achieving still lower levels. Consequently, there are no numeric definitions of dyslipidemia; the term is applied to lipid levels for which treatment has proven beneficial. Proof of benefit is strongest for lowering elevated low density lipoprotein (LDL) levels. In the overall population, evidence is less strong for a benefit from lowering elevated TG and increasing low high density lipoprotein (HDL) levels, in part because elevated TG and low HDL levels are more predictive of cardiovascular risk in women than in men.

HDL levels do not always predict cardiovascular risk. For example, high HDL levels caused by some genetic disorders may not protect against cardiovascular disorders, and low HDL levels caused by some genetic disorders may not increase the risk of cardiovascular disorders. Although HDL levels predict cardiovascular risk in the overall population, the increased risk may be caused by other factors, such as accompanying lipid and metabolic abnormalities, rather than the HDL level itself.

Classification

Dyslipidemias were traditionally classified by patterns of elevation in lipids and lipoproteins. A more practical system categorizes dyslipidemias as primary or secondary and characterizes them by increases in cholesterol only (pure or isolated hypercholesterolemia), increases in TGs only (pure or isolated hypertriglyceridemia), or increases in both cholesterol and TGs (mixed or combined hyperlipidemias). This system does not take into account specific lipoprotein abnormalities (eg, low HDL or high LDL) that may contribute to disease despite normal cholesterol and TG levels.

Lipoprotein Patterns (Fredrickson Phenotypes)

Phenotype	Elevated Lipoprotein (s)	Elevated Lipids
I	Chylomicrons	TGs
IIa	LDL	Cholesterol
IIb	LDL and VLDL	TGs and cholesterol
III	VLDL and chylomicron remnants	TGs and cholesterol
IV	VLDL	TGs
V	Chylomicrons and VLDL	TGs and cholesterol

LDL = low density lipoprotein; TGs = triglycerides; VLDL = very low density lipoprotein.

Etiology

Primary (genetic) causes and secondary (lifestyle and other) causes contribute to dyslipidemias in varying degrees. For example, in familial combined hyperlipidemia, expression may occur only in the presence of significant secondary causes.

Primary causes: Primary causes are single or multiple gene mutations that result in either overproduction or defective clearance of TG and LDL cholesterol, or in underproduction or excessive clearance of HDL. Primary disorders, the most common cause of dyslipidemia in children, do not cause a large percentage of cases in adults. The names of many reflect an old nomenclature in which lipoproteins were detected and distinguished by how they separated into α (HDL) and β (LDL) bands on electrophoretic gels.

Secondary causes: Secondary causes contribute to most cases of dyslipidemia in adults. The most important secondary cause in developed countries is a sedentary lifestyle with excessive dietary intake of saturated fat, cholesterol, and trans fats. Trans fats are polyunsaturated or monounsaturated fatty acids to which hydrogen atoms have been added; they are commonly used in many processed foods and are as atherogenic as saturated fat. Other common secondary causes include diabetes mellitus, alcohol overuse, chronic kidney disease, hypothyroidism, primary biliary cirrhosis and other cholestatic liver diseases, and drugs, such as thiazides, β-blockers, retinoids, highly active antiretroviral agents, estrogen and progestins, and glucocorticoids.

Diabetes is an especially significant secondary cause because patients tend to have an atherogenic combination of high TGs; high small, dense LDL fractions; and low HDL (diabetic dyslipidemia, hypertriglyceridemic hyperapo B). Patients with type 2 diabetes are especially at risk. The combination may be a consequence of obesity, poor control of diabetes, or both, which may increase circulating FFAs, leading to increased hepatic VLDL production. TG-rich VLDL then transfers TG and cholesterol to LDL and HDL, promoting formation of TG-rich, small, dense LDL and clearance of TG-rich HDL. Diabetic dyslipidemia is often exacerbated by the increased caloric intake and physical inactivity that characterize the lifestyles of some patients with type 2 diabetes. Women with diabetes may be at special risk for cardiac disease from this form.

Symptoms and Signs

Dyslipidemia itself usually causes no symptoms but can lead to symptomatic vascular disease, including coronary artery disease (CAD) and peripheral arterial disease. High levels of TGs (> 1000 mg/dL [> 11.3 mmol/L]) can cause acute pancreatitis. High levels of LDL can cause eyelid xanthelasmas; arcus corneae; and tendinous xanthomas at the Achilles, elbow, and knee tendons and over metacarpophalangeal joints. Patients with the homozygous form of familial hypercholesterolemia may have the above findings plus planar or cutaneous xanthomas. Patients with severe elevations of TGs can have eruptive xanthomas over the trunk, back, elbows, buttocks, knees, hands, and feet. Patients with the rare dysbetalipoproteinemia can have palmar and tuberous xanthomas.

Severe hypertriglyceridemia (> 2000 mg/dL [> 22.6 mmol/L]) can give retinal arteries and veins a creamy white appearance (lipemia retinalis). Extremely high lipid levels also give a lactescent (milky) appearance to blood plasma. Symptoms can include paresthesias, dypsnea, and confusion.

Diagnosis

- Serum lipid profile (measured total cholesterol, TG, and HDL-cholesterol and calculated LDL-cholesterol and VLDL)

Dyslipidemia is suspected in patients with characteristic physical

findings or complications of dyslipidemia (eg, atherosclerotic disease). Primary lipid disorders are suspected when patients have physical signs of dyslipidemia, onset of premature atherosclerotic disease (at < 60 yr), a family history of atherosclerotic disease, or serum cholesterol > 240 mg/dL (> 6.2 mmol/L). Dyslipidemia is diagnosed by measuring serum lipids. Routine measurements (lipid profile) include total cholesterol (TC), TGs, HDL-cholesterol, and LDL-cholesterol.

Lipid profile measurement: TC, TGs, and HDL-cholesterol are measured directly; TC and TG values reflect cholesterol and TGs in all circulating lipoproteins, including chylomicrons, VLDL, IDL, LDL, and HDL. TC values vary by 10% and TGs by up to 25% day-to-day even in the absence of a disorder. TC and HDL-cholesterol can be measured in the nonfasting state, but most patients should have all lipids measured while fasting for maximum accuracy and consistency.

Testing should be postponed until after resolution of acute illness, because TGs increase and cholesterol levels decrease in inflammatory states. Lipid profiles can vary for about 30 days after an acute MI; however, results obtained within 24 h after MI are usually reliable enough to guide initial lipid-lowering therapy.

LDL-cholesterol values are most often calculated as the amount of cholesterol not contained in HDL and VLDL. VLDL is estimated by TG ÷ 5 because the cholesterol concentration in VLDL particles is usually 1/5 of the total lipid in the particle. Thus, LDL-cholesterol = TC - [HDL-cholesterol + (TGs ÷ 5)] (Friedewald formula). This calculation is valid only when TGs are < 400 mg/dL and patients are fasting, because eating increases TGs. The calculated LDL-cholesterol value incorporates measures of all non-HDL, nonchylomicron cholesterol, including that in IDL and Lp(a). LDL can also be measured directly using plasma ultracentrifugation, which separates chylomicrons and VLDL fractions from HDL and LDL, and by an immunoassay method. Direct measurement may be useful in some patients with elevated TGs, but these direct measurements are not routinely necessary. The role of apo B testing is under study because values reflect al non-HDL cholesterol (in VLDL, VLDL remnants, IDL, and LDL) and may be more predictive of CAD risk than LDL alone.

Other tests: Patients with premature atherosclerotic cardiovascular disease, cardiovascular disease with normal or near-normal lipid levels, or high LDL levels refractory to drug therapy should probably have Lp(a) levels measured. Lp(a) levels may also be directly measured in patients with borderline high LDL-cholesterol levels to determine whether drug therapy is warranted. C-reactive protein and homocysteine measurement may be considered in the same populations.

Secondary causes: Tests for secondary causes of dyslipidemia—including measurements of fasting glucose, liver enzymes, creatinine, thyroid stimulating hormone (TSH), and urinary protein—should be done in most patients with newly diagnosed dyslipidemia and when a component of the lipid profile has inexplicably changed for the worse.

Screening: A fasting lipid profile (TC, TGs, HDL-cholesterol, and calculated LDL-cholesterol) should be obtained in all adults ⩾ 20 yr and should be repeated every 5 yr. Lipid measurement should be accompanied by assessment of other cardiovascular risk factors, defined as:

- Diabetes mellitus
- Cigarette use
- Hypertension
- Family history of CAD in a male 1st-degree relative before age 55 or a female 1st-degree relative before age 65.

A definite age after which patients no longer require screening has not been established, but evidence supports screening of patients into their 80s, especially in the presence of atherosclerotic cardiovascular disease.

Indications for screening patients < 20 yr are atherosclerotic risk factors, such as diabetes, hypertension, cigarette smoking, and obesity; premature CAD in a parent, grandparent, or sibling; or a cholesterol level > 240 mg/dL (> 6.2 mmol/L) or known dyslipidemia in a parent. If information on relatives is unavailable, as in the case of adopted children, screening is at the discretion of the health care practitioner.

Patients with an extensive family history of heart disease should also be screened by measuring Lp(a) levels. A definite age after which patients no longer require screening has not been established, but evidence supports screening of patients into their 80s, especially in the presence of atherosclerotic cardiovascular disease.

Patients with an extensive family history of heart disease should also be screened by measuring Lp(a) levels.

Treatment

- Risk assessment by explicit criteria
- Lifestyle changes (eg, exercise, dietary modification)

For high LDL-cholesterol, statins, sometimes bile acid sequestrants, ezetimibe For high TG or low HDL-cholesterol, niacin, fibrates, and sometimes other measures.

Adult Treatment - Approach to Dyslipidemias

1. Measure fasting lipoproteins (in mg/dL):

TC (mmol/L)	
< 200 (<5.17)	Desirable
200–239 (5.17–6.18)	Borderline high
⩾ 240 (⩾6.20)	High

LDL-cholesterol	
< 100 (<2.58)	Optimal
100–129 (2.58–3.33)	Near optimal/above optimal
130–159 (3.36–4.11)	Borderline high
160–189 (4.13–4.88)	High
⩾ 190 (⩾4.91)	Very high

HDL-cholesterol	
< 40 (< 1.03)	Low
⩾ 60 (⩾ 1.55)	High

TG	
< 150 (< 1.695)	Desirable
150–199 (1.695–2.249)	Borderline high
200–499 (2.26–5.639)	High
⩾ 500 (⩾5.65)	Very high

2. Identify CAD or CAD equivalents

CAD equivalents
Other atherosclerotic disease:
Peripheral arterial disease
Abdominal aortic aneurysm
Symptomatic carotid artery disease
Diabetes mellitus
Additional risk factors that confer 10-yr risk of MI or CAD death > 20%

3. Identify major CAD risk factors

Cigarette smoking
Hypertension (BP ⩾140/90 *or* on antihypertensive drug)
Low HDL (⩽40 mg/dL [1.03 mmol/L])
Family history of premature CAD (CAD in male 1st-degree relative < 55 or in female 1st-degree relative < 65)
Age (men ⩾45, women ⩾55)

4. If ⩾ 2 major risk factors are present without CAD or CAD equivalent, assess 10-yr risk of MI or CAD death using Framingham risk tables

CAD = coronary artery disease; HDL = high density lipoprotein; LDL = low density lipoprotein; TC = total cholesterol; TG = triglyceride.

Guidelines for Treatment of Hyperlipidemia

Risk Category	Begin Lifestyle Changes If	Consider Drug Therapy If	LDL Goal
High			
CAD or CAD equivalents (10-yr risk > 20%)	LDL ⩾100 mg/dL (⩾ 2.58 mmol/L)	LDL ⩾100 mg/dL (⩾2.58 mmol/L) Drugs optional if LDL < 100 mg/dL [< 2.58 mmol/L])	< 100 mg/dL < 70 mg/dL optional
Moderate high			
⩾ 2 risk factors with 10-yr risk 10 to 20%*	LDL ⩾130 mg/dL (⩾3.36 mmol/L)	LDL ⩾130 mg/dL (⩾3.36 mmol/L)	< 130 mg/dL < 100 mg/dL optional
Moderate			
⩾ 2 risk factors with 10-yr risk < 10%*	LDL ⩾130 mg/dL (⩾3.36 mmol/L)	LDL ⩾160 mg/dL (⩾4.13 mmol/L)	< 130 mg/dL < 100 mg/dL optional
Lower			
0–1 risk factor	LDL ⩾160 mg/dL (⩾4.13 mmol/L)	LDL ⩾190 mg/dL (⩾4.91 mmol/L) Drug optional if LDL 160–189 mg/dL [4.13–4.88 mmol/L])	< 160 mg/dL

CAD = coronary artery disease; LDL = low density lipoprotein;

HYPOLIPIDEMIA

Hypolipidemia is a decrease in plasma lipoprotein caused by primary (genetic) or secondary factors. It is usually asymptomatic and diagnosed incidentally on routine lipid screening. Treatment of secondary hypolipidemia involves treating underlying disorders. Treatment of primary hypolipidemia is often unnecessary, but patients with some genetic disorders require high-dose vitamin E and dietary supplementation of fats and other fat-soluble vitamins.

Etiology

Hypolipidemia is defined as a total cholesterol (TC) < 120 mg/dL (< 3.1 mmol/L) or low density lipoprotein cholesterol (LDL-cholesterol) < 50 mg/dL (< 0.13 mmol/L). Secondary causes are far more common than primary causes and include all of the following:

- Hyperthyroidism
- Chronic infections and other inflammatory states
- Hematologic and other cancers
- Undernutrition (including that accompanying chronic alcohol use)
- Malabsorption

The unexpected finding of low cholesterol or low LDL-cholesterol in a patient not taking a lipid-lowering drug should prompt a diagnostic evaluation, including measurements of AST, ALT, and thyroid-stimulating hormone; a negative evaluation suggests a possible primary cause.

There are 3 primary disorders in which single or multiple genetic mutations result in underproduction or increased clearance of LDL.

Abetalipoproteinemia (Bassen-Kornzweig syndrome): This is an autosomal recessive condition caused by mutations in the gene for microsomal triglyceride (TG) transfer protein, a protein critical to chylomicron and very low density lipoprotein (VLDL) formation. Dietary fat cannot be absorbed, and lipoproteins in both metabolic pathways are virtually absent from serum; TC is typically < 45 mg/dL (< 1.16 mmol/L), TGs are < 20 mg/dL (< 0.23 mmol/L), and LDL are undetectable. The condition is often first noticed in infants with fat malabsorption, steatorrhea, and failure to thrive. Mental retardation may result. Because vitamin E is distributed to peripheral tissues via VLDL and LDL, most affected people eventually develop severe vitamin E deficiency. Symptoms and signs include visual changes from slow retinal degeneration, sensory neuropathy posterior column signs, and cerebellar signs of dysmetria, ataxia, and spasticity, which can eventually lead to death. RBC acanthocytosis is a distinguishing feature on blood smear.

Diagnosis is made by the absence of apoprotein B (apo B) in plasma; intestinal biopsies show lack of microsomal transfer protein. Treatment is with high doses (100 to 300 mg/kg once/day) of vitamin E with supplementation of dietary fat and other fat-soluble vitamins. The prognosis is poor.

Hypobetalipoproteinemia: Hypobetalipoproteinemia is an autosomal dominant or codominant condition caused by mutations in the gene coding for apo B. Heterozygous patients have truncated apo B, leading to rapid LDL clearance. Heterozygous patients manifest no signs or symptoms except for TC < 120 mg/dL and LDL-cholesterol < 80 mg/dL. TGs are normal. Homozygous patients have either shorter truncations, leading to lower lipid levels (TC < 80 mg/dL, LDL-cholesterol < 20 mg/dL), or absent apo B synthesis, leading to symptoms and signs of abetalipoproteinemia. Diagnosis is by finding low levels of LDL-cholesterol and apo B; hypobetalipoproteinemia and abetalipoproteinemia are distinguished from one another by family history. People who are heterozygous and people who are homozygous with low but detectable LDL-cholesterol require no treatment. Treatment of people who are homozygous with no LDL is the same as for abetalipoproteinemia.

Chylomicron retention disease: Chylomicron retention disease is a very rare autosomal recessive condition caused by an unknown mutation leading to deficient apo B secretion from enterocytes. Chylomicron synthesis is absent, but VLDL synthesis remains intact. Affected infants have fat malabsorption, steatorrhea, and failure to thrive and may develop neurologic disorders similar to those in abetalipoproteinemia.

Diagnosis is by intestinal biopsy of patients with low cholesterol levels and absence of postprandial chylomicrons. Treatment is supplementation of fat and fat-soluble vitamins.

TROUBLESHOOTING

STERILITY (INFECTION CONTROL) MAINTENANCE IN RELATION TO DIAGNOSTIC LABORATORIES

HAND WASHING

Infection control tips on hand washing: Hand washing is the single most important procedure for preventing transmission of infections. In contact with body secretions, excretions, Health care workers hands can carry bacteria, viruses, and fungi that may be potentially infectious. Hand washing antiseptic liquid soap (HITMAX™) is recommended. After contact with blood and or body substances, mucous membranes, soiled linen waste, or contaminated equipment. Immediately after removing gloves. Gloves may become perforated and bacteria can multiply rapidly on gloved hands. Before and after performing clean or sterile procedures. Between tasks at different body sites on the same patient (to prevent cross contamination). Between patients contact, and when other wise indicated to avoid transfer of microorganisms to other patients or environments. Before starting your shift duty and after completion of your duty. Before and after, drinking, Smoking, applying cosmetics or preparing food. After personal use of toilet.

How to perform hand washing: Remove jewelery (rings, bracelets) and watches. Rinse hands and wrists under water. Apply antiseptic liquid soap (HITMAX™). Using friction, wash hands for at least 10 to 15 seconds cleaning between fingers, nail beds, palms, back of hands, wrists and forearms. If hands are visibly soiled, more time may be required. If there is no assistant to close the tap, cover the tap with fresh tissue paper and gently close. Taking care to see that the hand does not come in contact with unsterile tap. Hand washing technique charts are displayed near sinks and can be followed. Additional alcohol-based solutions with residual activity (STERIMAX™/ TRIOSEPT™) (HICC approved hand rub) are recommended for use in setting where hand-washing facilities are inadequate or inaccessible and hands are not visibly soiled. If these solutions are used as a substitute for hand washing, hand washing with antiseptic liquid soap and water should be performed as soon as possible after procedure.

Universal / Standard Precautions: Wash hands with antiseptic liquid soap and water immediately, if it become contaminated with blood or body fluids. Wash hands routinely before and after contact with a patient and after take off the gloves. Apply standard precautions to all patients regardless of their diagnosis, and to all contaminated equipment and materials. Use judgement in determining when protective barriers are necessary. Wear examination gloves when the hands are likely to be in contact with blood or body fluids, mucous membranes, skin that has open cuts or sores, or contaminated items or surfaces. Wear a protective gown (wear on) or apron when you are likely to soil the cloths with blood or body fluids. Use caution when handling contaminated sharps. Dispose them off immediately after use in a puncture resistant container. Avoid recapping needles. Use a one –handed recapping technique when absolutely necessary. Wear examination gloves whenever handling laboratory specimens and tubes of blood. While performing procedures, use technique that minimize the splashing or spraying of body fluids. Use protective eyewear and mask as needed. Use ambu bag with facemask when giving CPR (cardiac pulmonary resuscitation). Clean up spills of blood or body fluid promptly using gloves, a towel and a disinfectant (1% sodium hypochlorite or SURFAX™). Place soiled linen in a soiled linen room and take it to its final place of disposal Clean, disinfect or sterilize contaminated equipment between uses and before sending equipment for repairs. It is mandatory for all health care workers to take Hepatitis B vaccination. Report any needle stick injury or blood or body fluid exposures promptly to the infection control team.

How to handle a bloodspill? Wear gloves. Cover the spill with cotton cloth or newspaper or any other absorbent material. Pour 1% sodium hypochlorite or SURFAX™ solution over the spill. Wipe spilled area after 30 minutes. Discard soiled material in yellow colour waste bag. Clean / mop the area with hard surface disinfectant like MICROLYSE™. Discard gloves into red colour waste bag. Wash hands with antiseptic liquid soap and water.

Precautions while handling sharps: Use caution when handling all sharps. Do not bend, break or manipulate sharps by hand. Place disposable sharps in puncture resistant container immediately after use. Do not recap or remove needles from a syringe by hand unless no alternative exists. Use one handed recap technique if you must recap a needle. Use a device instead of your hand to pick up or remove contaminated needles. Use a mechanical device (dustpans) to clean up broken glassware. Avoid passing sharps from person to person.

WASTE MANAGEMENT: Purpose: Dispose biomedical waste as per the guidelines. **Responsibility:** Laboratory staff. **Equipment:** Plastic containers. Plastic liners (bags) of four different colours (yellow, red, white and black) and a variety of sizes (big, medium and small) for collection of different types of infected and non infected wastes, a sealing mechanism at the neck of the bag (tie). Gloves. Puncture proof can. Waste collection trolleys. **Procedure:** All hospital waste should be segregated at source by the generator in colour coded plastic bags. Disposal of infected waste – all infected waste is collected in a container with a yellow colour plastic liner (bag). When it is ¾ full seal and send it to the utility room for collection. Disposal of infected plastics and rubber - collected in a container with red colour plastic bag. When it is ¾ full seal and send it to the utility room for collection. Disposal of non-infective plastics, rubber and glass - collected in a container with white colour plastic bag. When ¾ full seal and send it to the utility room for collection. Disposal of non-infective general waste – in black plastic liners (bags) kept in every wastepaper bin. It is removed from the waste bins, collected in large bags and sent to the utility room for collection. The same person replaces fresh liners immediately (if required). Disposal of sharps – needles, syringes, must be placed in a puncture proof container. If syringe and needle cannot be disengaged, both are discarded together into the container. When the container ¾ full will be closed and send it to the utility room for collection. All the categories of waste are weighed and entered into the statement of bio-medical waste format at the generation area and also at the handover to the outsourced agency. This is entered into a computer. Disposal of infected liquid – urine, sputum and other body fluids of all patients are flushed down into the toilet flush. All staff must wear appropriate gloves and protective clothing when handling infected waste (linen, materials or equipment) and strict hand washing procedures must be followed after each contact with patients or infected materials. All new employees shouldl receive mandatory training on handling waste.

VENIPUNCTURE: Venipuncture involves piercing a vein with a needle and collecting blood in a syringe or evacuated tube. **Equipment:** Tourniquet, gloves, syringe or vacutainer tube, vacutainer needle and adapter, alcohol swab, label, laboratory requisition form. **Procedure:** Wash your hands with antiseptic liquid soap (HITMAX™) thoroughly and wear gloves to prevent cross-contamination. Explain the procedure to the patient. If the patient is on bed rest, ask him to lie supine, with his head slightly elevated and his arms at his sides. Ask the ambulatory patient to sit in a chair and support his arm securely on an armrest or table. Assess the patient's veins to determine the best puncture site. Observe the skin for the vein's blue color, or palpate the vein for a firm rebound sensation. Tie a tourniquet 2" (5cm) proximal to the area chosen. Then ask him to close his fist as you insert the needle and to open it again when needle is in place. Clean the Venipuncture site with an alcohol sponge. Wipe in a circular motion and allow the skin to dry before performing Venipuncture. Immobilize the vein by pressing just below the Venipuncture site with your thumb and drawing the skin taut. Position the needle holder or syringe with the needle bevel up and the shaft parallel to the path of the vein and at a 30-degree angle to the arm. If you are using a syringe, venous blood will appear in the hub; with draw the blood slowly, pulling the plunger of the syringe gently to create steady suction until you obtain the required sample. Pulling the plunger too forcibly may collapse the vein. If you are using a needle holder and evacuated tube, a drop of blood will appear just inside the needle holder. Grasp the holder securely to stabilize it in the vein, and push down on the collection tube until the needle punctures the rubber stopper. Blood will flow into the tube automatically. Remove the tourniquet as soon as blood flows adequately. After you have drawn the sample, apply gentle pressure to the puncture site for 2 or 3 minutes or until

bleeding stops. If you have used a syringe, transfer the sample to a collection tube. Note: Avoid using veins in the patient's legs for venipuncture, if possible, because this increases the risk of thrombophlebitis. **Documentation:** Record the date, time and site of venipuncture; name of the test; the time the sample was sent to the laboratory.

FUMIGATION: With excellent housekeeping techniques routine fumigation is not required. However, most often only average cleaning is possible. In such circumstances fumigation can be done monthly and after epidemics. Fumigation should be done with eco-friendly fumigant like SILVICIDE™. Formalin should be avoided since it is carcinogenic. Doors, windows, walls and floors must be scrubbed thoroughly with HICC approved alkaline disinfectant (pH 8 to 9%) like MICROLYSE™ and water. Procedure: Doors, windows, walls and floors must be scrubbed thoroughly with MICROLYSE™ and water. All central oxygen and suction lines should be shut off. Ventilator outlets, air conditioner vents and gaps in doors and windows should be sealed airtight. All movable equipment and furniture should be removed and disinfected. Use a hydrogen peroxide + silver nitrate-based fumigant (SILVICIDE™) for fumigation. A fogger machine that can generate fumes at 5-15 microns particle size should be used for fumigation. **Fumigation procedure:** First calculate the area to be fogged (length x breadth x height in cubic feet). According to area, calculate the required quantity of disinfectant solution & Demineralised/ distilled water. Put your personal protective equipment like gloves, mask, lab coat. Take a measuring cylinder. Measure the required quantity of disinfectant solution & pour into the tank of the fogger. Again take the measuring cylinder & measure the required quantity of DM water, pour into the tank of the fogger. Gently shake the tank in clockwise direction. With switch in OFF position, plug the cord into power outlet of the proper voltage. Place the fogger at a corner of the room with nozzle direction pointed in desired position. Before switching ON ensure that intake air filter is in its place. Then switch ON the machine, fogger will generate the fog. Fogger machine should generate fine spray (5-15 microns) of the liquid & if a thick spray like comes out of the fogger, then adjust the knob (black color) to get a fine mist. Once you put ON the machine, come out of the room & lock the room. The room should be air-tight for maximum effect. After the said time open the door & go inside & put OFF the machine. Take the machine out & again close the room for 1 hour (no one is supposed to go inside for 1 hour which is the recommended contact time). After 1 hour the room is ready for use.

Managing exposure to blood or potentially infectious body fluids: Parental (needlestick) exposure to HIV infection is 0.3% risk of transmission of HIV. This is because of the low concentration of virus in the blood of infected patients. The risk in the case of HBV infected specimen in similar situations is 5-30 %.

Immediate care: For needle- stick injury: Briefly induce bleeding from wound. Wash for 10 minutes with HITMAX™ and water. For non- intact skin exposure: Wash with antiseptic soap and water or antiseptic like ZYTALL™/SAVINOX™/NUSEPT™. For mucosal exposure (e.g. Splash into eyes): Irrigate copiously by running a pint of normal saline over 10 minutes, the eye being held open the another person.

Reporting: All sharps injury (break of skin with any sharp instrument such as hypodermic needle previously used on a patient) and mucosal exposure (blood or body fluids coming into contact with eyes, mouth etc.) should be reported to the Hospital infection control chairman / nurse , immediately following exposure. All blood and body fluids with visible blood are considered infectious. Other body fluids may be potentially infectious and must be evaluated on case - to - case basis.

BOUQUET

In Lighter Vein

Behind every successful man, there is a woman And behind every unsuccessful man, there are two.

There are two theories to arguing with women. Neither one works.

What is the difference between a wife and a girlfriend? About 45 pounds!!

Hubby - You always carry my photo in your handbag to the office. Why? Wife - When there is a problem, no matter how impossible, I look at your picture and the problem disappears.

Hubby - You see, how miraculous and powerful I am for you? Wife - Yes, I see your picture and say to myself, "What other problem can there be greater than this one?"

Make it idiot proof and someone will make a better idiot.

Multitasking means screwing up several things at once.

The quickest way to double your money is to fold it in half and put it back in your pocket.

Don't drink and drive. You might hit a bump and spill your drink.

Energizer Bunny arrested and charged with battery.

Error, no keyboard. Press F1 to continue.

Oops. My brain just hit a bad sector.

Girl to her boyfriend: One kiss and I'll be yours forever.

The guy replies: Thanks for the warning.

Why was Phillip's girlfriend annoyed? Coz she found out that Phillips 24 inch was a TV.

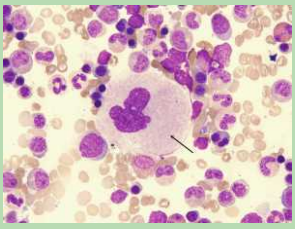
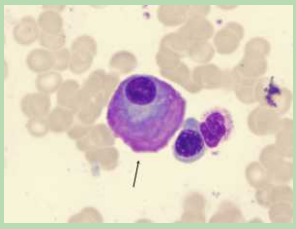
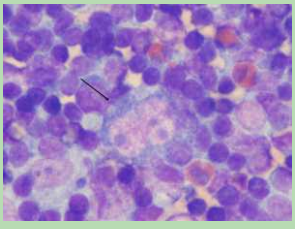
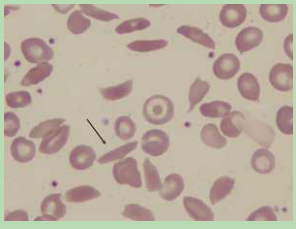
I'm not a complete idiot, some parts are missing!

Wisdom Whispers

- It is pretty hard to tell what does bring happiness; poverty and wealth have both failed.
- It is certain that the key to happiness is low expectations.
- When you relinquish the desire to control your future, you can have more happiness.
- One of the keys to happiness is a bad memory.
- Remember that happiness is a way of travel - not a destination.
- No man is happy who does not think himself so.
- One cannot believe that the inscrutable universe turns on an axis of suffering; surely the strange beauty of the world must somewhere rest on pure joy!

Brain Teasers

Identify the following cells marked by arrows in the pictures given below.

1. 
2. 
3. 
4. 

Answers: 1.Megakaryocyte, 2. Flame (plasma) cell, 3. Reed Sternberg cell, 4. Sickle cell

INTERPRETATION

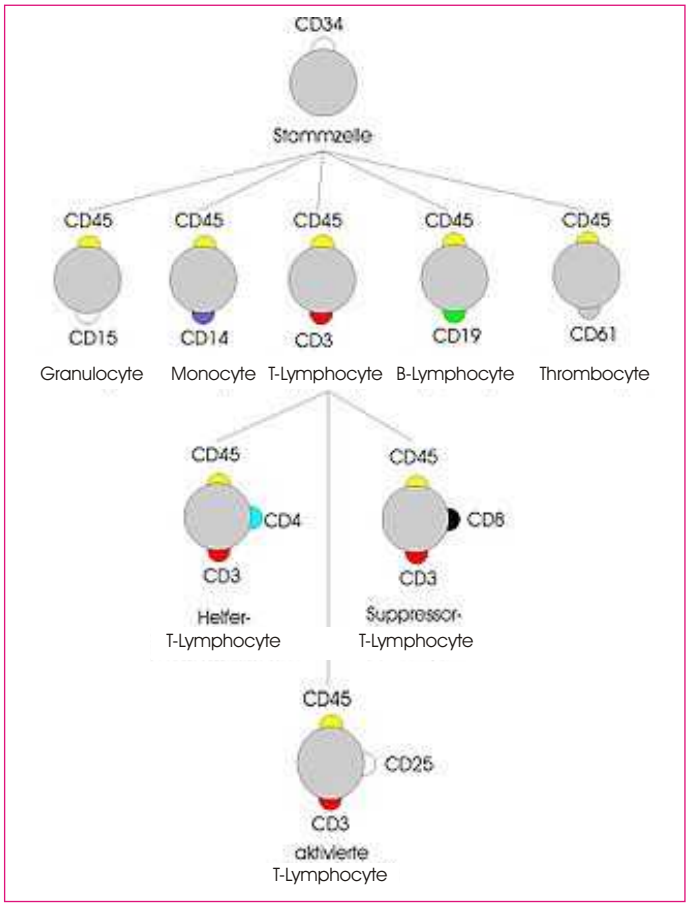
CLUSTER OF DIFFERENTIATION

The cluster of differentiation (cluster of designation) (often abbreviated as CD) is a protocol used for the identification and investigation of cell surface molecules present on leukocytes. CD molecules can act in numerous ways, often acting as receptors or ligands (the molecule that activates a receptor) important to the cell. A signal cascade is usually initiated, altering the behavior of the cell. Some CD proteins do not play a role in cell signaling, but have other functions, such as cell adhesion. There are approximately 250 different proteins.

Nomenclature

The CD nomenclature was proposed and established in the 1st International Workshop and Conference on Human Leukocyte Differentiation Antigens (HLDA), which was held in Paris in 1982. This system was intended for the classification of the many monoclonal antibodies (mAbs) generated by different laboratories around the world against epitopes on the surface molecules of leukocytes (white blood cells). Since then, its use has expanded to many other cell types, and more than 320 CD unique clusters and subclusters have been identified. The proposed surface molecule is assigned a CD number once two specific monoclonal antibodies (mAb) are shown to bind to the molecule. If the molecule has not been well-characterized, or has only one mAb, it is usually given the provisional indicator "w" (as in "CDw186").

Cell markers



CD differentiation.

The CD system is commonly used as cell markers, allowing cells to be defined based on what molecules are present on their surface. These markers are often used to associate cells with certain immune functions. While using one CD molecule to define populations is uncommon (though a few examples exist), combining markers has allowed for cell types with very specific definitions within the immune system.

CD molecules are utilized in cell sorting using various methods including flow cytometry. Cell populations are usually defined using a '+' or a '–' symbol to indicate whether a certain cell fraction expresses or lacks a CD molecule.

For example, a "CD34+, CD31–" cell is one that expresses CD34, but not CD31. This CD combination typically corresponds to a stem cell, opposed to a fully-differentiated endothelial cell.

Type of cell	CD markers
Stem cells	CD34+,CD31-
All leukocyte groups	CD45+
Granulocyte	CD45+,CD15+
Monocyte	CD45+,CD14+
T lymphocyte	CD45+,CD3+
T helper cell	CD45+,CD3+,CD4+
Cytotoxic T cell	CD45+,CD3+,CD8+
B lymphocyte	CD45+,CD19+ or CD45+,CD20+
Thrombocyte	CD45+,CD61+
Natural killer cell	CD16+,CD56+,CD3-

Two commonly-used CD molecules are CD4 and CD8, which are, in general, used as markers for helper and cytotoxic T cells, respectively. These molecules are defined in combination with CD3+, as some other leukocytes also express these CD molecules (some macrophages express low levels of CD4; dendritic cells express high levels of CD8).

Human immunodeficiency virus (HIV) binds CD4 and a chemokine receptor on the surface of a T helper cell to gain entry. The number of CD4 and CD8 T cells in blood is often used to monitor the progression of HIV infection.

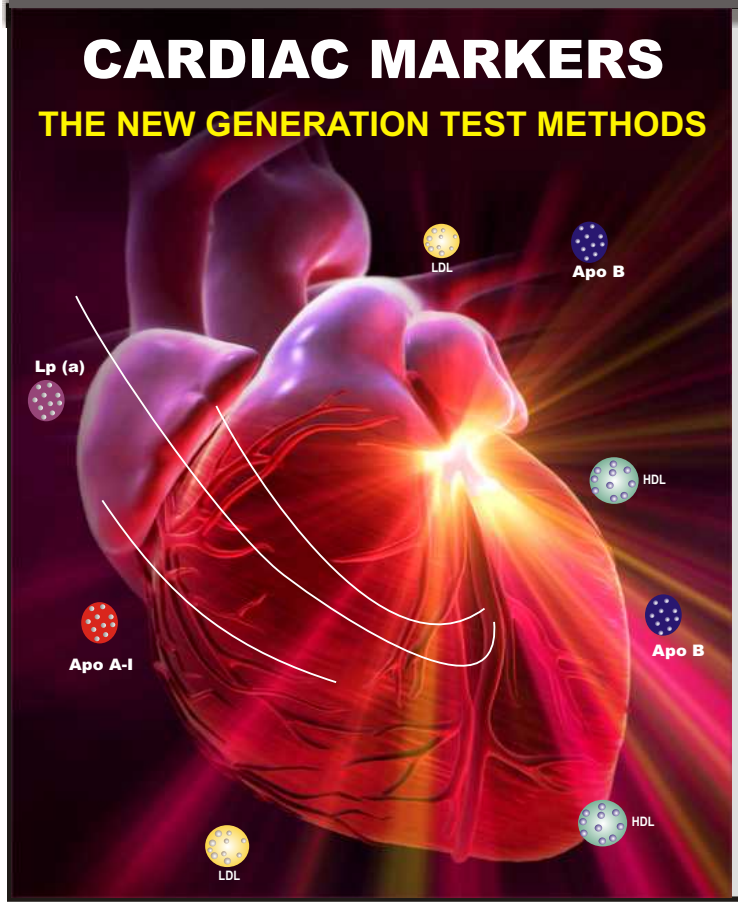
Other uses

It is important to note that, while CD molecules are very useful in defining leukocytes, they are not merely markers on the cell surface. While only a fraction of known CD molecules have been thoroughly characterised, most of them have an important function. In the example of CD4 & CD8, these molecules are critical in antigen recognition.

TULIP NEWS

CARDIAC MARKERS

THE NEW GENERATION TEST METHODS



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Quantia™ Apo A-I

Quantia™ Lp (a)

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presents

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DIRECT LDL-D CHOLESTEROL

Presentation: 40/160/240 ml

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