

1.1 INTRODUCTION

Physiologically, mother becomes almost a new person during the period of pregnancy. Profound local and systemic changes in maternal physiology are initiated by conception and continued throughout pregnancy. As pregnancy progresses, a well-integrated metabolic shift occurs to ensure an adequate supply of nutrients to a constantly feeding fetus from an intermittently fasting and feeding mother. Cholesterol belongs to the sterol group of fats and is present in the egg yolk, dairy products, fatty meals and meat. It has good side effects because it performs number of vital functions in the body such as providing an essential component of membranes and serving as a precursor of bile acids, steroid hormones. The blood stream carries cholesterol in particles called lipoprotein. But too much of circulating cholesterol can injure arteries especially coronary arteries that supply blood to the heart. This leads to the accumulation of cholesterol laden “Plaque” in vessel linings a condition called Atherosclerosis. When blood flow to the heart is impeded, the heart muscle becomes starved of oxygen causing chest pain (angina). If a blood clot completely obstructs a coronary artery affected by atherosclerosis, heart attack (myocardial infarction) or death can occur, lipid profile is a group of tests that are used to determine risk of coronary heart diseases. The lipid profile tests include Total cholesterol, Triglycerides, HDL cholesterol, LDL cholesterol, VLDL cholesterol. The purpose of this study was to determine whether the changes that occur in the rate of lipid pose a real danger to the pregnant woman. This study is based upon the effect of pregnancy on serum lipid profile and so that steps may be minimized to solve cardiovascular and other complications, which may help to promote and preserve women’s reproductive health.

1.2 literature review

1.2.1 Pregnancy

Pregnancy is the fertilization and development of one or more offspring, known as an embryo or fetus, in a woman's uterus. It is the common name for gestation in humans. A multiple pregnancy involves more than one embryo or fetus in a single pregnancy, such as with twins. Childbirth usually occurs about 38 weeks after conception; in women who have a menstrual cycle length of four weeks, this is approximately 40 weeks from the start of the last normal menstrual period. .(David , 2011)

An embryo is the developing offspring during the first 8 weeks following conception, and subsequently the term fetus is used until birth in many societies' medical or legal definitions, human pregnancy is somewhat arbitrarily divided into three trimester periods, as a means to simplify reference to the different stages of prenatal development. The first trimester carries the highest risk of miscarriage (natural death of embryo or fetus). During the second trimester, the development of the fetus can be more easily monitored and diagnosed. The third trimester is marked by further growth of the fetus and the development of fetal fat stores. The point of fetal viability, or the point in time at which fetal life outside of the uterus is possible, usually coincides with the late second or early third trimesters, and is typically associated with high degrees of morbidity and mortality (David , 2011).

1.2.1.2 Stages of pregnancy:

First trimester (week 1-week 12):

During the first trimester the body undergoes many changes. Hormonal changes affect almost every organ system in body. These changes can trigger symptoms even in the very first weeks of pregnancy. Period stopping is a clear sign that you are pregnant. Other changes may include:

- Extreme tiredness
- Tender, swollen breasts. Nipples might also stick out.
- Upset stomach with or without throwing up (morning sickness)

- Cravings or distaste for certain foods
- Mood swings
- Constipation (trouble having bowel movements)
- Need to pass urine more often
- Headache
- Heartburn
- Weight gain or loss(buff, 2010).

Second Trimester

By the end of the second trimester, the expanding uterus has created a visible "baby bump". Although the breasts have been developing internally since the beginning of the pregnancy, most of the visible changes appear after this point. Weeks 13 to 28 of the pregnancy are called the second trimester. Most women feel more energized in this period, and begin to put on weight as the symptoms of morning sickness subside and eventually fade away. The uterus, the muscular organ that holds the developing fetus, can expand up to 20 times its normal size during pregnancy. Although the fetus begins to move and takes a recognizable human shape during the first trimester, it is not until the second trimester that movement of the fetus, often referred to as "quickening", can be felt. This typically happens in the fourth month, more specifically in the 20th to 21st week, or by the 19th week if the woman has been pregnant before. However, it is not uncommon for some women not to feel the fetus move until much later. During the second trimester, most women begin to wear maternity clothes (Stacey *et al*,2011).

Third Trimester

The uterus expands making up a larger and larger portion of the woman's abdomen. During the final stages of gestation before childbirth the fetus and uterus will drop to a lower position. Final weight gain takes place, which is the most weight gain throughout the pregnancy. The woman's abdomen will transform in shape as it drops due to the fetus turning in a downward position ready for birth. During the second trimester, the woman's abdomen would have been very upright, whereas in the third trimester it will drop down quite low, and the woman will be able to lift her abdomen up and down. The fetus begins to move regularly, and is felt by the woman. Fetal

movement can become quite strong and be disruptive to the woman. The woman's navel will sometimes become convex, "popping" out, due to her expanding abdomen. Head engagement, where the fetal head descends into cephalic presentation, relieves pressure on the upper abdomen with renewed ease in breathing. However, it severely reduces bladder capacity, increases pressure on the pelvic floor and the rectum. It is also during the third trimester that maternal activity and sleep positions may affect fetal development due to restricted blood flow. For instance, the enlarged uterus may impede blood flow by compressing the lower pressured vena cava, with the left lateral laying positions appearing to providing better oxygenation to the infant (Stacey *et al*,2011).

1.2.1.3 Physiological Changes in Pregnancy

Pregnancy is associated with normal physiological changes that assist fetal survival as well as preparation for labour. It is important to know what 'normal' parameters of change are in order to diagnose and manage common medical problems of pregnancy, such as hypertension, gestational diabetes, anemia and hyperthyroidism.

Endocrine system (non-reproductive)

Pituitary

- FSH/LH falls to low levels.
- ACTH and melanocyte-stimulating hormone increase.
- Prolactin increases.

Thyroid and parathyroid

- Thyroxin-binding globulin (TBG) concentrations rise due to increased estrogen levels.
- T4 and T3 increase over the first half of pregnancy but there is a normal to slightly decreased amount of free hormone due to increased TBG-binding.
- TSH production is stimulated, although in healthy individuals this is not usually significant. A large rise in TSH is likely to indicate iodine deficiency or subclinical hypothyroidism (Lazarus , et al ;2004).

- Serum calcium levels decrease in pregnancy, which stimulates an increase in parathyroid hormone (PTH).
- Colecalciferol (vitamin D3) is converted to its active metabolite, 1,25-dihydroxycolecalciferol, by placental 1 α -hydroxylase (Lazarus et al ;2004).

Adrenal and pancreas

- Cortisol levels increase in pregnancy, which favours lipogenesis and fat storage.
- Insulin response also increases so blood sugar should remain normal or low.
- Peripheral insulin resistance may also develop over the course of pregnancy and gestational diabetes is thought to reflect a pronounced insulin resistance of this sort (Butte et al ; 2000).

1.2.2 Lipid

Lipids are a group of naturally occurring molecules that include fats, waxes, sterols, fat-soluble vitamins (such as vitamins A, D, E, and K), monoglycerides, diglycerides, triglycerides, phospholipids, and others. The main biological functions of lipids include storing energy, signaling, and acting as structural components of cell membranes. Lipids have applications in the cosmetic and food industries as well as in nanotechnology (Mashaghi *et al*; 2013).

Lipids may be broadly defined as hydrophobic or amphiphilic small molecules; the amphiphilic nature of some lipids allows them to form structures such as vesicles, liposomes, or membranes in an aqueous environment. Biological lipids originate entirely or in part from two distinct types of biochemical subunits or "building-blocks": ketoacyl and isoprene groups. Using this approach, lipids may be divided into eight categories: fatty acids, glycerolipids, glycerophospholipids, sphingolipids, saccharolipids, and polyketides (derived from condensation of ketoacyl subunits) and sterol lipids and prenol lipids (derived from condensation of isoprene subunits) (Mashaghi *et al*; 2013).

Although the term lipid is sometimes used as a synonym for fats, fats are a subgroup of lipids called triglycerides. Lipids also encompass molecules such as fatty acids and

their derivatives (including tri-, di-, monoglycerides, and phospholipids), as well as other sterol-containing metabolites such as cholesterol. Although humans and other mammals use various biosynthetic pathways to both break down and synthesize lipids, some essential lipids cannot be made this way and must be obtained from the diet (Mashaghi *et al*; 2013).

1.2.2.1 Cholesterol :-

Cholesterol, from the Ancient Greek chole- (bile) and stereos (solid) followed by the chemical suffix -ol for an alcohol, is an organic molecule. It is a sterol (or modified steroid).and an essential structural component of animal cell membranes that is required to establish proper membrane permeability and fluidity. Cholesterol is thus considered within the class of lipid molecules.

In addition to its importance within cells, cholesterol also serves as a precursor for the biosynthesis of steroid hormones, bile acids, and vitamin D. Cholesterol is the principal sterol synthesized by animals, all cells; in vertebrates the liver typically produces greater amounts than other cells. It is almost completely absent among prokaryotes (i.e., bacteria), although there are some exceptions such as *Mycoplasma*, which require cholesterol for growth (Razin *et al*, 1970).

Physiology of cholesterol :-

Since cholesterol is essential for all animal life, each cell synthesizes it from simpler molecules, a complex 37-step process that starts with the intracellular protein enzyme HMG-CoA reductase. However, normal and particularly high levels of fats (including cholesterol) in the blood circulation, depending on how they are transported within lipoproteins, are strongly associated with the progression of atherosclerosis.

For a man of about 68 kg (150 lb), typical total body-cholesterol synthesis is approximately 1 g (1,000 mg) per day, and total body content is approximately 35 g, primarily located within the membranes of all the cells of the body. Typical daily dietary intake of additional cholesterol, in the United States, is 200–300 mg. most ingested cholesterol is esterified, and esterified cholesterol is poorly absorbed. The body also compensates for any absorption of additional cholesterol by reducing

cholesterol synthesis. For these reasons, cholesterol intake in food has little, if any, effect on total body cholesterol content or concentrations of cholesterol in the blood (Lecerf *et al* ; 2011).

Cholesterol is recycled. The liver excretes it in a non-esterified form (via bile) into the digestive tract. Typically about 50% of the excreted cholesterol is reabsorbed by the small bowel back into the bloodstream (Lecerf *et al* ; 2011).

Plants make cholesterol in very small amounts. Plants manufacture phytosterols (substances chemically similar to cholesterol produced within plants), which can compete with cholesterol for reabsorption in the intestinal tract, thus potentially reducing cholesterol reabsorption. When intestinal lining cells absorb phytosterols, in place of cholesterol, they usually excrete the phytosterol molecules back into the GI tract, an important protective mechanism (Lecerf *et al* ; 2011).

Function of cholesterol :-

Cholesterol is required to build and maintain membranes; it modulates membrane fluidity over the range of physiological temperatures. The hydroxyl group on cholesterol interacts with the polar head groups of the membrane phospholipids and sphingolipids, while the bulky steroid and the hydrocarbon chain are embedded in the membrane, alongside the nonpolar fatty-acid chain of the other lipids. Through the interaction with the phospholipid fatty-acid chains, cholesterol increases membrane packing, which reduces membrane fluidity. The structure of the tetracyclic ring of cholesterol contributes to the decreased fluidity of the cell membrane as the molecule is in a Trans conformation making all but the side chain of cholesterol rigid and planar. In this structural role, cholesterol reduces the Permeability of the plasma membrane to neutral solutes, protons, (positive hydrogen ions) and sodium ions (Sadava ; 2011).

Within the cell membrane, cholesterol also functions in intracellular transport, cell signaling and nerve conduction. Cholesterol is essential for the structure and function of invaginated caveolae and clathrin-coated pits, including caveola-dependent and clathrin-dependent endocytosis. The role of cholesterol in such endocytosis can be investigated by using methyl beta cyclodextrin (M β CD) to remove cholesterol from

the plasma membrane. Recently, cholesterol has also been implicated in cell signaling processes, assisting in the formation of lipid rafts in the plasma membrane. Lipid raft formation brings receptor proteins in close proximity with high concentrations of second messenger molecules. In many neurons, a myelin sheath, rich in cholesterol, since it is derived from compacted layers of Schwann cell membrane, provides insulation for more efficient conduction of impulses (Sadava ; 2011).

Within cells, cholesterol is the precursor molecule in several biochemical pathways. In the liver, cholesterol is converted to bile, which is then stored in the gallbladder. Bile contains bile salts, which solubilize fats in the digestive tract and aid in the intestinal absorption of fat molecules as well as the fat-soluble vitamins, A, D, E, and K. Cholesterol is an important precursor molecule for the synthesis of vitamin D and the steroid hormones, including the adrenal gland hormones cortisol and aldosterone, as well as the sex hormones progesterone, estrogens, and testosterone, and their derivatives(Sadava ; 2011) .

Dietary sources of cholesterol :-

Animal fats are complex mixtures of triglycerides, with lesser amounts of phospholipids and cholesterol. As a consequence, all foods containing animal fat contain cholesterol to varying extents. Major dietary sources of cholesterol include cheese, egg Yolks, beef, pork, poultry, fish, and shrimp. Human breast milk also contains significant quantities of cholesterol (Weingärtner *et al*;2011).

From a dietary perspective, cholesterol is not found in significant amounts in plant sources. In addition, plant products such as flax seeds and peanuts contain cholesterol-like compounds called phytosterols, which are believed to compete with cholesterol for absorption in the intestines. Phytosterols can be supplemented through the use of phytosterol-containing functional foods or nutraceuticals that are widely recognized as having a proven LDL cholesterol-lowering efficacy. Current supplemental guidelines recommend doses of phytosterols in the 1.6-3.0 grams per day range (Health Canada, EFSA, ATP III, and FDA) with a recent meta-analysis demonstrating an 8.8% reduction in LDL-cholesterol at a mean dose of 2.15 gram per day. However, the benefits of a diet supplemented with phytosterol have been questioned.

Fat intake also plays a role in blood-cholesterol levels. This effect is thought to come about by changes in the quantity of cholesterol and lipoproteins that are synthesized by the body. Isocalorically replacing dietary carbohydrates with monounsaturated and polyunsaturated fats has been shown to lower serum LDL and total cholesterol levels and increase serum HDL levels, while replacing carbohydrates with saturated fat was shown to increase HDL, LDL, and total cholesterol levels. Trans fats have been shown to reduce levels of HDL while increasing levels of LDL. Based on such evidence and evidence implicating low HDL and high LDL levels in cardiovascular disease, many health authorities advocate reducing LDL cholesterol through changes in diet in addition to other lifestyle modifications(Weingärtner *et al*;2011).

Biosynthesis of cholesterol :-

All animal cells manufacture cholesterol with relative production rates varying by cell type and organ function. About 20–25% of total daily cholesterol production occurs in the liver; other sites of higher synthesis rates include the intestines, adrenal glands, and reproductive organs. Synthesis within the body starts with one molecule of acetyl CoA and one molecule of acetoacetyl-CoA, which are hydrated to form 3-hydroxy-3-methylglutaryl CoA (HMG-CoA). This molecule is then reduced to mevalonate by the enzyme HMG-CoA reductase. This is the regulated, rate-limiting and irreversible step in cholesterol synthesis and is the site of action for the statin drugs (HMG-CoA reductase competitive inhibitors).

Mevalonate is then converted to 3-isopentenyl pyrophosphate in three reactions that require ATP. Mevalonate is decarboxylated to isopentenyl pyrophosphate, which is a key metabolite for various biological reactions. Three molecules of isopentenyl pyrophosphate condense to form farnesyl pyrophosphate through the action of geranyl transferase. Two molecules of farnesyl pyrophosphate then condense to form squalene by the action of squalene synthase in the endoplasmic reticulum. Oxidosqualene cyclase then cyclizes squalene to form lanosterol. Finally, lanosterol is converted to cholesterol through a 19-step process (Espenshade *et al*;2007).

Regulation of cholesterol synthesis

Biosynthesis of cholesterol is directly regulated by the cholesterol levels present, though the homeostatic mechanisms involved are only partly understood. A higher intake from food leads to a net decrease in endogenous production, whereas lower intake from food has the opposite effect. The main regulatory mechanism is the sensing of intracellular cholesterol in the endoplasmic reticulum by the protein SREBP (sterol regulatory element-binding protein 1 and 2). In the presence of cholesterol, SREBP is bound to two other proteins: SCAP (SREBP cleavage activating protein) and Insig1. When cholesterol levels fall, Insig-1 dissociates from the SREBP-SCAP complex, which allows the complex to migrate to the Golgi apparatus. Here SREBP is cleaved by S1P and S2P (site-1 and -2 protease), two enzymes that are activated by SCAP when cholesterol levels are low (Brown *et al*,1997).

The cleaved SREBP then migrates to the nucleus, and acts as a transcription factor to bind to the sterol regulatory element (SRE), which stimulates the transcription of many genes. Among these are the low-density lipoprotein (LDL) receptor and HMG-CoA reductase. The LDL receptor former scavenges circulating LDL from the bloodstream, whereas HMG-CoA reductase leads to an increase of endogenous production of cholesterol. Cholesterol synthesis can also be turned off when cholesterol levels are high. HMG-CoA reductase contains both a cytosolic domain (responsible for its catalytic function) and a membrane domain. The membrane domain senses signals for its degradation. Increasing concentrations of cholesterol (and other sterols) cause a change in this domain's oligomerization state, which makes it more susceptible to destruction by the proteasome. This enzyme's activity can also be reduced by phosphorylation by an AMP-activated protein kinase. Because this kinase is activated by AMP, which is produced when ATP is hydrolyzed, it follows that cholesterol synthesis is halted when ATP levels are low(Brown *et al*,1997).

Plasma transport and regulation of cholesterol absorption :-

Cholesterol is only slightly soluble in water; it can dissolve and travel in the water-based bloodstream at exceedingly small concentrations. Since cholesterol is insoluble in blood, it is transported in the circulatory system within lipoproteins, complex discoidal particles that have an exterior composed of amphiphilic proteins and lipids whose outward-facing surfaces are water-soluble and inward-facing surfaces are lipid-soluble; triglycerides and cholesterol esters are carried internally. Phospholipids and cholesterol, being amphipathic, are transported in the surface monolayer of the lipoprotein particle. In addition to providing a soluble means for transporting cholesterol through the blood, lipoproteins have cell-targeting signals that direct the lipids they carry to certain tissues. For this reason, there are several types of lipoproteins in blood, called, in order of increasing density, chylomicrons, very-low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL). The more lipids and less protein a lipoprotein has, the less dense it is. The cholesterol within all the various lipoproteins is identical, although some cholesterol is carried as the "free" alcohol and some is carried as fatty acyl esters referred to as cholesterol esters. However, the different lipoproteins contain apolipoproteins, which serve as ligands for specific receptors on cell membranes. In this way, the lipoprotein particles are molecular addresses that determine the start- and endpoints for cholesterol transport (Tymoczko *et al*, 2002).

Chylomicrons, the least dense type of cholesterol transport molecules, contain apolipoprotein B-48, apolipoprotein C, and apolipoprotein E in their shells. Chylomicrons are the transporters that carry fats from the intestine to muscle and other tissues that need fatty acids for energy or fat production. Cholesterol that is not used by muscles remains in more cholesterol-rich chylomicron remnants, which are taken up from here to the bloodstream by the liver. VLDL molecules are produced by the liver and contain excess triacylglycerol and cholesterol that is not required by the liver for synthesis of bile acids. These molecules contain apolipoprotein B100 and apolipoprotein E in their shells. During transport in the bloodstream, the blood vessels cleave and absorb more triacylglycerol from IDL molecules, which contain an even higher percentage of cholesterol. The IDL molecules have two possible fates: Half are

metabolized by HTGL, taken up by the LDL receptor on the liver cell surfaces, and the other half continue to lose triacylglycerols in the bloodstream until they form LDL molecules, which have the highest percentage of cholesterol within them. LDL molecules, therefore, are the major carriers of cholesterol in the blood, and each one contains approximately 1,500 molecules of cholesterol ester. The shell of the LDL molecule contains just one molecule of apolipoprotein B100, which is recognized by the LDL receptor in peripheral tissues. Upon binding of apolipoprotein B100, many LDL receptors become localized in clathrin-coated pits. Both the LDL and its receptor are internalized by endocytosis to form a vesicle within the cell. The vesicle then fuses with a lysosome, which has an enzyme called lysosomal acid lipase that hydrolyzes the cholesterol esters. Now within the cell, the cholesterol can be used for membrane biosynthesis or esterified and stored within the cell, so as to not interfere with cell membranes. Synthesis of the LDL receptor is regulated by SREBP, the same regulatory protein as was used to control synthesis of cholesterol *de novo* in response to cholesterol presence in the cell. When the cell has abundant cholesterol, LDL receptor synthesis is blocked so new cholesterol in the form of LDL molecules cannot be taken up. On the converse, more LDL receptors are made when the cell is deficient in cholesterol. When this system is deregulated, many LDL molecules appear in the blood without receptors on the peripheral tissues. These LDL molecules are oxidized and taken up by macrophages, which become engorged and form foam cells. These cells often become trapped in the walls of blood vessels and contribute to atherosclerotic plaque formation. Differences in cholesterol homeostasis affect the development of early atherosclerosis (carotid intima-media thickness). These plaques are the main causes of heart attacks, strokes, and other serious medical problems, leading to the association of so-called LDL cholesterol (actually a lipoprotein) with "bad" cholesterol. Also, HDL particles are thought to transport cholesterol back to the liver for excretion or to other tissues that use cholesterol to synthesize hormones in a process known as reverse cholesterol transport (RCT). Having large numbers of large HDL particles correlates with better health outcomes. In contrast, having small numbers of large HDL particles is independently associated with atheromatous disease progression in the arteries (Tymoczko et al, 2002).

Metabolism, recycling and excretion of cholesterol :-

Cholesterol is susceptible to oxidation and easily forms oxygenated derivatives known as oxysterols. Three different mechanisms can form these; autooxidation, secondary oxidation to lipid peroxidation, and cholesterol-metabolizing enzyme oxidation. A great interest in oxysterols arose when they were shown to exert inhibitory actions on cholesterol biosynthesis. This finding became known as the “oxysterol hypothesis”.

Additional roles for oxysterols in human physiology include there: participation in bile acid biosynthesis, function as transport forms of cholesterol, and regulation of gene transcription. In biochemical experiments radiolabelled forms of cholesterol, such as tritiated-cholesterol are used. These derivatives undergo degradation upon storage and it is essential to purify cholesterol prior to use. Cholesterol can be purified using small Sephadex LH-20 columns. Cholesterol is oxidized by the liver into a variety of bile acids. These, in turn, are conjugated with glycine, taurine, glucuronic acid, or sulfate. A mixture of conjugated and nonconjugated bile acids, along with cholesterol itself, is excreted from the liver into the bile. Approximately 95% of the bile acids are reabsorbed from the intestines, and the remainders are lost in the feces. The excretion and reabsorption of bile acids forms the basis of the enterohepatic circulation, which is essential for the digestion and absorption of dietary fats. Under certain circumstances, when more concentrated, as in the gallbladder, cholesterol crystallises and is the major constituent of most gallstones. Although, lecithin and bilirubin gallstones also occur, but less frequently. Every day, up to 1 g of cholesterol enters the colon. This cholesterol originates from the diet, bile, and desquamated intestinal cells, and can be metabolized by the colonic bacteria. Cholesterol is converted mainly into coprostanol, a nonabsorbable sterol that is excreted in the feces. A cholesterol-reducing bacterium origin has been isolated from human feces (Lewis and Rader ;2005).

Clinical significance of cholesterol

Hypercholesterolemia :-

According to the lipid hypothesis, abnormal cholesterol levels (hypercholesterolemia) — actually higher concentrations of LDL particles and lower

concentrations of functional HDL particles — are strongly associated with cardiovascular disease because these promote atheroma development in arteries (atherosclerosis). This disease process leads to myocardial infarction (heart attack), stroke, and peripheral vascular disease. Since higher blood LDL, especially higher LDL particle concentrations and smaller LDL particle size, contribute to this process more than the cholesterol content of the HDL particles, LDL particles are often termed "bad cholesterol" because they have been linked to atheroma formation. On the other hand, high concentrations of functional HDL, which can remove cholesterol from cells and atheroma, offer protection and are sometimes referred to as "good cholesterol". These balances are mostly genetically determined, but can be changed by body build, medications, food choices, and other factors. Resistin, a protein secreted by fat tissue, has been shown to increase the production of LDL in human liver cells and also degrades LDL receptors in the liver. As a result, the liver is less able to clear cholesterol from the bloodstream. Resistin accelerates the accumulation of LDL in arteries, increasing the risk of heart disease. Resistin also adversely impacts the effects of statins, the main cholesterol-reducing drug used in the treatment and prevention of cardiovascular disease (Lewington *et al* ;2007).

Conditions with elevated concentrations of oxidized LDL particles, especially "small dense LDL" (sdLDL) particles, are associated with atheroma formation in the walls of arteries, a condition known as atherosclerosis, which is the principal cause of coronary heart disease and other forms of cardiovascular disease. In contrast, HDL particles (especially large HDL) have been identified as a mechanism by which cholesterol and inflammatory mediators can be removed from atheroma. Increased concentrations of HDL correlate with lower rates of atheroma progressions and even regression. elevated levels of the lipoprotein fractions, LDL, IDL and VLDL are regarded as atherogenic (prone to cause atherosclerosis). Levels of these fractions, rather than the total cholesterol level, correlate with the extent and progress of atherosclerosis. Conversely, the total cholesterol can be within normal limits, yet be made up primarily of small LDL and small HDL particles, under which conditions atheroma growth rates would still be high. Recently, a post hoc analysis of the IDEAL and the EPIC prospective studies found an association between high levels of HDL cholesterol (adjusted for apolipoprotein A-I And apolipoprotein B) and increased risk of cardiovascular disease, casting doubt on the cardio protective role of "good

cholesterol. Total cholesterol is defined as the sum of HDL, LDL, and VLDL. Usually, only the total, HDL, and triglycerides are measured. For cost reasons, the VLDL is usually estimated as one-fifth of the triglycerides and the LDL is estimated using the Friedewald formula (or a variant): $\text{estimated LDL} = [\text{total cholesterol}] - [\text{total HDL}] - [\text{estimated VLDL}]$. VLDL can be calculated by dividing total triglycerides by five. Direct LDL measures are used when triglycerides exceed 400 mg/dL. The estimated VLDL and LDL have more error when triglycerides are above 400 mg/dL (Lewington *et al* ;2007).

Hypocholesterolemia

Abnormally low levels of cholesterol are termed hypocholesterolemia. Research into the causes of this state is relatively limited, but some studies suggest a link with depression, cancer, and cerebral hemorrhage. In general, the low cholesterol levels seem to be a consequence, rather than a cause, Of an underlying illness. A genetic defect in cholesterol synthesis causes Smith-Lemli-Opitz syndrome, which is often associated with low plasma cholesterol levels (Lewington *et al* ;2007).

Causes of hypercholesterolemia :-

Possible causes of low cholesterol are:

- statins
- hyperthyroidism, or an overactive thyroid gland
- adrenal insufficiency
- liver disease
- malabsorption (inadequate absorption of nutrients from the intestines), such as in celiac disease
- malnutrition
- Abetalipoproteinemia - a rare genetic disease that causes cholesterol readings below 50 mg/dl. It is found mostly in Jewish populations.
- hypobetalipoproteinemia - a genetic disease that causes cholesterol readings below 50 mg/dl
- manganese deficiency
- Smith-Lemli-Opitz syndrome

- Marfan syndrome
- leukemias and other (Lewington S *et al* ;2007).

1.2.2.2 Triglyceride

A triglyceride (TG, triacylglycerol, TAG, or triacylglyceride) is an ester derived from glycerol and three fatty acids. As a blood lipid, it helps enable the bidirectional transference of adipose fat and blood glucose from the liver. There are many triglycerides: depending on the oil source, some are highly unsaturated, some less so. Saturated compounds are "saturated" with hydrogen — all available places where hydrogen atoms could be bonded to carbon atoms are occupied. Unsaturated compounds have double bonds (C=C) between carbon atoms, reducing the number of places where hydrogen atoms can bond to carbon atoms. Saturated compounds have single bonds (C-C) between the carbon atoms, and the other bond is bound to hydrogen atoms (for example =CH-CH=, -CH₂-CH₂-, etc.). Unsaturated fats have a lower melting point and are more likely to be liquid. Saturated fats have a higher melting point and are more likely to be solid at room temperature. Triglycerides are the main constituents of vegetable oil (typically more unsaturated) and animal fats (typically more saturated). Triglycerides are a major component of human skin oils(Nelson; 2000).

Metabolism of Triglyceride:

The enzyme pancreatic lipase acts at the ester bond, hydrolyzing the bond and "releasing" the fatty acid. In triglyceride form, lipids cannot be absorbed by the duodenum. Fatty acids, monoglycerides (one glycerol, one fatty acid), and some diglycerides are absorbed by the duodenum, once the triglycerides have been broken down. in the intestine, following the secretion of lipases and bile, triglycerides are split into monoacylglycerol and free fatty acids in a process called lipolysis. They are subsequently moved to absorptive enterocyte cells lining the intestines. The triglycerides are rebuilt in the enterocytes from their fragments and packaged together with cholesterol and proteins to form chylomicrons. These are excreted from the cells and collected by the lymph system and transported to the large vessels near the heart before being mixed into the blood. Various tissues can capture the chylomicrons,

releasing the triglycerides to be used as a source of energy. Fat and liver cells can synthesize and store triglycerides. When the body requires fatty acids as an energy source, the hormone glucagon signals the breakdown of the triglycerides by hormone-sensitive lipase to release free fatty acids. As the brain cannot utilize fatty acids as an energy source (unless converted to a ketone), the glycerol component of triglycerides can be converted into glucose, via gluconeogenesis by conversion into Dihydroxyacetone phosphate and then into Glyceraldehydes 3-phosphate, for brain fuel when it is broken down. Fat cells may also be broken down for that reason, if the brain's needs ever outweigh the bodies (Nelson ;2000).

Triglycerides cannot pass through cell membranes freely. Special enzymes on the walls of blood vessels called lipoprotein lipases must break down triglycerides into free fatty acids and glycerol. Fatty acids can then be taken up by cells via the fatty acid transporter (FAT) (Nelson ;2000).

Clinical significant of triglyceride :-

Hypertriglyceridemia:

Hypertriglyceridemia denotes high (hyper-) blood levels (emia) of triglycerides, the most abundant fatty molecule in most organisms. Elevated levels of triglycerides are associated with atherosclerosis, even in the absence of hypercholesterolemia (high cholesterol levels), and predispose to cardiovascular disease. Very high triglyceride levels also increase the risk of acute pancreatitis. Hypertriglyceridemia itself is usually symptomless, although high levels may be associated with skin lesions known as xanthomas. The diagnosis is made on blood tests, often performed as part of screening. Once diagnosed, other blood tests are usually required to determine whether the raised triglyceride level is caused by other underlying disorders ("secondary hypertriglyceridemia") or whether no such underlying cause exists ("primary hypertriglyceridaemia"). There is a hereditary predisposition to both primary and secondary hypertriglyceridemia (Nelson ;2000).

Weight loss and dietary modification may be effective in hypertriglyceridemia. The decision to treat hypertriglyceridemia with medication depends on the levels and on the presence of other risk factors for cardiovascular disease. Very high levels that would increase the risk of pancreatitis is treated with a drug from the fibrate class.

Niacin and omega-3 fatty acids as well as drugs from the statin class may be used in conjunction, with statins being the main drug treatment for moderate hypertriglyceridemia where reduction of cardiovascular risk is required(Nelson ;2000).

Signs and symptoms of hypertriglyceridemia :-

Most people with elevated triglycerides experience no symptoms. Some forms of primary hypertriglyceridemia can lead to specific symptoms: both familial chylomicronemia and primary mixed hyperlipidemia include skin symptoms (eruptive xanthoma), eye abnormalities (lipemia retinalis), hepatosplenomegaly (enlargement of the liver and spleen), and neurological symptoms. Some experience attacks of abdominal pain that may be mild episodes of pancreatitis. Eruptive xanthomas are 2–5 mm papules, often with a red ring around them, that occur in clusters on the skin of the trunk, buttocks and extremities. Familial dysbetalipoproteinemia causes larger, tuberous xanthomas; these are red or orange and occur on the elbows and knees. Palmar crease xanthomas may also occur. Acute pancreatitis occurs in people whose triglyceride levels are above 1000 mg/dl (11.3 mmol/l). Hypertriglyceridemia is associated with 1–4% of all cases of pancreatitis. The symptoms are similar to pancreatitis secondary to other causes, although the presence of xanthomas or risk factors for hypertriglyceridemia may offer clues (Nelson ;2000).

Causes of hypertriglyceridemia :-

- High carbohydrate diet
- Idiopathic (constitutional)
- Obesity
- Diabetes mellitus and insulin resistance - it is one of the defined components of metabolic syndrome (along with central obesity, hypertension, and hyperglycemia)
- Excess alcohol intake
- renal failure, Nephrotic syndrome
- Genetic predisposition; some forms of familial hyperlipidemia such as familial combined hyperlipidemia i.e. Type II hyperlipidemia

- Lipoprotein lipase deficiency - Deficiency of this water soluble enzyme, that hydrolyzes triglycerides in lipoproteins, leads to elevated levels of triglycerides in the blood.
- Lysosomal acid lipase deficiency or Cholesteryl ester storage disease
- Certain medications e.g. isotretinoin, estrogen, hydrochlorothiazide diuretics, beta blockers, protease inhibitors
- Hypothyroidism (underactive thyroid)
- Systemic Lupus Erythematosus
- Glycogen storage disease type 1.
- Propofol
- HIV medications (Nelson ;2000).

1.2.2.3 Lipoprotein

A **lipoprotein** is a biochemical assembly that contains both proteins and lipids. The lipids or their derivatives may be covalently or non-covalently bound to the proteins. Many enzymes, transporters, structural proteins, antigens, adhesins and toxins are lipoproteins. Examples include the high density (HDL) and low density (LDL) lipoproteins which enable fats to be carried in the blood stream, the transmembrane proteins of the mitochondrion and the chloroplast, and bacterial lipoproteins function of lipoprotein particles is to transport water-insoluble lipids (fats) and cholesterol around the body in the blood (Gorge N;2014).

All cells use and rely on fats and, for all animal cells, cholesterol as building blocks to create the multiple membranes which cells use to both control internal water content, internal water soluble elements and to organize their internal structure and protein enzymatic systems (Gorge N;2014).

The lipoprotein particles have hydrophilic groups of phospholipids, cholesterol and apoproteins directed outward. Such characteristics make them soluble in the salt water-based blood pool. Triglyceride-fats and cholesterol esters are carried internally, shielded from the water by the phospholipids monolayer and the apoproteins(Gorge ;2014).

The interaction of the proteins forming the surface of the particles with (a) enzymes in the blood, (b) with each other and (c) with specific proteins on the surfaces of cells determine whether triglycerides and cholesterol will be added to or removed from the lipoprotein transport particles.

Regarding atheroma development and progression as opposed to regression, the key issue has always been cholesterol transport patterns, not cholesterol concentration itself (Gorge ;2014).

Transmembrane lipoproteins

The lipids are often an essential part of the complex. even if they seem to have no catalytic activity by themselves. To isolate transmembrane lipoproteins from their associated membranes, detergents are often needed (Gorge ;2014).

Classification of lipoprotein :-

Lipoproteins may be classified as follows, listed from larger and less dense ones to smaller and denser ones. Lipoproteins are larger and less dense, if they consist of more fat than of protein.

- Chylomicrons carry triglycerides (fat) from the intestines to the liver, skeletal muscle, and to adipose tissue.
- Very low density lipoproteins (VLDL) carry (newly synthesized) triacylglycerol from the liver to adipose tissue.
- Intermediate density lipoproteins (IDL) are intermediate between VLDL and LDL. They are not usually detectable in the blood.
- Low density lipoproteins (LDL) carry cholesterol from the liver to cells of the body. LDLs are sometimes referred to as the "bad cholesterol" lipoprotein.
- High density lipoproteins (HDL) collect cholesterol from the body's tissues, and bring it back to the liver. HDLs are sometimes referred to as the "good cholesterol" lipoprotein (Gorge ;2014).

Alpha and beta

It is also possible to classify lipoproteins as "alpha" and "beta", according to the classification of proteins in serum protein electrophoresis. This terminology is sometimes used in describing lipid disorders such as Abetalipoproteinemia (Gorge ;2014).

Lipoprotein Metabolism

The handling of lipoproteins in the body is referred to as lipoprotein metabolism. It is divided into two pathways, exogenous and endogenous, depending in large part on whether the lipoproteins in question are composed chiefly of dietary (exogenous) lipids or whether they originated in the liver (endogenous) (Gorge ;2014).

Exogenous pathway

Epithelial cells lining the small intestine readily absorb lipids from the diet. These lipids, including triglycerides, phospholipids, and cholesterol, are assembled with apolipoprotein B-48 into chylomicrons. These nascent chylomicrons are secreted from the intestinal epithelial cells into the lymphatic circulation in a process that depends heavily on apolipoprotein B-48. As they circulate through the lymphatic vessels, nascent chylomicrons bypass the liver circulation and are drained via the thoracic duct into the bloodstream (Gorge ;2014).

In the bloodstream, HDL particles donate apolipoprotein C-II and apolipoprotein E to the nascent chylomicron; the chylomicron is now considered mature. Via apolipoprotein C-II, mature chylomicrons activate lipoprotein lipase (LPL), an enzyme on endothelial cells lining the blood vessels. LPL catalyzes the hydrolysis of triacylglycerol (i.e. glycerol covalently joined to three fatty acids) that ultimately releases glycerol and fatty acids from the chylomicrons. Glycerol and fatty acids can then be absorbed in peripheral tissues, especially adipose and muscle, for energy and storage (Gorge ;2014).

The hydrolyzed chylomicrons are now considered chylomicron remnants. The chylomicron remnants continue circulating until they interact via apolipoprotein E

with chylomicron remnant receptors, found chiefly in the liver. This interaction causes the endocytosis of the chylomicron remnants, which are subsequently hydrolyzed within lysosomes. Lysosomal hydrolysis releases glycerol and fatty acids into the cell, which can be used for energy or stored for later use (Gorge ;2014).

Endogenous pathway

The liver is another important source of lipoproteins, principally VLDL. Triacylglycerol and cholesterol are assembled with apolipoprotein B-100 to form VLDL particles. Nascent VLDL particles are released into the bloodstream via a process that depends upon apolipoprotein B-100.

As in chylomicron metabolism, the apolipoprotein C-II and apolipoprotein E of VLDL particles are acquired from HDL particles. Once loaded with apolipoproteins C-II and E, the nascent VLDL particle is considered mature.

Again like chylomicrons, VLDL particles circulate and encounter LPL expressed on endothelial cells. Apolipoprotein C-II activates LPL, causing hydrolysis of the VLDL particle and the release of glycerol and fatty acids. These products can be absorbed from the blood by peripheral tissues, principally adipose and muscle. The hydrolyzed VLDL particles are now called VLDL remnants or intermediate density lipoproteins (IDLs). VLDL remnants can circulate and, via an interaction between apolipoprotein E and the remnant receptor, be absorbed by the liver, or they can be further hydrolyzed by hepatic lipase.

Hydrolysis by hepatic lipase releases glycerol and fatty acids, leaving behind IDL remnants, called low density lipoproteins (LDL), which contain a relatively high cholesterol content. LDL circulates and is absorbed by the liver and peripheral cells. Binding of LDL to its target tissue occurs through an interaction between the LDL receptor and apolipoprotein B-100 or E on the LDL particle. Absorption occurs through endocytosis, and the internalized LDL particles are hydrolyzed within lysosomes, releasing lipids, chiefly cholesterol (Gorge N;2014).

1.2.3 Pregnancy and body Mass index (BMI):-

BMI is the relationship between height and weight and Is used to determine whether we are underweight, just right, Overweight or obese. BMI is recorded in pregnancy notes and is a useful measurement for pregnancy(Nelson ;2000).

- BMI less than 19.8= Underweight
- BMI 19.9 - 25.9= healthy weight
- BMI 26 - 29.9= mildly overweight
- BMI 30 - 35= moderately overweight
- BMI over 35= overweight

Weight gain

The amount of weight a woman may gain in pregnancy can vary a great deal. Only some of it is due to increased body fat – the baby, placenta, amniotic fluid and increases in maternal blood and fluid retention all contribute. However, must be encouraged to keep weight gain to the Institute of Medicine (IOM) guidelines (Nelson Y; 2000)

- BMI of less than 19.8=12.5 - 18kg (28 - 40lbs)
- BMI 19.9 - 25.9=11.5 - 16kg (25 - 35lbs)
- BMI 26 - 29 =7 - 11.5kg (15 - 25lbs)
- BMI 30 or more=6kgs (15lbs) or less(Nelson; 2000).

1.3 Rationale

Lipid is essential for the synthesis of many hormones in human body and plays vital roles in many bioactivities and reactions.

Despite the obvious importance of lipid, elevation over the normal levels can lead to drastic consequences, such as heart diseases.

Many factors that increase or decrease its serum level affect lipid. Therefore these factors can be harmful or beneficial accordingly.

Pregnancy is one of the factors that can affect the physiological state of the body, thus it might have some sort of affect on lipid level. Determination of changes in lipid level at different stage of pregnancy gives clear picture to deal with pregnant women.

1.4 Objectives

1.4.1 General objective:

To assessment serum lipid profile in pregnant women

1.4.2 Specific objectives:

To measure serum Cholesterol, Triglyceride , HDL and LDL in pregnant women.

To compare lipid profile during trimesters of pregnancy.

To determine the effect of metabolic change on level of lipid in pregnant women.

2. Materials and Methods

2.1 Study design:

This is a Cross sectional study during the period March- June 2014

2.2 Study area:

The study was carried out in Sidg Al-Tom Health Center Al-Haj Yousif Khartoum state-Sudan

2.3 Sample size :

100 pregnant women

2.4 Inclusion criteria:

pregnant women with normal uncomplicated pregnancy were included in this study.

2.5 Exclusion criteria:

Pregnant with hypertension, diabetes, heart diseases or other disease that may affect parameters and study.

2.6 Ethical consideration:

Approval was taken from faculty management Verbal consent was taken from subjects under study. After conducting the test, every subject was shown her result

2.7 Methods and Tools:

2.7.1 Data collection:

Data were collected using a structure interviewing questionnaire, carefully constructed to collect and maintain all valuable information concern each case examined. Demographic data were collected from every sample investigated .

2.7.2 Reagent preparation, storage and stability:

Biomed lipid profile reagents are supplied ready-to-use and stable up to the expiry date labeled on the bottles. Once opened vial is stable for 3 months at 2-8°C

2.7.3 Instruments:

-Automatic micropipette.

-Centrifuge.

-Spectrophotometer.

2.7.4 Specimen collection and preservation:

2.5ml of venous blood collected in plain container immediately centrifuge at 3000 rpm for 5 minutes to separate serum for investigation of Cholesterol, Triglyceride, HDL and LDL and stored at -21⁰C until used.

2.7.5 Data analysis:

The data was analyzed using statistical package for social science (SPSS).

2.8 Lipid profile:

2.8.1 Cholesterol estimation:

Principle :

Under the action of Cholesterol esterase (CHE) cholesterol esters resolve in cholesterol and fatty acids. Cholesterol Oxidase (CHOD) oxidize the above mentioned cholesterol together with the loose cholesterol releasing Cholesterol-3-one and hydrogen peroxide. In presence of Peroxidase(POD), the released hydrogen peroxide reacts with a phenol substitute and 4-aminoantipyrine to form a red compound. The intensity of the red color produced is directly proportional to the total cholesterol in the sample when read at 520 nm.

Procedure:

1000 ul was taken from cholesterol reagent in three glass tubes. Then added 10ul from standard in one tube and 10ul from sample. Tube was mixed, incubate for 10 min at 25°C and color of sample and standard was read against reagent blank.

Calculation:

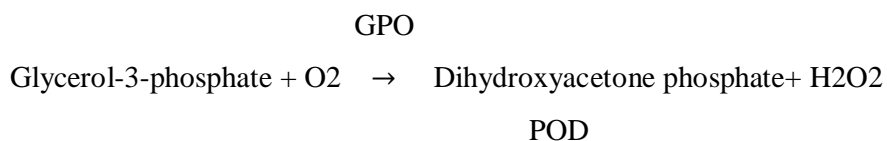
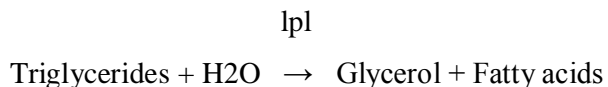
Concentration of sample = $\frac{\text{Absorbance of sample}}{\text{Absorbance of STD}} \times \text{concentration of STD}$

Absorbance of STD

2.8.2 Triglyceride estimation:

Principle

For the enzymatic determination of Triglycerides according to the following reaction:



The intensity of the red color produced is directly proportional to Triglycerides in the sample.

Procedure:

From triglyceride reagent 1000ul was taken and putted into three glass tube .then added 10 ul from standard in one tube and 10ul from serum sample in another tube then was mixed, incubated for 10min at room temperature (25°C) and was read sample and standard extinction. .

Calculation:

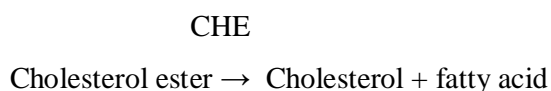
$$\text{Concentration of sample} = \frac{\text{Absorbance of sample}}{\text{Absorbance of STD}} \times \text{concentration of STD}$$

2.8.3 HDL estimation:

Principle:

HDL-Cholesterol is obtained through selective precipitation of LDL and VLDL lipoproteins, thus

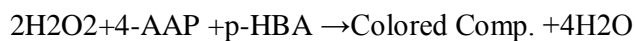
HDL lipoproteins remain in solution. HDL-Cholesterol in supernatant is treated as a sample for cholesterol assay according to the following reaction:



CHOD



POD



Procedure:

Precipitation:

500 ul from precipitant reagent was added to 200 ul of serum sample then Mixed and allowed to stand for 10 minutes at 25°C . Centrifuge for 10 minutes at 4000 rpm. Separated of the clear supernatant within two hours and determine the cholesterol content by the CHOD-PAP method.

1000 ul was taken from cholesterol reagent in two tubes , in one tube we added 100ul from supernatant and 100 ul from distilled water to another tube. Mixed, and incubated for 10 min. at 20-25°C. Measure absorbance of specimen (Specimen) against reagent blank within 30 minutes.

Calculation:

Concentration of HDL cholesterol (mg/dl) in supernatant = Absorbance of sample \times Factor

2.9.4 LDL

Calculation:

LDL Cholesterol (mg/dl) = total cholesterol – triglyceride \div 5 – HDL cholesterol

3. Results

This study was conducted on 100 apparently pregnant women on sidig altom health center at sharg alneel-khartoum state.

The results obtained after conducting the appropriate test were as following:

Table 3.1 showed that mean and standard deviation of total cholesterol and triglyceride in pregnant women (164 ± 31 mg/dl) (130 ± 38 mg/dl) with p-value(0.05) (0.9) respectively.

Table 3.2 showed that mean and standard deviation of HDL and LDL in pregnant women (55 ± 18 mg/dl) (81 ± 31 mg/dl) with p-value (0.07) (0.6) respectively

Figure (3.1): scatter plot between pregnancy and Serum total cholesterol negative correlation (P.value =0.9 $r = - 0.03$).

Figure (3.2): scatter plot between pregnancy and triglyceride no correlation (P.value =0.9 $r = 2.8$).

Figure (3.2): scatter plot between pregnancy and HDL negative correlation (P.value =0.07 $r = - 0.03$).

Figure (3.2): scatter plot between pregnancy and LDL no correlation (P.value =0.6 $r = - 0.002$)

Table 3.1 the mean and standard deviation of total cholesterol and triglyceride in pregnant women

parameter	Mean \pm SD	P-value
Cholesterol	164 \pm 31	0.05
Triglyceride	130 \pm 38	0.9

- Correlation test was used
- P value considered significant level 0.05

Table 3.2 the mean and standard deviation of HDL and LDL in pregnant women

parameter	Mean \pm SD	p-value
HDL	55 \pm 18	0.07
LDL	81 \pm 31	0.6

- Correlation test was used
- P value considered significant level 0.05

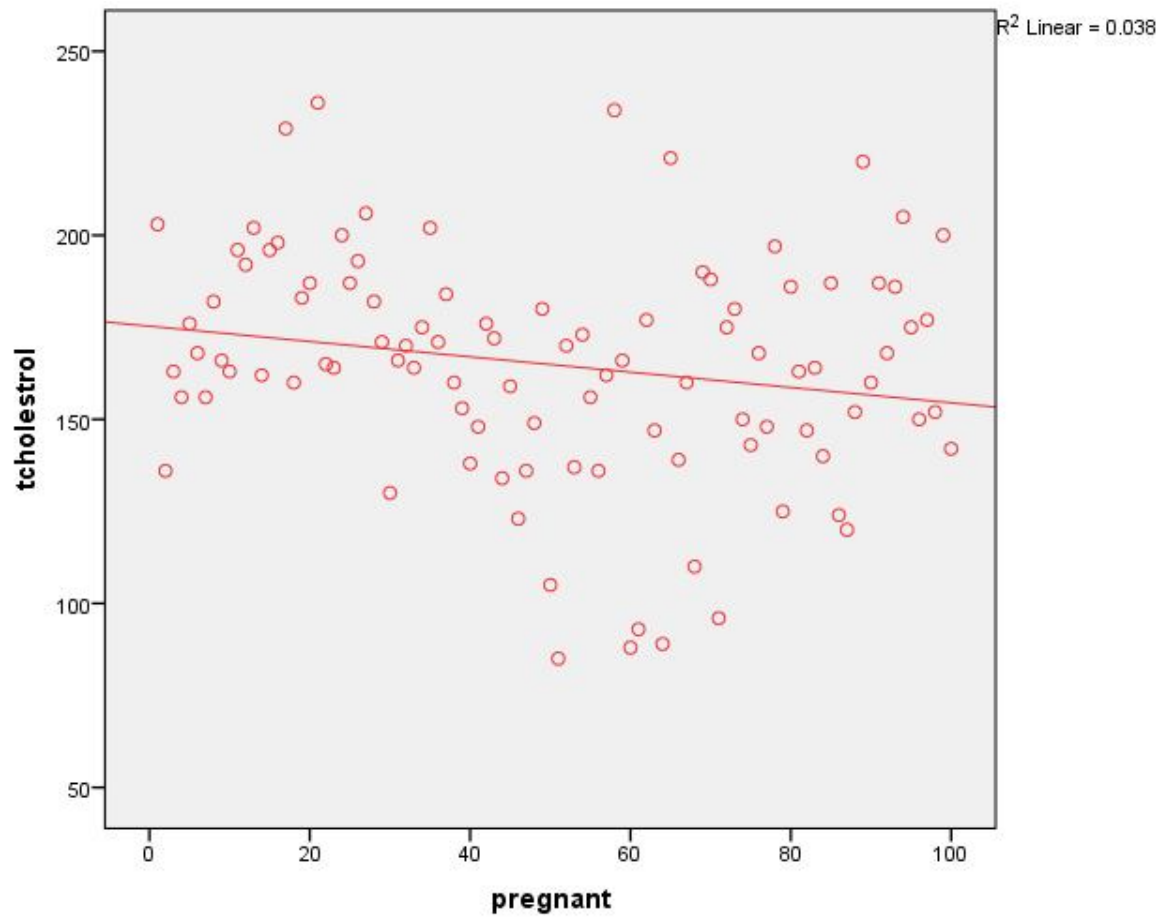


Figure (3.1): scatter plot between pregnancy and Serum total cholesterol negative correlation (P.value =0.9 $r = - 0.03$)

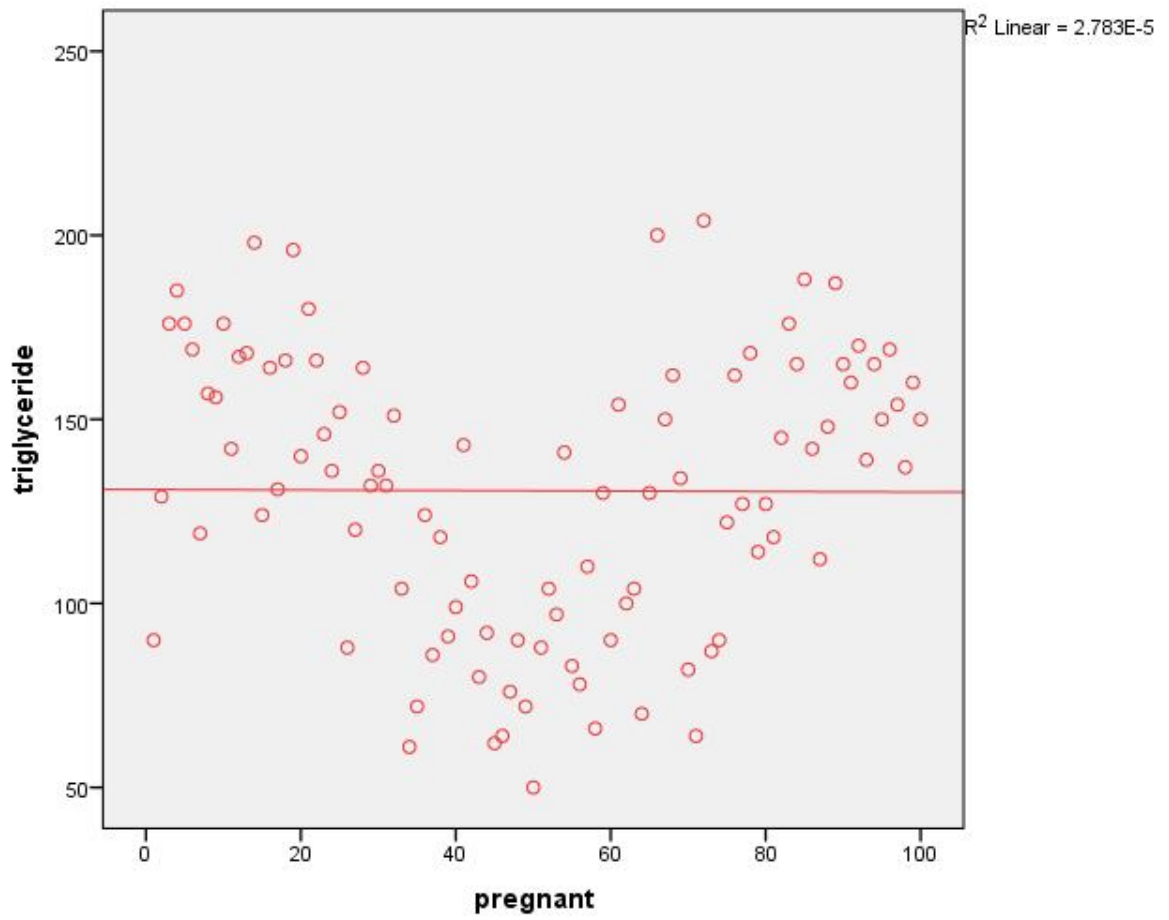


Figure (3.2): scatter plot between pregnancy and triglyceride no correlation (P.value =0.9 r = 2.8)

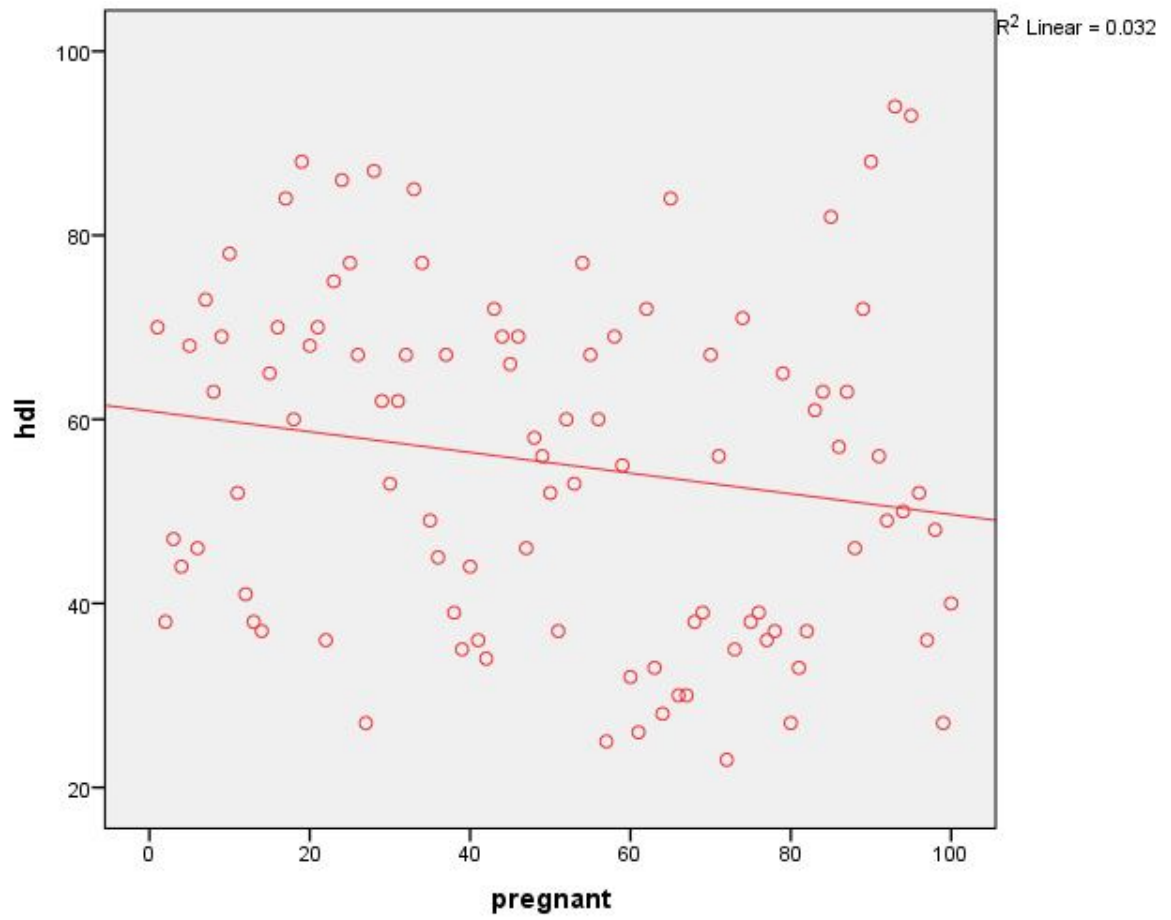


Figure (3.3): scatter plot between pregnancy and HDL negative correlation (P.value =0.07 $r = -0.03$)

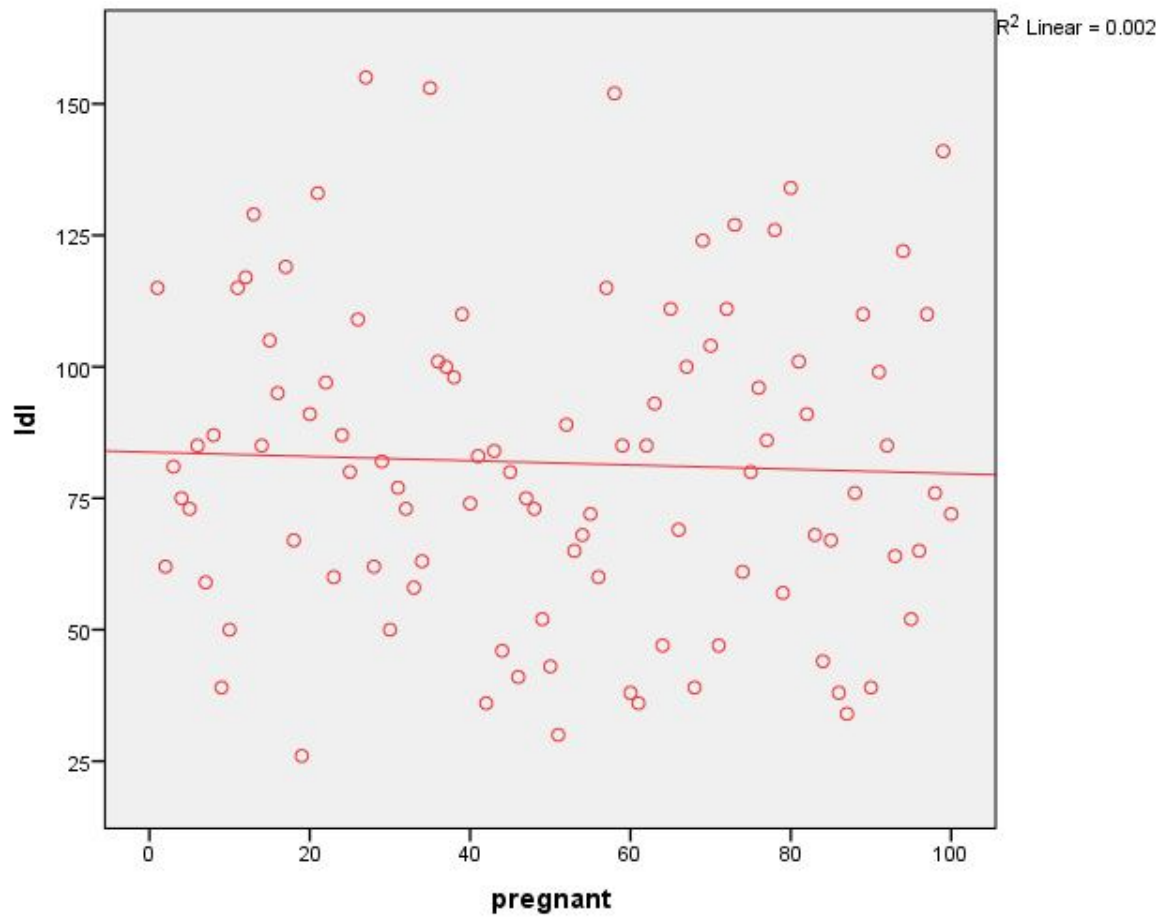


Figure (3.2): scatter plot between pregnancy and LDL no correlation (P.value = 0.6 $r = -0.002$)

4.1 Discussion

This study was carried out in Khartoum state (Sudan) on 100 pregnant women to assessment of lipid profile during pregnancy .

Through finding of this research we found the following:

Total cholesterol decrease during pregnancy. This result also report in study of E Koukkou *et al* that found African/Afro-Caribbean pregnant women had lower serum concentrations of total cholesterol .

this result is differ from result obtained in study conducted by P Brizzi *et al* who found increase in total cholesterol during pregnancy in all women.

This result different from result obtain in studies of Festus O *et al* who found increase in triglyceride with p-value (0.05) .

Also this study found decrease HDL during pregnancy. This result same to result found in studies of Abdelhai A *et al* who found lowering in HDL in pregnant women ,but differ from study obtained by J C Mazurkiewicz *et al* who found increase in HDL with p-value(0.03).

Also this study found LDL not affected during pregnancy. This result different from result obtain from study of Also by J C Mazurkiewicz *et al* who found The pregnant women had significantly higher concentrations LDL during pregnancy.

Other difference in results found in study did by E Koukkou *et al*. That said Lipoprotein was not significantly high ($p > 0.05$) in the first trimester but became significant ($p < 0.05$) in the second and third trimester when compared with the control.

This result also differ from result obtained in study conducted by Deepak Parchw *et al* that found a progressive rise was observed in serum LDL-C as pregnancy advances.

4.2 Conclusion

In this study :

- Serum total cholesterol and HDL decrease during pregnancy
- Triglyceride and LDL not affect during pregnancy
- Increase in BMI lead to increase of total cholesterol and LDL
- Triglyceride and HDL not affect by BMI

4.3 Recommendation

From the result obtained from this study, we may recommend the following:-

- Monitoring lipid profile during pregnancy.
- the study was conducted on different people at every stage of pregnancy, therefore we recommend for more reliable and accurate results, further studies should be carried out with Follow-up of the same people in the three stages of pregnancy and other variable should be considered, such as the life style and level of activity.

REFRANCE

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Appendix

Cholesterol:

REAGENTS COMPOSITION:

R1

Cholesterol Standard	200mg/dl
----------------------	----------

R2

Good's Buffer	100mmol/L
---------------	-----------

Cholesterol esterase	300u/L
----------------------	--------

Cholesterol oxidase	1500u/L
---------------------	---------

Peroxidase	5500u/L
------------	---------

4-AAP	1mmol/L
-------	---------

Phenol derivatives	5mmol/L
--------------------	---------

Triglyceride

REAGENTS COMPOSITION:

R1 Standard

Triglycerides Standard	200mg/dl
	2.28mmol/l

R2

Good's Buffer	100mmol/L
---------------	-----------

Magnesium Chloride	15mmol/L
--------------------	----------

ATP (Adenosina-5-Triphosphate)	4mmol/L
--------------------------------	---------

4AAP (4-Aminoantipyrine)	1mmol/L
--------------------------	---------

4-CHLOROPHENOL	0.1mmol/L
LPL (Lipoprotein Lipase)	2500U/L
GK (Glycerol kinas)	1000U/L
GPO (Glycerol-3-phosphate oxidase)	5500U/L
POD (Peroxidase)	1800U/L

HDL

Precipitating Reagent:

Phosphotungstic acid	0.55mM
Magnesium Chloride	25mM

Cholesterol reagent

Ranges of lipid profile

Cholesterol	up to 5mmol/L
	200mg/dl

Triglyceride:

Men	40 – 160 mg/dl
	0.45 – 1.82 mmol/l

Women	35 – 135 mg/dl
	0.4- 1.54 mmol/l

HDL:

Women	30-85 mg/dl
-------	-------------

Men	30-70 mg/dl
LDL	
Adults	66-178 mg/dl

جامعة السودان للعلوم والتكنولوجيا

كلية الدراسات العليا-مختبرات طبية

قسم الكيمياء السريرية

Questionnaire

Association between pregnancy and level of lipid profile

Date serial number.....

Age Name.....

Stage of pregnancy.....

Weight..... Height.....

Body mass index.....

Do you suffer from any of the following disease?

Hypertension Yes ☐ No ☐

Diabetes Yes ☐ No ☐

Hypothyroidism Yes ☐ No ☐

Hyperthyroidism Yes ☐ No ☐

Result of:

Total cholesterol

Triglyceride.....

HDL.....

LDL.....

