# Prognosis of Different Disease Due to Alteration in Lipid Profile

**Thesis Submitted to**

**Lovely Professional University, Punjab**
**in partial fulfilment of the requirements**
**for the degree of**
**Master of Science**
**In Clinical Biochemistry**


**Submitted by**

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**May, 2015**

# *RECOMMENDATION*

This is to certify that ***Ms. Kirti*** bearing regd. No. ***11311129*** has completed her M.Sc project titled ***"Prognosis of different diseases due to alteration in Lipid Profile"*** under my guidance and supervision. This report is record of the candidate own work carried out by her under my supervision. I certify that the matter embodied in this report is original and has been not submitted anywhere for the reward of any other degree.

Date: *Dr Pranav Kumar Prabhakar*

Place: LFAMS, LPU Punjab

# *CERTIFICATE*

This is to certify that Ms. Kirti bearing regd. No. 11311129 has completed her MSc project titled under my guidance and supervision. This report is record of the candidate own work carried out by him under my supervision. The matter embodied in this report is original and has been not submitted for the reward of any other degree.

The project report is fit for the submission and the partial fulfilment of the conditions for the award of MSc. Clinical Biochemistry.

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Place: LPU, Punjab (Miss. Kirti)

Date:

# *Prognosis of different Diseases due to Alteration in Lipid Profile*

## *Abstract*

**Background**: I work on the topic of "***Prognosis of different diseases due to Alteration in Lipid Profile".*** Alteration of lipid profile in different diseases like Coronary artery disease, obesity and stroke have been estimated from values of lipid profile. TG affect on HDL cholesterol, TG is the good marker for obesity and the ratio of total to HDL cholesterol is a more specific marker of CAD than is LDL cholesterol. TC is best marker for Stroke.

**Objective:** The objective was to evaluate the plasma lipid abnormalities in different disease, and define the role of age and sex.

**Methods:** Performed an analysis of 150 patients, and from these 74 patients were selected for further studies that have abnormal lipid profile.

**Results**: for this study took the data of 74 patients who have abnormal lipid profile. Patient's age range was more than 50 and less than 50 years. Obesity was present in 44% of patients. Men were more affected with obesity, according to the age and according to the sex. CAD was present in 29% of patients. Patients who have age less than 50 were more affected. According to the sex male and female were equally affected. Stroke was present in 25% of patients. According to age , more than 50 year age patient have stroke. According to the sex significantly there is no much difference.

**Conclusion:** Conclusion of the study is mostly patients were affected with Obesity. The main cause of this, less physical activity, Food Habits, and their life style. They have to do regular physical exercise twice in a day.

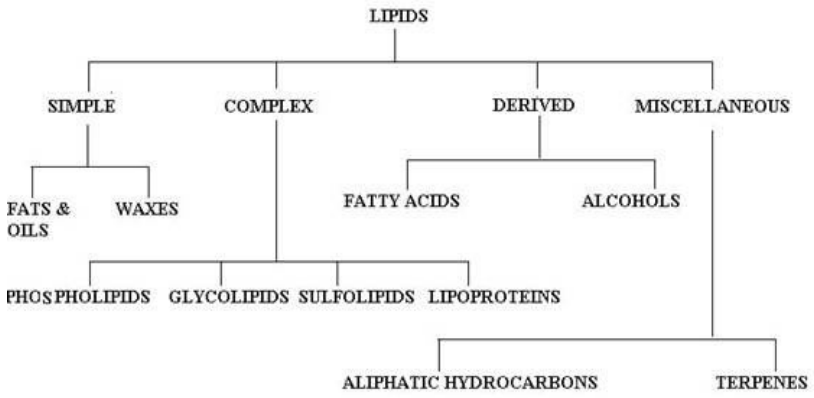
# *Chapter 1*

# *Introduction*

## *1. Introduction of lipids*

Lipids are organic compounds that are poorly soluble in water but soluble in organic solvents like ether, chloroform, acetone, benzene. Lipids play a critical role in important physiological function in human body. Lipids are large diverse group of organic compounds including fatty acids and their derivatives, tarpenes, steroids and bile acids. Steroid hormones serve as chemical messengers between Cells, tissues and organs. Lipids are structural components in cell and are involved in metabolic and hormonal pathways [Gotto A, Pownall H 1999; 16]. Lipids are group of hydrophobic or amphilic molecules. These molecules form structures such as vesicles, liposome's, or membranes in an aqueous environment. Most lipids are amphipatic which interact with other molecules with aqueous solvents by hydrogen bonding and electrostatic interaction. Lipids classified on the bases on their different chemical properties, their chemical composition and their building blocks, including their ability for saponification. We can say sapnofication is a technique which has ability of lipids to be hydrolyzed by basic solutions into compounds such as glycerol and fatty acids. On the basis of their chemical composition, lipids are classified into two simple lipids and complex lipids. Simple lipids are defined as that hydrolysis yield at most two type of primary product per mole. On the other way complex lipids yield three or more primary hydrolysis products per mole (e.g. glycerol, FAs, and sugar). Lipids are divided into two ketoacyl and isoprene groups one the bases of their building blocks. On the basis of their chemically functional backbone lipids are divided into eight categories FAs, Glycerolipids, Sphingolipids, Glycerophospholipids, Saccharolipids and polyketides. These eight compounds are derived from ketoacyl subunits and sterol lipids and phenol lipids derived from isoprene subunits [Boelsma E, Hendriks HF etal, 2001].

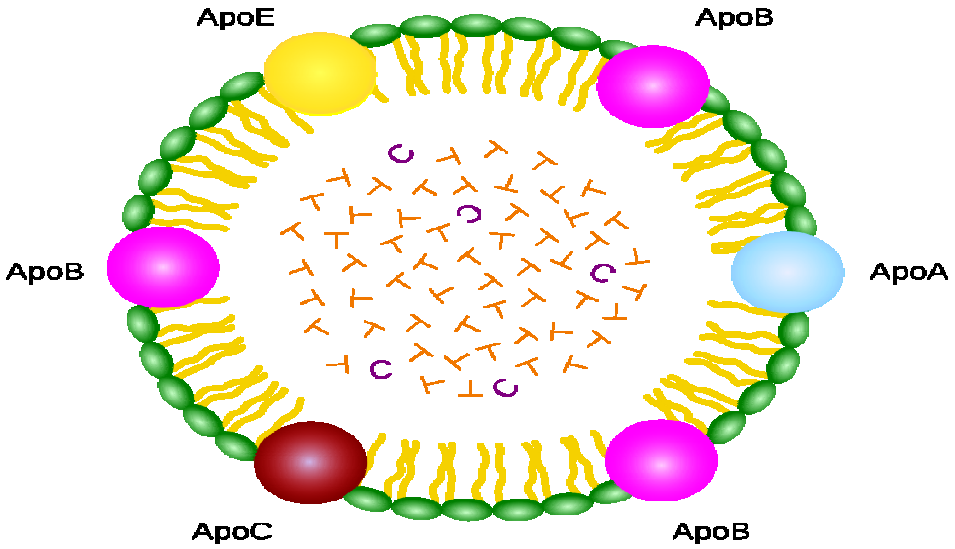
## *1.1 Classification of Lipids*




The image is described that lipids have mainly four classes. This is divided further on the basis of their nature.

## *1.2 Lipoproteins*

Lipoproteins are biochemical transporters, Lipoproteins are composed of protein and lipids. Lipoproteins help all lipids derived from food and synthesized in specific organ to be transported through the body by circulatory system. Lipoproteins are an energy supplier which is used by intestine and liver, they supply energy to other tissues. Intestine and liver also use them as structural materials in their membrane. [Boelsma E, Hendriks HF etal, 2001] There is an amphipatic monolayer of lipids in the layer of intestine and liver, this layer is composed of polar head groups of phospholipids, and free cholesterol with Apo proteins which is contact with an aqueous environment and cover the hydrophobic part of their structure the non polar part of layer consist of TAGs and cholesterol esters. Lipoproteins are divided into five major classes [Boelsma E, Hendriks HF etal, 2001].




Structure is show that Lipoproteins have Apo proteins in hydrophobic part and TGs and Cholesterol esters present in the core of the Lipoprotein.

### *1.3Apo lipoproteins*

Apo lipoproteins are the polypeptide found in lipoproteins. Apo lipoproteins are commonly using the abbreviation Apo. Different type of apolipoprotein are A, B, C and E. They have role in transport of Chylomicrons, triglycerides, cholesterol, and fatty acids.

## *1.4 Classification of Lipoproteins*

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**Table I.** Lipoprotein Classification

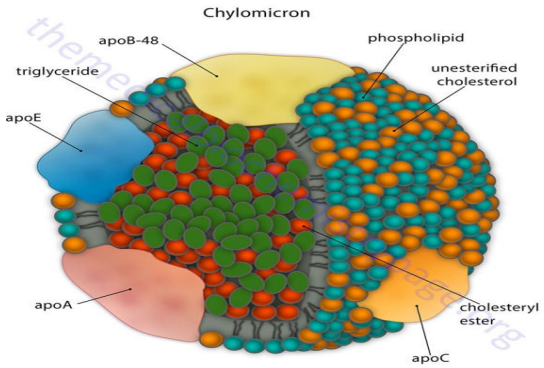
| LIPOPROTEIN | MAJOR LIPID COMPONENT | MAJOR APOLIPOPROTEINS | SOURCE |
|---|---|---|---|
| Chylomicrons | TG | ApoA-I, A-II, A-IV; ApoC-I, C-II, C-III; ApoB-48; ApoE | Intestine |
| Very low-density lipoprotein (VLDL) | TG | ApoB-100; ApoC-I, C-II, C-III; ApoE | Liver |
| Intermediate-density lipoprotein (IDL) | CE | ApoB-100; ApoE, ApoC | Catabolism of VLDL |
| Low-density lipoprotein (LDL) | CE | ApoB-100 | Catabolism of IDL |
| High-density lipoprotein (HDL) | CE, PL | ApoA-I, A-II, A-IV; ApoC-I, C-II, C-III; ApoE | Liver, intestine, other |

TG=triglyceride; CE=cholesterol ester; PL=phospholipid
Data derived from Dominiczak MH. Apolipoproteins and lipoproteins in human plasma. In: Rifai N, Warnick GR, Dominiczak MH, eds. *Handbook of Lipoprotein Testing*. Washington, DC: AACC Press; 1997:1–17 and Gotto A, Pownall H. *Manual of Lipid Disorders: Reducing the Risk for Coronary Heart Disease*. 2nd ed. Baltimore, MD: Williams & Wilkins; 1999:2–10.

Source: Prog Cardiovasc Nurs © 2003 Le Jacq Communications, Inc.

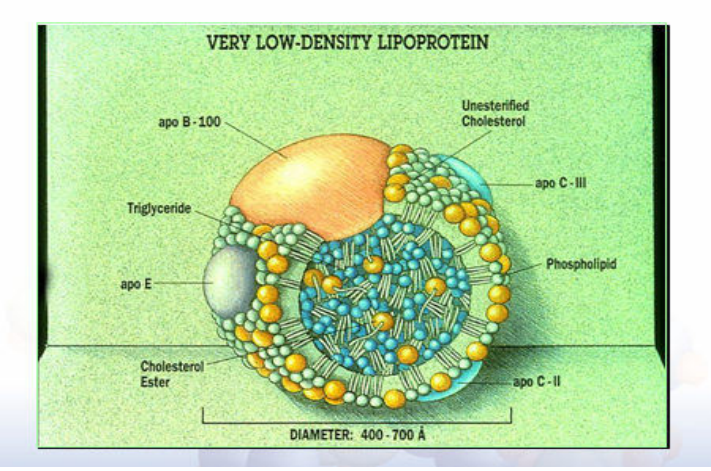
### ❖ *Chylomicrons*

Chylomicrons are the largest lipoproteins. The diameter of Chylomicrons are 75-600nm.It is formed in the intestinal cell wall by dietary fats and cholesterol. Chylomicrons contain 99% of total lipid and 2% cholesterol.




### ❖ *VLDL*

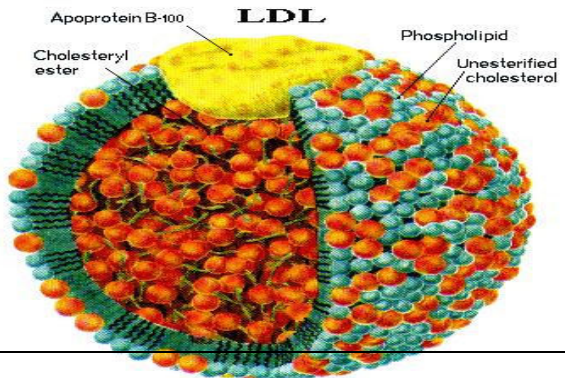
It is lipoprotein synthesis by liver transport endogenous lipid from the liver to cells. Itcontains91%oftotallipids.




❖ ***IDL*** Intermediate density lipoproteins which are formed during the conversion of VLDL to LDL.IDL contain 20% of proteins 7% of free cholesterol.

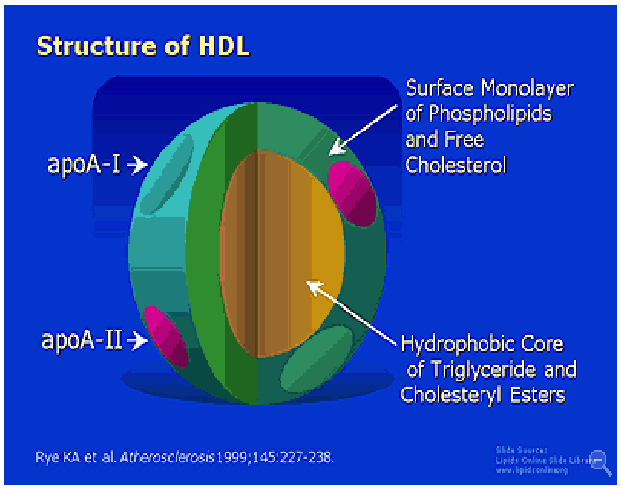
❖ ***LDL***

LDL is rich particles of cholesterol. Near about 70% of plasma cholesterol occurs in this form. It contains 80% of total lipids.



❖ ***HDL***

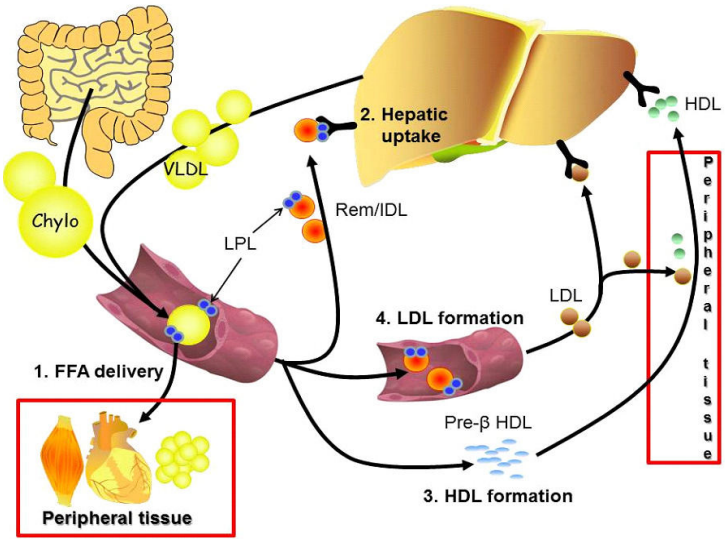
HDL is smallest class of lipoproteins.HDL removes the excess cholesterol from cell. It contains 44% of total lipids and 7% of cholesterol.HDL also contain Apo1 and Apo2 proteins. [Joint British Recommendation 2000]




## *2. MECHANISM OF LIPOPROTIENS METABOLISM*

There are mainly three pathways by which transportation of lipids occur in the body.

The pathways are exogenous pathways, the endogenous pathway, and pathway of reverse cholesterol transport. [Vaughn G. 1999, Rifai N, Warnick GR etal, 1997]




## *2.1Exogenous Pathway*

When we take diet digestion occur than absorption of dietary fat, TG and cholesterol is taking place. These are packaged to form Chylomicrons in the epithelial cells of the intestine through the intestinal lymphatic system. Chylomicrons circulate by circulating system. Chylomicrons interact the capillaries of Adipose tissue, and muscle cells releasing TG to the adipose tissue to be stored for the availability of body's energy. Free fatty acids are released with the hydrolization of TG by LPL enzyme. Some components of the Chylomicrons are going back in to the other lipoproteins, like endogenous pathway.

## *2.2Endogenous Pathway*

In this pathway now liver synthesizing lipoproteins.TG and cholesterol ester are released by the liver and add into VLDL particles and it is released into the circulation. Know LPL is working on VLDL in tissues to release Fatty acids and glycerol. Fatty acids are divided according to body need Energy. Muscle cells taken up fatty acids and by the Adipose cells for storage. When the action of LPL is complete VLDL becomes a VLDL remnant. Now there is LDL receptor present by which liver taken up. Majority of VLDL remnants. The remnant which is remaining after this become LDL, it is necessary that some of particles of LDL have to reabsorb by liver with the action of LDL receptor. To form LDL hydrolization of IDL particles occur in the liver by hepatic-triglyceride lipase.LDL is smaller than IDL. Cholesterol is circulating in the body and the carrier for this circulating cholesterol is LDL. Cholesterol is used by extra hepatic cells for cell membrane and synthesis of steroid hormones. Most of the LDL particles are taken up by receptor of LDL which is present in liver. At the cellular level remaining LDL is removed by the pathways known as scavenger pathway. LDL is not present because it is taken up by receptors, free cholesterol is released and increase within the cells. LDL receptor become activate and take LDL and regulate plasma LDL concentration by several mechanisms, it also slow down the synthesis of hydroxi-3-methyglutaryl co-enzyme A (HMG-CoA) reductase. This is the enzyme which control the cholesterol synthesis, decrease the rate of new LDL receptor synthesis in the cells, than activation of enzyme occur like acyl-coenzyme A cholesterol acyltransferase,it break down free cholesterol in to cholesterol ester, and storing cholesterol in the cell [Genest J Jr. 1990].

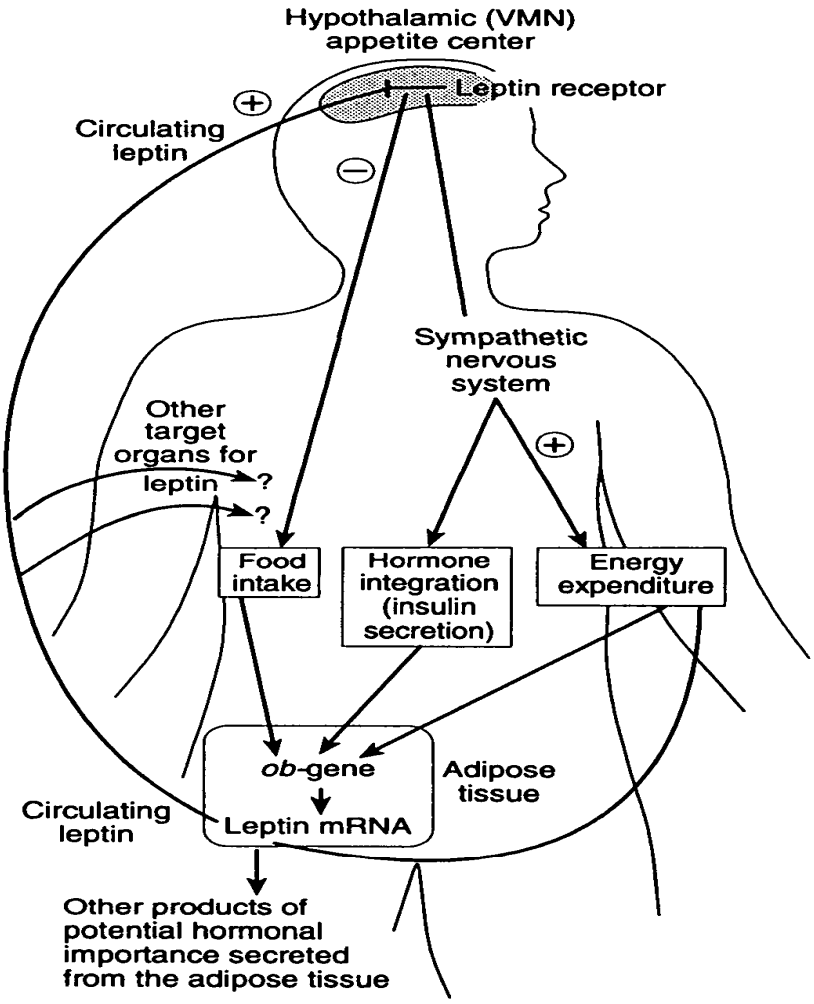
## *2.3REVERSE CHOLESTEROL*

It is working as a transporting system .In this transportation cholesterol is removed from the tissues and returned to the liver [Genest J Jr. 1990]. HDL is main lipoprotein which is known as key lipoprotein involved in reverse cholesterol transport and it transfer cholesteryl esters between lipoproteins [Gotto A, Pownall H. 1999]. HDL is the smallest lipoprotein. Liver and intestine secreted precursor particles also known as nascent HDL. Which formed HDL through a maturation process, and it is proceed through a series of conversions known as HDL cycle. It takes cholesterol from cell membranes and free cholesterol to the core of the HDL particle.HDL2 and HDL3 are sub-classes of HDL particles. The mechanism is not still clear by which the HDL delivers cholesterol esters to the liver, but the many mechanisms have been suggested. One of them is transfer of proteins by the action of cholesteryl ester. This transfer HDL in to a TG rich particle which react with hepatic triglyceride lipase. Cholesterol ester with rich HDL which is directly taken up by the receptor in the liver [Genest J Jr. 1990, Gotto A, Assmann G, 2000].

## *3. Alteration of Lipid profile*

***3.1Obesity*** Obesity is worldwide health problem which affecting increases number of people. Obesity is defined as excessive accumulation of energy in the form of body fat. The main cause of obesity is our eating habits. We have no control on food, it cause obesity. Especially foods which are rich in fats, overweight and obesity has a direct correlation with cardiovascular disease. In obesity always there is an increase in plasma triglycerides [13]. If we compare obese person and non obese person's serum value, then the obese person have high serum TNFa, triglyceride, and insulin and CRP levels than non-obese person. We can say that obesity is an increased risk for metabolic syndrome. On the basis of central distribution of body fat intra- abdominal fat is more risk factor than distribution of peripheral obesity. According to weight and height we estimate the body fat. The formula which we use

for the calculating body fat is known as BMI. By using the value of BMI we can see difference between overweight and obesity, it gives the information about body fatness [Hu, D., J. Hannah, et al., 2000, Lerario, D.D., S.G. Gimeno, et al 2002]. Our fat cells (adipocytes) produced leptin hormone. When the fat which is stored in adipocytes, when it increases, leptin is act as a lipostat. Leptin come in blood flow and gives signals to the brain that the body has enough to eat. mostly the people who are overweight, they have increase level of leptin in their blood stream, it indicating that other molecules of body also effect feeling of satiety and it is regulate the body weight [Goran, M.I. et al 1999] . Early onset obesity is controlled by gives the leptin injections subcutaneously; significantly it is helpful in weight loss without any alteration in energy expenditure. Decrease level of basal metabolic rate was counter balanced by during more physical activity [Misra, A., et al 2006]. Leptin is helpful in weight loss because it have suppressive effect on food intake [National Cholesterol Education Program, 1994]. Obesity effect on cardiovascular function and it also cause sleep breathing abnormalities. According to waist circumference and waist hip ratio HDL-C is decreased in obese person [National Cholesterol Education Program, 2002]. In obesity there is a metabolic defect occur. In obese person the level of free fatty acids become high because of insulin resistance, there is also increases the level of LDL cholesterol, VLDL and triglycerides also become high, but the level of HDL-cholesterol become low. In obesity when the level of free fatty acids is high in liver then there is over production of VLDL. Because the level of VLDL is known become high so the level of production of LDL is also become high. Via the sequence: VLDL production of intermediate density lipoprotein (IDL).then production of LDL [Wolf RN, Grundy SM. et al 1983]. The production of VLDL is related to insulin level in our body [Grundy SM, et al 1979]. And % of body fat [Equsa G, et al 1985].




In this picture it shows the role of the leptin. Adipose tissue secretes leptin. Leptin binds to db gene receptor.

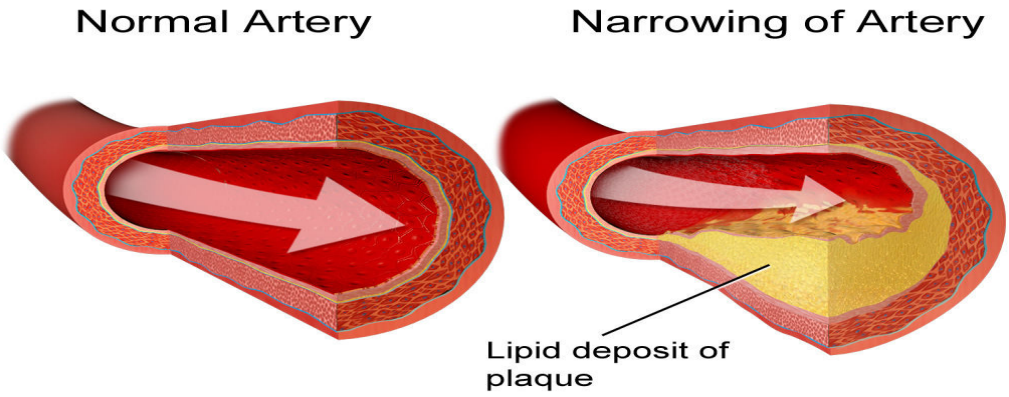
## *3.2Mechanism of Obesity*

### *Adiposity and TGs*

Lipoproteins in adiposity with hyperglyceridemia. TG level is used to measure the TG content of all lipoproteins which is circulating in our body, this have majority of apoprotein B

containing Lipoproteins. The main function of these particles they act like a primary carrier of TG in blood, they transport TG to the periphery. Lipoproteins which are containing Apo B are subdivided into two classes. Apo B48 and Apo B100. Apo B48 have Chylomicrons and the remnant of their particles taking from dietary lipid, it is compile with enterocytes of the gut. second Apo B is known as Apo B100, it have VLDL, intermediate density lipoprotein(IDL), also have LDL and a glycoprotein derivative LDL like particle, lipoprotein. Lipoproteins are formed in liver. Lipoproteins which are secreted from liver they are VLDL with postsecretory action of various Lipase when the particles circulate through peripheral tissues. Adiposity means in the liver and peripheral tissues the action of insulin is resistance, it cause the alteration in intracellular signalling, resulting in the level of secretion of TG is increased [Howard BV et al, 2003]. This is combined with LPL is and the activity decreases or insufficient.

***3.3Coronary artery disease*** Coronary artery disease is a condition in which build up of plaque in the pericardial coronary arteries. In this disease plaque grows in the coronary




**Coronary Artery Disease**

arteries and it limited the blood flow to the heart's muscle. When the plaques formed thenthe formation of occlusive thrombi and the precipipitation of acute events, and increase risk of myocardial infarctions, these all problems increase the oxidative stress in the

pathophysiology of coronary artery disease [Cavalca V, et al, 2001, Harrison D, et al, 2003, Stocker R et al, 2004]. Rise in the prevalence of CAD in developing countries like India. Prevalence of CAD in India is increased due to their lifestyle, neo-affluence and mainly due to changes in food habits. Stress and strain is also responsible for CAD but besides of this, food habits, like they use to eat high lipid, salt with less of fibre and one of the main determinants is intake of green vegetable [Mandal S, et al, 1994].

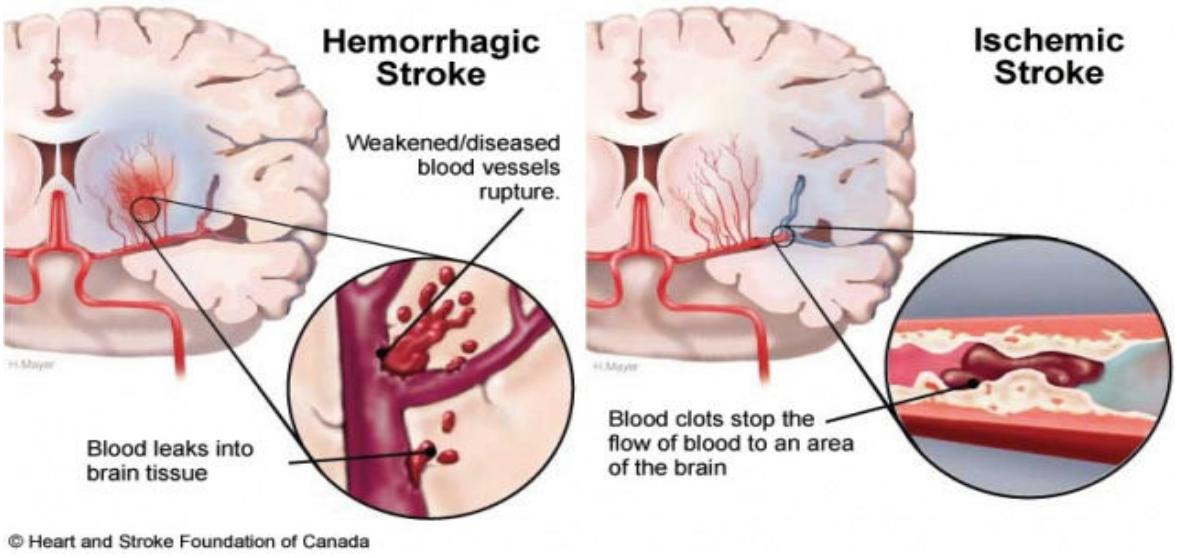
In CAD there is lower level of HDL Cholesterol with higher levels of serum triglycerides [Marcelo KL, et al, 2013, Chi JT, et al, 2003, Regan ER, et al, 2012, Berg KE, et al, 2012].Smallest Lipoprotein is a HDL, it is present in outer layer of phospholipids and free cholesterol it is controlled by Apo-lipoproteins and in the core of lipid cholesterol ester and triglycerides are present [Silva RA, et al, 2008, Rye KA. Et al, 2013, Huang R, et al, 2011], by the action of several enzymes and interactions with specific receptor HDL components are modified along its biogenesis [Rye KA, et al, 2014]. the main HDL protein is Apo A-1, it is produced by hepatocytes, it is also synthesized in the intestine, with the help of phospholipids and free cholesterol form lipid poor complex [Rye KA, et al, 2014], the enzyme which is known as lecithin cholesterol acyl- transferase increases the spherical HDL particles which generate the cholesterol esters in the lipid core pool, they exchange lipids with other VLDL and LDL Lipoproteins, resolve by phospholipids transfer protein, and cholesterol ester transfer protein [Rye KA. Et al, 2013, Sorci-Thomas MG, et al, 2012]. The level of LDL is involved in the risk of coronary disease and myocardial infarction.

### *3.4Mechanism of action*

Active oxygen species which have free radicals they are involved in CAD [William D Misner. Et al, 2003]. In CAD production of free radical and capacity of antioxidant is altered

in favour of the former, it is become to oxidative stress. On the basis of “Oxidation hypothesis" LDL absorbed in the intimae, under the oxidative modification binding to proteoglycans by the free radicals [Peter Libby, et al, 2002]. when Oxidation of LDL is done then there is chemical modification of some moieties of apolipoprotein by the products of lipid peroxidation.in the lipid fraction of atheroma lipidhydroperoxides, carbonyl compounds and lysophospholipids are localised [Parthasarathy S et al, 1992, Torzewski M et al, 2006] for activate expression of Adhesion molecules, immune response is enhance by oxidised LDL. it activate the Adhesion molecules, Cytokines, chemokines and many mediators of LDL particles which are ox datively modified, enhance the level of triglycerides, along with oxidative stress HDL-C is decreased which is the major risk of CAD.

***3.5Stroke*** Stroke is a clinical Syndrome, in this condition people loss of cerebral functions. Its symptoms show more than 24 hours it leads to death with no appropriate cause other than




vascular origin [NICE guideline 2008]. We defined stroke as the stoppage of blood flow to the brain and it is a leading cause of death. 12% of strokes are hemorrhagic. In hemorrhagic cerebral blood vessel is become rupture, and 88% are ischemic. When there is blockage in cerebral artery stop the blood flow and also interrupt the supply of nutrients, glucose and

oxygen to the brain. The metabolism of glucose and oxygen supply energy to brain for the phosphorylation of ADP to ATP. The ATP which is generated by brain is used to maintain intracellular homeostasis and transmembrane ion. These are gradients of sodium, potassium and calcium. When the energy is not delivered then ion gradients are collapse and high secretion of neurotransmitters such as dopamine and glutamate (Adibhatla and Hatcher 2006), result in neuronal death and develop an infraction. Increase release of glutamate and the receptor are stimulate then the activation of phospholipase/ sphingomyelinase (Adibhatla and Hatcher 2006; Adibhatla, et al., 2006 a) hydrolation of phospholipids occur and it secrete second messengers ArAc and ceramide (Adibhatla and Hatcher, 2006; Adibhatla 2006 b; Mehta, etal, 2007) the process which mentioned above lead to necrotic cell death. Some scientific community see the difference between blood lipids levels and cardiovascular disease [Fruchart JC, et al, 2004]. So they found the main difference between levels of cholesterol, the level of serum cholesterol found high, mainly low-density lipoprotein cholesterol, which develop the atherosclerosis. When they see the level of High density Lipoprotein cholesterol which play a protective role [Fruchart JC, et al, 2004]. Mostly the change in diet and Life style cause, increased hyperlipidemic, which is the risk factor for stroke. In cerebrovascular disease relationship of serum lipids and lipoproteins they are risk factors in coronary heart disease [Khan NI, et al, 2009, Ali L, et al, 1997]. Some clinical trials see difference between high concentrations of serum cholesterol and ischemic stroke [Khan NI, et al, 2009, Bashir K, et al, 2007].

# ***CHAPTER 2***

# ***Review of literature***

## *4 Review of literature*

Roya Kelishadi et al did study in clinics of Isfahan Endocrine and metabolism. Research centre (Iran). They work on the topic of ***"Changes in serum lipid profile of obese or overweight children and adolescents following a lifestyle modification course".*** The aim of their study is to evaluate the effect of an educational course on changes of lipid profile in children. The study was performed on children's. The ages of the children's were 4-18 year old. They were taken the fasting blood samples. In blood they perform some Biochemical test. The parameters which they used total cholesterol; Triglycerides, high-density lipoprotein cholesterol and low density lipoprotein cholesterol were also carried out. They give one educational session to children in whom they taught about ways and benefits of regular physical activity once a day and benefits' of healthy foods. Then the children's were followed the instruction for about four months. After than biochemical tests were repeated. They were taken 412 children's, in which 245 were girls and 167 were boys. Then they divided children's into four age groups, 6-9, 10-13, and 14-18 year old. Study shows there is significantly no difference between boys and girls lipid profile. Level of LDL-C and TC were reduced in boys older than 10 year. In girls which were over 10 year of age they had increase value of HDL-C. The conclusion of that study showed that there is no educational effect on children's anthropometric measurements. [Roya Kelishadi et.al, in 2012]

Ugwuja El. et.al, did study on the topic of ***"Over weight and Obesity, lipid profile and Atherogenic Indices among civil servants in Abakaliki, South Eastern Nigeria".*** The aim of their study to determine the prevalence of overweight, and obesity, plasma lipid profile and atherogenic indices as markers for CVD among civil servants. They take 205 apparently healthy civil servants. Ages of the servants were 21-60 years, mean and standard deviation

years. In 205 apparently healthy civil servants there were 106 males and 99 females. In the study weight and height of the participants were measured. According to weight and height they were calculate the BMI. The result of their prevalent rates of overweight and obesity, 34.2% were overweight and 68% were obsessed. Study showed that men were affected more than women. The civil servants who had older age groups and higher body mass index (BMI) they were most affected. Male servants were found to have lower LDL-C and have higher HDL-C than the females. So the conclusion of their study is show the females with advanced in age , who have higher BMI and have unfavourable plasma lipids and social habits, they do less physical activity, they have higher risk of CVD. They have to take good nutrition, control on weight, smoking and alcohol cessation, do more physical activity and regularly medical check-up. [Ugwuja El et.al 2013]

Ashish Shukla did study on under graduate medical students. The study did in Subharti Medical College, Meerut Uttar Pradesh, and India in the year of 2015. They work on the topic of ***"Altered High density Lipoprotein, triglycerides and anthropometric measurements in normal, overweight and obese under graduate medical students".*** The study is done by Ashish Shukla, Jaskiran Kaur, and Akash Gupta. The aim of their study was they find out changes in metabolic indicators like HDL, TG and anthropometric measurement s of normal, overweight and obese Undergraduate medical students. In their study they took 194 students of Subharti Medical College, Uttar Pradesh. They measure height weight and waist circumferences were measured of all participants by standard methods. The serum of their participants estimated for HDL, TG and fasting sugar. They use fully automatic victors 250 dry chemistry analyser from ortho clinical diagnostics from Johnson Johnson USA. Result of their study shows that female's students were more overweight. 16% females were overweight and 8% males were overweight. 18% female and 9% male were obese. So the

results show that females were more overweight and obese than males. The level of BP, weight and waist circumference, HDL and TG levels were high in overweight and obese group compared to normal group. The conclusion of their study had shown an increased trend of obesity and derangement of metabolic indicators in under graduated medical students. The main reason of this more use of electronic gadgets and spent more time on television, mobiles and computer they have lack of physical activity which causes obesity. [Ashish Shukla etal, 2014]

M.Mohsen Ibrahim a1 et al did study on Egyptian patients. the main aim of their study is they see plasma lipid abnormalities in Egyptians with CAD, Define what is the role of age in CAD , CAD type and also level of Hypertension on lipid profile. They collected the data from specialized cardiac clinic records. They took 1000 patients with CAD and 1920 non-coronary individuals as a group of control for the comparison. They set age of the patients was 19-90 years. By enzymatic methods they were determined the plasma concentration of total cholesterol and triglycerides by phosphotungstate method they determined VLDL and LDL. After the precipitation of VLDL and LDL they measured HDL cholesterol. When TG levels did not exceed 300mg/dl then LDL was estimated by the using of friedwald formula. They calculated their result on the basis of age and gender, lipid profile, BMI, HT near about 56.7% patients has Hypertension. The level of HDL-C was decreased in men and increased plasma Triglycerides in women. According to their result no significant difference in lipid profile was present between normotensive and hypertensive in CAD patients. The conclusion of their study is 80% of patients have abnormalities in lipids. The difference of plasma lipid abnormality according to age and gender, and mode of presentation of CAD. Hypertension had limited effect on the changes in lipid profile. So the lipid abnormalities in Egyptian

patients seen. They have to take healthy diet and do physical activity. [M. Mohsen Ibrahim etal 2012]

Servin Assari 1, 2 etal, did study on association between lipid profile and sexual function among men with coronary artery disease, paucity of knowledge about this association among women with coronary artery disease. They work on the topic of ***"Gender differences in the association between lipid profile and sexual artery disease".*** The aim of their study ids they see the link between lipid profile and sexual function in men and women with coronary artery disease. They take 120 patients with documented coronary artery disease. They collect data on the basis of age, sex; family income and education level also see the patient history of hypertension, cigarette smoking and diabetes mellitus. They check the lipid profile and sexual relationship in patients by using the relation and sexuality scale. They measure the symptoms of anxiety and depression by Hospital Anxiety and depression scale by rose angina questionnaire they measured characteristics of chest pain. In their study there were 91 males and 29 female's patient. The result of their study showed that low density lipoprotein cholesterol was correlated with sexual function and total sexual relationship. The correlation between level of HDL and sexual frequency score was observed. These correlations moderated by gender. In males they correlate serum cholesterol and low density lipoproteins were correlated to sexual function. In females they correlated LDL to the total sexual relationship and HDL was correlated to sexual frequency. The conclusion of their study showed a relationship between lipid profile and sexual relationship among in male and female patients who had coronary artery disease. There is just effect of lipid profile on erectile dysfunction when we see link between lipid profile and sexual function of the patients with CAD. [Servin Assari 1, 2 etal, 2014]

Cynthia A' etal, did study of dyslipidemia with stroke. They did their study in the hospital medical record department. They work on the topic of ***"Dyslipidemia in stroke".*** The aim of their study to see the relationship between lipid profile components and incident ischemic Stroke in a first- ever Stroke cohort. They took 100 patient record were accessed from the medical records department in father Muller medical patients were between 30 to 90 years. They were collected the history and physical examination details of patient records. They analysed lipid profile according to the ATP 3. According to their study 56% of patients had dyslipidemia, 40% had high total cholesterol, 7 % of the patient had high triglycerides, and only 3% of the patient had high LDL. Whereas 28% of the patients showed low HDL levels and 10% of the patients had both high total cholesterol and low HDL. The conclusion of their study shows a significant association of 56% of between dyslipidemia and stroke. High LDL is responsible for cerebrovascular accidents and significant proportion of patients with low HDL so this study shows primary prevention is the key to overcoming the burden of stroke in our country [Cynthia A1, etal 2014].

Dr.Jayita Dasgupta (Ghosh) etal, did study on the morbidity and mortality caused by coronary artery disease. They did their study in rural population of Bihar. They work on the topic of ***"Study of Lipid Profile in patients of coronary artery disease among rural population".*** They took 100 consecutive cases. In this study they were undertaken to study they were undertaken to study dyslipidemia among the rural patients with coronary artery disease. The age of the patients were 30 to 90 years who were diagnosed as coronary artery disease they were compared to 50 ages and sex matched healthy controls. They were recorded in each subject like age, gender, blood pressure, history of smoking and body mass

index. Blood sample used for investigation of lipid profile. The conclusion of the case control study shows that high lipid profile are clinical significant in all age groups above 40 years. This study reveals a distinct association of dyslipidemia with CAD and highlights patients with dyslipidemia, abnormal lipid profile and its proper management by life style modification [Dr. Jayita Dasgupta etal, 2015]

J.woo etal, did their study on the topic of ***"Acute and long-term changes in serum lipids after acute Stroke".*** They took 171 patients. They studied serum lipid profile in total patients. In 171 patients 83 patients suffering from cerebral infarction. Who had significantly higher serum concentration of lipid profile? According to the lipid profile 53 patients were suffering Lacunars infarction. The conclusion of their studies of serum lipid and lipoprotein concentrations is risk factors for cerebral infarction [J.woo etal 2015].

# ***CHAPTER 3***

# ***MATERIAL AND METHODS***

## *5 Material and Methods*

The study I have done is in the department of Biochemistry of "Satguru Partap Singh Hospital" Ludhiana. In my study I use to evaluate the Lipid Profile of the patients in Punjab. My topic of study is ***"Prognosis of different diseases due to Alteration in Lipid Profile".*** This study complete in between ***19 Jan- 2 may.*** Clinically and Biochemically newly diagnosed patients who have shown alteration in lipid profile. I took both sexes who had suffering from some diseases because of the alteration in the lipid profile. The two groups of patients, who have age less than 50 years and more than 50 years. Patients included with Dyslipidemia, Obesity, Coronary artery disease and stroke in this study. Disease was diagnosed by clinical history, physical examinations and relevant laboratory investigations. Total 150 subjects were included in the study and out of the 74 subject's show abnormal lipid profile. Cases were further grouped on the basis of their diseases and age groups. In Group A subjects involved who have Obesity. In Group B who have Coronary Artery Disease. In Group C subjects involved who have Stroke. All specimens was collected in fasting condition, allowed to clot, serum was separated and analyzed for serum total cholesterol, Triglycerides, HDL, LDL, VLDL, was calculated. I used fully automated instruments for the analysis of different parameters. Also check the history of the patients. Along with their History and Lipid Profile, BMI also calculated with the help of height and weight.

The following investigations were performed:

- Measurements of body mass index, Skin fold thickness and waist/hip ratio.
- BMI = Weight(kg)/ Height
- Estimation of
    - Serum Cholesterol

- Serum Triglyceride
- Serum HDL Cholesterol
- VLDL and LDL were estimated

## *5.1Statically Analysis*

Statically Analyses were performed by using SPSS. Mean values of the findings were compared among and between groups. Results were classified into 2 age groups and 3 groups of diseases for Male and Female.

# ***CHAPTER 4***

# ***RESULT AND DISCUTTION***

## *6. Results and discussion*

| *S.No* | *Lab ID* | *Age/Sex* | *TC* | *TG* | *HDL* | *LDL* | *VLDL* | *Diagnose* |
|---|---|---|---|---|---|---|---|---|
| **1** | 83913 | 59/F | 338 | 715 | 50 | 175 | 114 | CAD |
| **2** | 336909 | 36/M | 231 | 266 | 50 | 137 | 44 | Obesity |
| **3** | 196294 | 37/F | 207 | 85 | 42 | 148 | 17 | Obesity |
| **4** | 178387 | 33/M | 193 | 313 | 40 | 105 | 48 | over weight |
| **5** | 3717 | 63/M | 101 | 168 | 35 | 52 | 14 | Stroke |
| **6** | 71565 | 38/F | 200 | 167 | 40 | 132 | 28 | Obesity |
| **7** | 337047 | 52/M | 241 | 547 | 40 | 102 | 99 | Obesity |
| **8** | 337068 | 62/F | 294 | 245 | 41 | 127 | 26 | CAD |
| **9** | 337211 | 68/F | 226 | 150 | 46 | 152 | 28 | Stroke |
| **10** | 6377 | 29/M | 258 | 149 | 43 | 179 | 36 | CAD |
| **11** | 337059 | 52/M | 248 | 495 | 54 | 126 | 68 | Obesity |
| **12** | 8279 | 54/M | 229 | 119 | 27 | 56 | 21 | CAD |
| **13** | 337383 | 44/M | 223 | 207 | 45 | 128 | 50 | CAD |
| **14** | 1338 | 46/M | 196 | 212 | 61 | 111 | 24 | over weight |
| **15** | 9999 | 65/F | 210 | 58 | 57 | 132 | 21 | Stroke |
| **16** | 337219 | 73/F | 223 | 166 | 50 | 136 | 37 | Stroke |
| **17** | 232962 | 24/M | 180 | 211 | 50 | 102 | 28 | Obesity |
| **18** | 133584 | 84/F | 250 | 127 | 68 | 152 | 30 | Stroke |
| **19** | 123608 | 37/F | 233 | 126 | 34 | 177 | 22 | CAD |
| **20** | 68480 | 57/M | 203 | 162 | 54 | 117 | 32 | Stroke |
| **21** | 337470 | 57/M | 153 | 173 | 36 | 79 | 38 | Obesity |
| **22** | 134901 | 64/F | 200 | 148 | 76 | 96 | 29 | Obesity |
| **23** | 250082 | 56/M | 214 | 174 | 46 | 136 | 32 | CAD |
| **24** | 178011 | 59/F | 264 | 254 | 56 | 159 | 47 | Overweight |
| **25** | 187633 | 38/F | 248 | 150 | 49 | 156 | 43 | Stroke |
| **26** | 337655 | 30/M | 164 | 153 | 37 | 105 | 23 | Obesity |
| **27** | 337775 | 50/F | 199 | 228 | 68 | 105 | 26 | Obesity |
| **28** | 249291 | 59/M | 173 | 192 | 39 | 108 | 27 | CAD |
| **29** | 337733 | 32/M | 200 | 320 | 36 | 122 | 42 | Obesity |
| **30** | 337779 | 98/M | 265 | 261 | 46 | 184 | 34 | Stroke |
| **31** | 337809 | 42/M | 248 | 256 | 47 | 174 | 27 | Stroke |
| **32** | 337813 | 48/F | 231 | 230 | 57 | 155 | 19 | CAD |
| **33** | 337834 | 65/F | 131 | 215 | 49 | 71 | 11 | Obesity |
| **34** | 337914 | 64/F | 205 | 77 | 58 | 133 | 14 | CAD |
| **35** | 96511 | 47/M | 197 | 255 | 37 | 126 | 34 | Obesity |
| **36** | 339152 | 49/F | 214 | 201 | 40 | 148 | 26 | Stroke |
| **37** | 339008 | 56/M | 264 | 259 | 35 | 200 | 29 | Obesity |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **38** | 105065 | 65/M | 222 | 137 | 42 | 154 | 27.4 | Stroke |
| **39** | 167303 | 57/M | 256 | 120 | 57 | 183 | 16 | Obesity |
| **40** | 339196 | 35/M | 220 | 110 | 59 | 152 | 9 | CAD |
| **41** | 339195 | 90/M | 227 | 232 | 51 | 153 | 24 | Stroke |
| **42** | 339203 | 50/F | 240 | 219 | 54 | 155 | 31 | CAD |
| **43** | 329208 | 50/F | 251 | 310 | 48 | 164 | 39 | Obesity |
| **44** | 339233 | 86/M | 274 | 277 | 55 | 187 | 32 | Obesity |
| **45** | 339142 | 46/M | 232 | 221 | 52 | 165 | 15 | Obesity |
| **46** | 164112 | 59/F | 197 | 204 | 40 | 128 | 29 | Obesity |
| **47** | 277017 | 41/M | 206 | 227 | 36 | 131 | 39 | CAD |
| **48** | 277027 | 35/M | 170 | 266 | 33 | 104 | 33 | Obesity |
| **49** | 339387 | 52/M | 268 | 275 | 54 | 176 | 38 | Stroke |
| **50** | 334038 | 61/M | 165 | 300 | 35 | 86 | 44 | Obesity |
| **51** | 1299355 | 38/M | 211 | 190 | 37 | 138 | 36 | Stroke |
| **52** | 222689 | 47/F | 219 | 133 | 49 | 161 | 9 | CAD |
| **53** | 339525 | 31/M | 167 | 227 | 43 | 99 | 25 | Obesity |
| **54** | 12808 | 40/M | 217 | 272 | 35 | 159 | 23 | CAD |
| **55** | 119140 | 18/M | 200 | 117 | 34 | 158 | 8 | CAD |
| **56** | 339323 | 52/F | 258 | 244 | 49 | 181 | 28 | Stroke |
| **57** | 339559 | 34/M | 217 | 570 | 40 | 81 | 96 | Obesity |
| **58** | 300238 | 40/M | 245 | 219 | 49 | 168 | 28 | Stroke |
| **59** | 339588 | 54/F | 257 | 208 | 49 | 187 | 21 | CAD |
| **60** | 196674 | 48/F | 216 | 189 | 49 | 153 | 37.8 | CAD |
| **61** | 245874 | 50/M | 188 | 265 | 26 | 130 | 32 | Obesity |
| **62** | 339550 | 41/M | 170 | 258 | 32 | 104 | 34 | Obesity |
| **63** | 62306 | 56/M | 197 | 844 | 35 | 55 | 103 | Obesity |
| **64** | 339732 | 49/M | 182 | 224 | 33 | 129 | 20 | CAD |
| **65** | 174320 | 61/F | 226 | 136 | 61 | 148 | 17 | CAD |
| **66** | 339691 | 80/F | 202 | 319 | 37 | 112 | 53 | Obesity |
| **67** | 339891 | 73/F | 238 | 140 | 61 | 167 | 10 | CAD |
| **68** | 292481 | 52/M | 200 | 130 | 35 | 157 | 8 | Obesity |
| **70** | 339641 | 25/F | 195 | 261 | 24 | 136 | 34 | Obesity |
| **71** | 115828 | 80/F | 204 | 97 | 75 | 121 | 7 | Stroke |
| **72** | 339560 | 37/F | 147 | 176 | 43 | 83 | 21 | Obesity |
| **73** | 339545 | 39/M | 176 | 157 | 31 | 129 | 16 | CAD |
| **74** | 339544 | 29/M | 185 | 216 | 62 | 107 | 16 | Stroke |

Table Shows the Lipid Parameters between different Diseases. The level of Cholesterol, HDL Cholesterol, LDL cholesterol and Triglycerides level in cases were high. In 74 subjects there were 33 subjects were obese, 19 subjects of stroke and 22 subjects have coronary artery disease.

## *6.1Obesity*

| | More than 50 | Less than 50 |
|---|---|---|
| **Age** | 14 subjects | 19 subjects |
| **Male** | 9 subjects | 13 subjects |
| **Female** | 5 subjects | 6 subjects |

In this table shows comparison of Lipid Profile parameters between two age groups in Obesity. There was significant difference among the age groups. Males were more affected by Obesity than female. There is 9 male subject have obesity who were more than age of 50. And 13 male subjects who were less than age of 50. Whereas 5 females who have age more than 50 and 6 females who have age less than 50 were affected with Obesity. Total according to age groups there were 14 subjects who were more than 50, and 19 subjects who have less than 50 years of age have obesity. There were Difference in values because of their life style, food habits and physical activity of subjects.

## *6.2Coronary Artery Disease*

| | More than 50 | Less than 50 |
|---|---|---|
| **Age** | 9 subjects | 13 subjects |
| **Male** | 3 subjects | 8 subjects |
| **Female** | 6 subjects | 5 subjects |

In this table shows comparison of Lipid Profile parameters between two age groups in CAD. There was significant difference among the age groups, but there is no significant difference among the sex groups. Males and Females were equally affected with CAD.

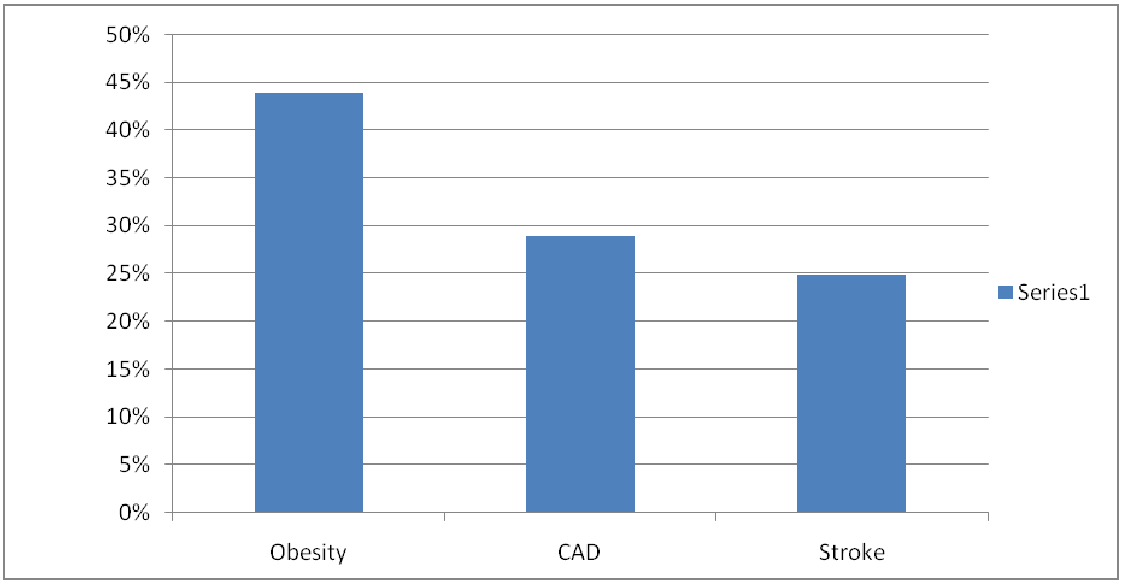
There was 3 male subject have CAD who were more than age of 50. And 13 male subjects who were less than age of 50. Whereas 6 females who have age more than 50 and 5 females who have age less than 50 were affected with CAD. Total according to age groups there were 9 subjects who were more than 50, and 13 subjects who have less than 50 years of age have CAD. There were Difference in values because of their life style, food habits and physical activity of subjects.

### 6.3*Stroke*

| | **More than 50** | **Less than 50** |
|---|---|---|
| **Age** | 12 subjects | 7 subjects |
| **Male** | 6 subjects | 4 subjects |
| **Female** | 6 subjects | 2 subjects |

In this table shows comparison of Lipid Profile parameters between two age groups in Stroke. There was significant difference among the age groups, but there is no significant difference among the sex groups. Males and Females were equally affected with Stroke. There was 6 male subject have Stroke who were more than age of 50. And 4 male subjects who were less than age of 50. Whereas 6 females who have age more than 50 and 2 females who have age less than 50 were affected with Stroke. Total according to age groups there were 12 subjects who were more than 50, and 7 subjects who have less than 50 years of age have Stroke. There were Difference in values because of their life style, food habits and physical activity of subjects.

### *6.4Column Chart according to the Diseases*




The chart shows that mostly patients have obesity, and CAD is the second disease found in patients and some patients have stroke.

## Conclusion

Conclusion of my study is, subjects who were taken in this study have affected with different diseases. Mostly patients affected with Obesity. There is 44% were obsessed, 29% were CAD, and 25% have Stroke. Out of 150 patients 74 subjects have abnormal Lipid Profile. Patients have high TC, TG, LDL and lower level of HDL, because of altered lipoprotein metabolism. Several common reasons are also responsible for the alteration of lipid profile like, their sedentary lifestyle. Here am concluded my study we can reduced chance of alteration of lipid profile by changing our eating habits, life style and by doing physical activities once in a day.

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