

1.INTRODUCTION

1.1 Lipid :

Lipids commonly referred to as fats ,have a dual role ,first because they are composed of mostly carbon hydrogen (c-h) bonds they are a rich source of energy are an efficient way for the body to store excess calories

The lipids transported by lipoproteins ,namely fatty acid triglyceride ,phospholipids ,cholesterol and cholesterol esters . ⁽¹⁾

1.2 Lipoproteins:-

A lipoprotein is biochemical assembly whose purpose is to transport hydrophobic lipid molecules in water, as in blood or ECF, they have a single layer phospholipid and cholesterol outer shell, with the hydrophilic proteins oriented outward toward the surrounding water ⁽²⁾

1.2 .1 Classification of lipoproteins:-

Lipoproteins are classified on the basic of their densities as demonstrated by their ultra centerfigual separation. There are five main classes of lipoproteins, triglyceride rich particles include:-

- chylomicrons
- VLDL(very low density lipoprotein)
- IDL (intermediate density lipoprotein)
- LDL(low density lipoprotein)
- HDL[high density lipoprotein]
- HDL(good) cholesterol:- helps keep cholesterol from building up in the arteries and protects against heart disease, so for HDL, higher numbers are better.

A level less than 40 mg/DL is low and is considered a major risk factor because it increases risk for developing heart disease. HDL levels of 60 mg/DL or more help to lower the risk for heart disease.

- LDL (bad) cholesterol :- the main source of cholesterol building up and blockage in the arteries. A level less than 100 mg/DL is optimal and a level more than 190 mg/DL is considered a major risk factor because it increases the risk for developing heart disease. ⁽²⁾

1 .2.2Low Density Lipoprotein [LDL]

This is the "bad" cholesterol which increase your risk .of heart disease . if found too much in the blood can lead to cholesterol build- up and artery blockages.

Smoking is associated with decrease high density lipoprotein and increased level of triglycerides .also smoking associated with increased low density lipoprotein. ⁽³⁻⁴⁾

The optimal level of LDL :

Less than 100 mg/dl

1.3 Rationale

smoking has high risk for cardiovascular disease which became the major cause of death ,this study will conducted to evaluate the effect of smoking on serum LDL level.

1.4 OBJECTIVES

General objective:

To assess the effect of cigarette smoking on low density lipoprotein in smoker students in university of Elsheik Abdulla Elbadari

*** Specific objective:-**

- * To measure serum LDL level of smokers and non smokers.
- * To determine the relation between duration of smoking and serum LDL level.
- * To determine the relationship between the period of smoking and LDL level.

2-Smoking and cholesterol:

Smoking is a bad behavior for health it harms nearly every organ in the body .

Cigarette smoking is a major cause of preventable morbidity and mortality worldwide ,more than 3 million people currently die each year from cigarette smoking .

The risk of death in the smokers measured by the number of cigarette smoked daily , the duration of smoking ,the degree of inhalation and the age of inhalation .

Cigarette smoking contains over 4000 different chemicals, 400 of which are proven to be carcinogenic ; it also contains various oxidants such as oxygen free radicals and volatile aldehydes which are probably the major causes of damage to bio molecules

Of lung cancer death and cigarette smoking cause 87 lung inflammation heart problem like blood vessel disease coronary heart and cardiovascular disease as well as effects and sudden infant death syndrome reproductive

Also responsible for yellow brown staining on smoker finger and teeth
smoking elevated total cholesterol in the body.

Smoking is associated with decrease high density lipoprotein and increased level of triglycerides .also smoking associated with increased low density lipoprotein⁽³⁻⁴⁾

Previous study, In a survey of a healthy population ($n = 197$), LDL cholesterol, plasma triglycerides and VLDL triglycerides were found to be substantially increased and plasma HDL cholesterol decreased in smokers. The lipid-associated atherogenic risk in smokers as assessed by the LDL/HDL ratio was significantly higher [2.89 (SD 1.18, $n = 63$)] than in non-smokers [2.38 (SD 0.98, $n = 86$) $P < 0.01$].⁽⁵⁾

Another Previous study, Conflicting reports on the effect of smoking and coffee drinking on lipoproteins prompted us to study the combined effect of these two associated, widely prevalent habits in 361 persons randomly sampled from the Evans County cohort. Low-density lipoprotein (LDL) cholesterol levels were significantly higher [$P < 0.01$].among persons who smoked cigarettes and consumed five or more cups of coffee per day than among nonsmokers who abstained from coffee. (6).

2.1 Cholesterol :

Its organic molecule ,its sterol or modified steroid (2)

Cholesterol can also exist in an esterified form called cholesteryl ester. Cholesterol esters are classified as a natural lipid and are not found on the surface of lipid layers but instead are located in the center of lipids drops and lipoproteins.

Good cholesterol and bad cholesterol

Cholesterol can be good or bad. HDL is called "good cholesterol" that is good for the cardiovascular system and LDL is called "bad cholesterol" that is bad for the cardiovascular system. These are the form in which cholesterol travels in the blood. LDLs have little protein and high levels of cholesterol and HDL has a lot of protein and very little cholesterol. LDL is the main source of artery clogging plaque. HDL actually works to clear cholesterol from the blood. (2)

2 .1.1sources of cholesterol:-

- Endogenous cholesterol is formed in the body almost in all nucleated cells from Acetyl-coA.
- Exogenous cholesterol occurs only in food animal origin such as egg yolk, meat, liver and brain . (2)

2.1 .2Transport of cholesterol:-

1- cholesterol is hydrophobic it is transported in plasma in the more soluble lipoprotein forms: LDL, VLDL and HDL.

2- free cholesterol is removed from tissues by HDL and transported to be excreted by the liver.

3- cholesterol ester is the storage form of cholesterol: it is formed in both tissues and plasma. ⁽²⁾

2.1 .3function of cholesterol:-

1- cholesterol enters in the structure of every body cell.

2- cholesterol is the precursor of :

- vitamin D .

- steroid hormones.

- bile acids.

The factor affect cholesterol levels:-

A variety of factors can affect in cholesterol levels. They include:

1- Diet. Saturated fat, trans fat and cholesterol in the food eat increase cholesterol levels. Reducing the amount of saturated fat and trans fat and cholesterol in diet helps lower blood cholesterol level.

2-weight. In addition to being a risk factor for heart disease, being overweight can also increase cholesterol. Losing weight can help lower LDL, total cholesterol levels and triglyceride levels, as well as raise HDL.

3- exercise. R cholesterol , should try to be physically active for 30 minutes on most days.

4- Age and gender. As we get older, cholesterol levels rise. Before menopause, women tend to have lower total cholesterol levels than men of the same age. After menopause, however, women's LDL levels tend to rise.

5- Hereditary. Genes partly determine how much cholesterol body makes. High blood cholesterol can run in families.

6- Medical conditions. Occasionally a medical condition may cause an elevation of cholesterol levels in the blood. These include hypothyroidism, liver disease and kidney disease.

7- Medications. Some medicines, like steroids and progestins may increase the "bad" cholesterol and decrease the "good" cholesterol⁽²⁾.

2.1 .4Hypercholesterolemia :

hypercholesterolemia is the lipid abnormality most closely linked to heart disease.

One form of the disease which is associated with genetic abnormalities that predispose affected individuals to elevated cholesterol levels is called familial hypercholesterolemia (FH) .

-homozygotes for (FH) are fortunately rare and can have total cholesterol concentrations as high as 800 to 1000 mg/DL.

These patients frequently have their first heart attack when still their teenage years.

-too much cholesterol can lead to:

Heart disease stroke atherosclerosis, a clogging or hardening of arteries.

Risk factor for high cholesterol:

Family history of high cholesterol or heart disease, overweight or obese, drink alcohol, frequently smoke cigarettes, kidney disease, polycystic ovary syndrome and hypothyroidism. ⁽²⁾

3.MATERIAL

3.1 BioSystems S.A :

3.1.1 Principle of the method

A specific detergent solubilizes the cholesterol from high density lipoproteins (HDL), very low density lipoproteins (VLDL) and chylomicrons. The cholesterol esters are broken down by cholesterol esterase and cholesterol oxidase in a non-color forming reaction. The second detergent, present in the reagent B, solubilizes cholesterol from low density lipoproteins (LDL) in the sample. The LDL cholesterol is then spectrophotometrically measured by means of the coupled reactions described below³

3.1.2 Content :

	COD 21585	COD 23585
Reagent A	2×60 mL	1×60 MI
Reagent B	2×20 mL	1×20 MI

3.1.3 Samples :

Serum, or sodium heparinized plasma collected by standard procedures. LDL cholesterol in serum is stable for 5 days at 2-8°C.

3.1.4 Metrological characteristic :

The metrological characteristics described below have been obtained using a BA400 analyzer and following the guidelines of the Clinical & Laboratory Standards Institute (CLSI).

- Detection limit: 0.44 mg/dL = 0.012 mmol/L.
- Linearity limit: 990 mg/dL = 25.6 mmol/L.

- Precision:

3.1.5 BA200 procedure :

- **General :**

Name | CHOL LDH DIRECT

Short Name | LDH DIR

Sample type | serum / plasma

Analysis mode | differential by reagent

Unit | mg/dL

Decimals | 0

Reaction type | increasing

- **Procedure :**

Reading mode | monochromatic

Main filter | 535

Reference filter | -

Sample | 3

Vol. R1 | 300

Vol. R2 | 100

Reading 1(cycle)|17

Reading 2(cycle)| 35

Predilution factor | -

- **CALIBRATION AND BLANK**

Blank type | distilled water

Calibration mode | experimental calibration

Number of calibrators | 1

Calibration curve | -

3.1.6 BA400 procedure :

- **General :**

Name | CHOL LDH DIRECT

Short Name | LDH DIR

Sample type | serum / plasma

Analysis mode | differential bireagent

Unit | mg/dL

Decimals | 0

Reaction type | increasing

- **Procedure :**

Reading mode | monochromatic

Main filter | 535

Reference filter | -

Sample | 3

Vol. R1 | 300

Vol. R2 | 100

Reading 1(cycle)| 35

Reading 2(cycle)| 70

Predilution factor | -

- **CALIBRATION AND BLANK**

Blank type | distilled water

Calibration mode | experimental calibration

Number of calibrators | 1

Calibration curve | -

3.2 Methods

3.3 Study Design:

Case control study

3.4 Study Area:

Berber ,river Nile state.

3.5 Study Duration:

From February 2 to May 5

3.6 Study population:

smokers and non smokerd.

3.7 Sample Size Formula:

Sample size will take from 60 person , 30 are non smokerd control , and other 30 are smoker

3.8 Criteria of selection:

3.8.1 Inclusion criteria:

Smokers males for test group and non Smokers for control

3.8.2 Exclusion criteria:

Any disease or other disturbance that can interfere with the result such as heart disease and alcoholism.

3.9 Data Collection:

The primary data will collected by a standard questionnaire and the secondary data was analyzed.

3.10 Data Analysis:

Data will analyzed by SPSS .

3.11 Ethical consideration:

The individuals included in the study were notified well about the objective and the need of this study and they accept to give blood samples before the start of the collection process.using questionnaire

Methodology

A total of (60) subject were included in this study (30) subjects were cigarette smoking males and (30) normal control non cigarette smokers males groups for comparison , collected 2.5 my of venous blood in lithium heparin , then centerfugation of blood sample , and analyzed of serum LDL in cigarette smokers males and normal control non cigarette smokers by Biosystem S.A

Equipment :

syringe, EDTA container , centrifuge, reagent , Glass tubes and rake, Micro automatic pipette

Result :

A total of (60) subject were included in this study (30) subjects were cigarette smokers males and (30) normal control non cigarette smokers male groups for comparison, the normal control groups selected according to inclusion criteria in berber locality. Table (3.1) shows the comparison of mean (SD), mean difference of (LDL) of cigarette smokers and normal control groups. There was significant means difference of LDL cigarette smokers and normal control (p.value=0.0027).

This result reflects the effect of cigarette smoking on LDL

Table(4.1) correlation between LDL in control (non smokers) and smokers

	Mean	Median	Std. Deviation	P. value
LDL Control	53.127	55.550	11.4191	0.0027
LDL Test	86.523	79.600	16.7831	

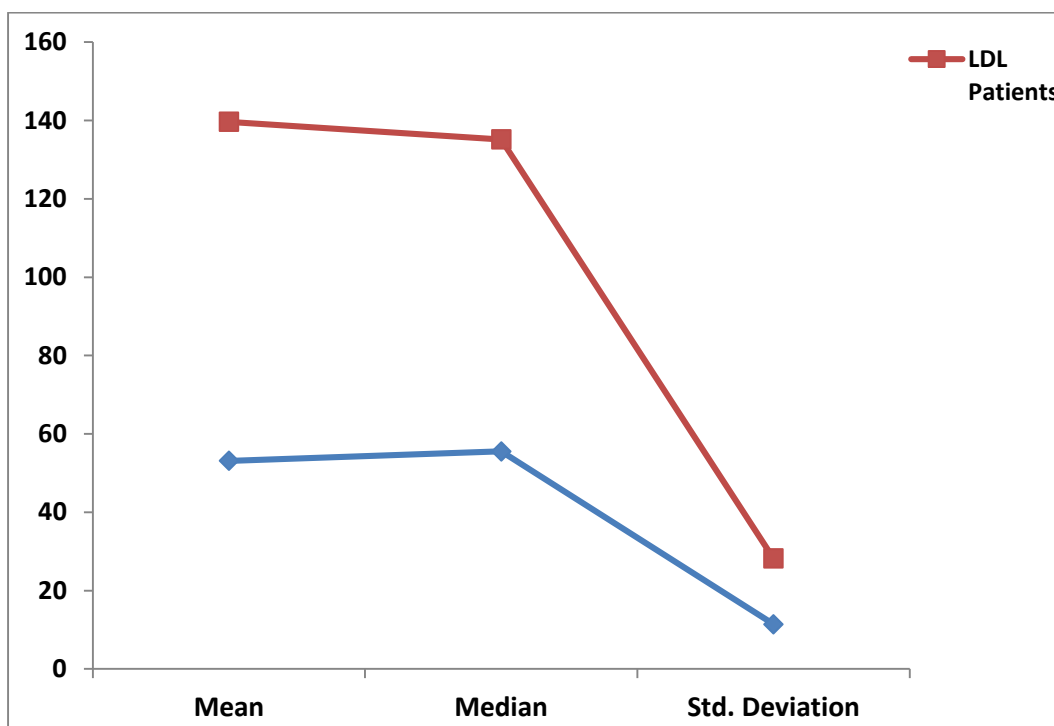
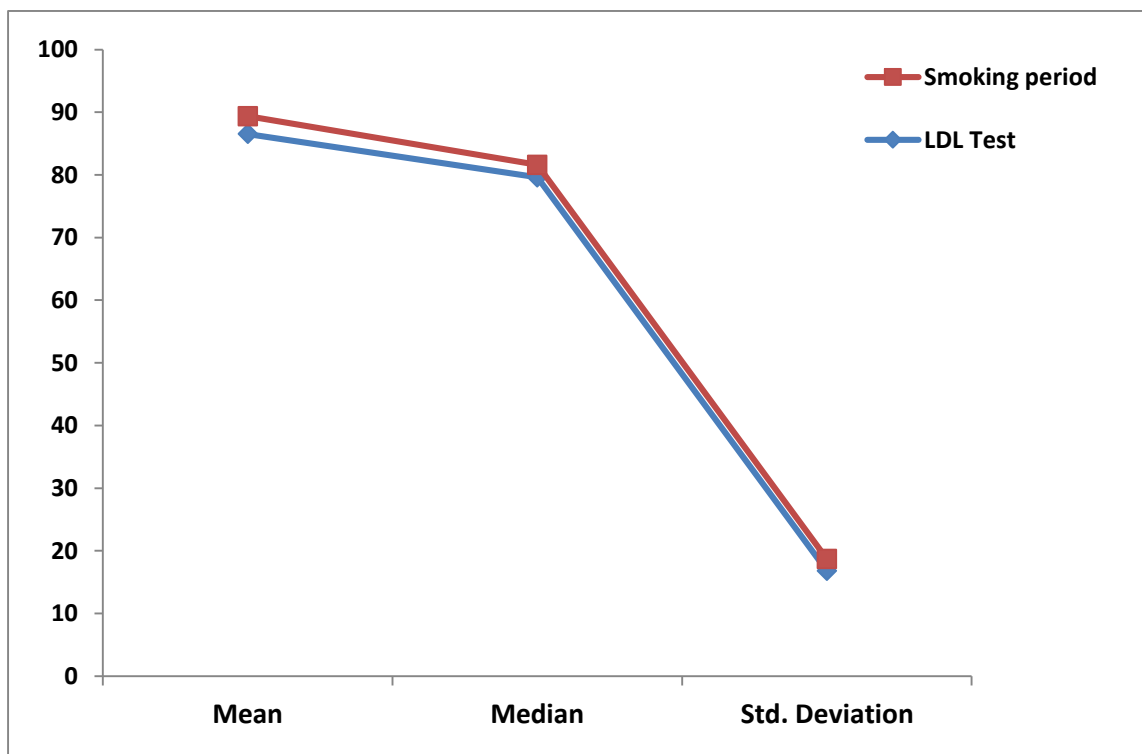


Table (4.2) correlation period of smoking (more than one year) on LDL

	Mean	Median	Std. Deviation	P. value
LDL Test	86.523	79.600	16.7831	0.0023
Smoking period	2.850	2.000	1.8899	



Descriptive Statistics

		Statistic	Bootstrap ^a			
			Bias	Std. Error	95% Confidence Interval	
					Lower	Upper
LDL	Mean	86.523	-.056-	3.012	80.450	92.709
	Std. Deviation	16.7831	-.4869-	2.7229	11.2962	21.4310
	N	30	0	0	30	30
LDLcontrol	Mean	53.127	.063	2.005	49.212	56.867
	Std. Deviation	11.4191	-.5789-	2.5679	6.7761	15.8500
	N	30	0	0	30	30

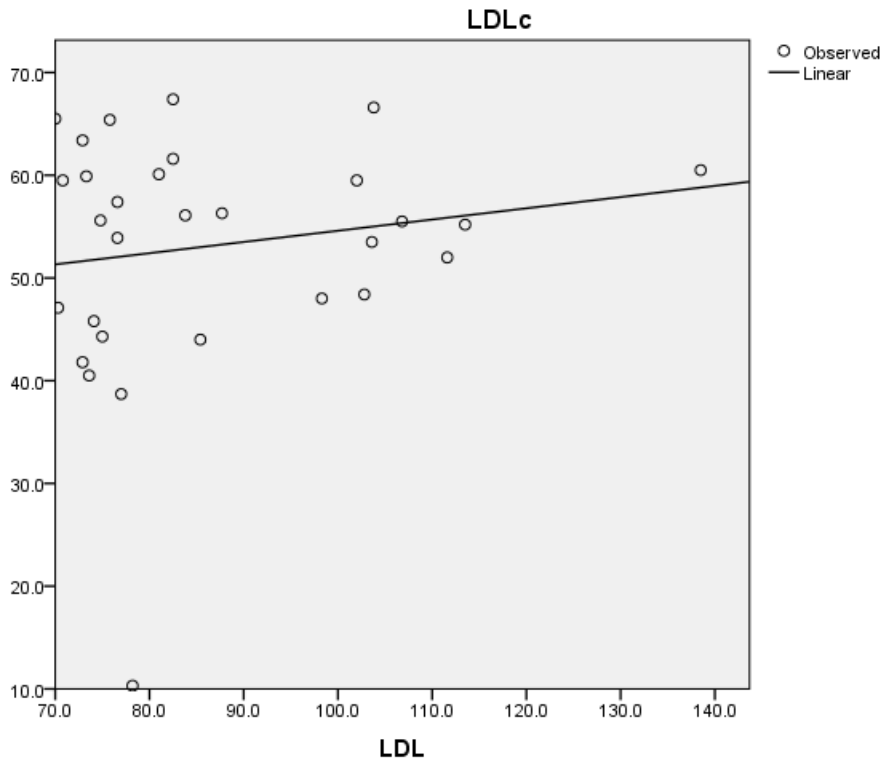
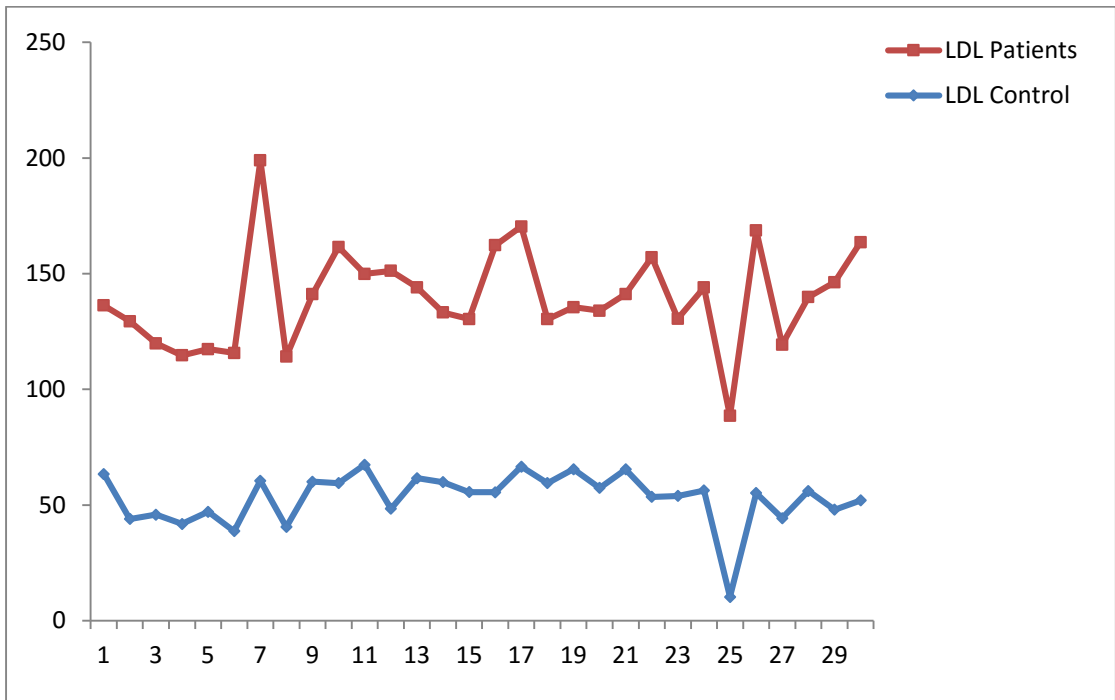
a. Unless otherwise noted, bootstrap results are based on 1000 bootstrap samples

Correlations

		LDL	LDLc	
LDL	Pearson Correlation	1	.161	
	Sig. (2-tailed)		.397	
	N	30	30	
	Bootstrap ^c	Bias	0	-.004-
		Std. Error	0	.120
		95% Confidence Interval	Lower Upper	1 -.111- .371
	LDLcontrol	Pearson Correlation	.161	1
		Sig. (2-tailed)	.397	
		N	30	30
		Bootstrap ^c	Bias	-.004-
Std. Error			.120	0
95% Confidence Interval			Lower Upper	1 -.111- .371

c. Unless otherwise noted, bootstrap results are based on 1000 bootstrap samples

Correlation Between LDL Control and LDL Patients:



Discussion:

In this study the level of low density lipoproteins (LDL) was measured in 60 person , 30 are healthy people control , and other 30 are smoker , both groups were matched for age .

The current study show significant difference in the mean of serum low density lipoproteins (LDL) levels in smoker patient group when compared with control group(0.0027) this agrees with study done by D.J. FREEMAN

also agree with SIEGFRIED HEYDEN, M.D., PH.D.

Conclusion :-

According to this study it is concluded in healthy and smoker patients

- 1- serum low density lipoproteins (LDL) level are significantly raised in smoker patient
- 2- There is a significant change in serum low density lipoproteins (LDL) level between smoker and healthy
- 3- There is a positive correlation between the serum low density lipoproteins (LDL) level and smoker
- 4- There is a significant relationship between duration of smoking and serum low density lipoproteins (LDL) level

Recommendation :-

- 1- Study on a large sample size should be done to obtain accurate results about serum low density lipoproteins (LDL) in smoker.
- 2- Study should be done to assess the effect of smoking on liver function tests

REFRANCE:-

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Appendix :-



بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ



AL-BADRI ABDULLAH ELBADRI UNIVERSITY

FACULTY OF HEALTH SCIENCES

DEPARTMENT OF MEDICAL LABORATORY

**Effect Of Cigarette Smoking On Low Density Lipoprotein
Level In Albadry University**

Questionnaire

General Information:

Name

Age.....

Serial number

Duration Of smoking.....[years]

Range of smoking daily :

3 cigarette []

3-5 cigarette []

Above 5 cigarette[]

Present of history disease

Drugs intake

RESULT:-

Serum of LDL(mg/dl)