

# Original Article

## Lipid Profile and Arterial Stiffness in a Study Population

Sajjan Lal Verma

Associate Professor, Department of Physiology, Mayo Institute of Medical Sciences Gadia, Barabanki, U.P., India

### Corresponding author:

Dr. Sajjan Lal Verma

Associate Professor,

Department of Physiology,

Mayo Institute of Medical Sciences

Gadia, Barabanki, U.P.,

India

**Received:** 28-08-2013

**Revised:** 18-09-2013

**Accepted:** 28-09-2013

### ABSTRACT:

**Background:** Lipid profile is better predictor of cardiovascular diseases. This study was conducted to determine the lipid profile and central arterial stiffness in study population.

**Materials & Methods:** This study was conducted in the department of Physiology on 120 healthy subjects. In all pulse wave velocity and intima media thickness was done.

**Results:** Males were 55 and females were 65. The difference was non-significant ( $P > 0.05$ ). Maximum number of subjects had s. cholesterol  $<150$  (56) followed by 150-250 (38) and  $>250$  (26). Males have higher Av C-F PWV and Av CIMT as compared to females. The difference was significant ( $P < 0.05$ ). C-F PWV and CIMT determination based on LDL also revealed that both values were significantly higher in males as compared to females ( $P < 0.05$ ). Distribution of C-F PWV and CIMT according to HDL showed decrease in both values as HDL level increases. Level was more in males than females and difference was significant ( $P < 0.05$ ).

**Conclusion:** Increased level of LDL, VLDL and decrease level of HDL indicates increase in arterial stiffness and carotid intima media thickness.

**Key words:** Intima, Lipid, Pulse

This article may be cited as: Verma SL. Lipid Profile and Arterial Stiffness in a Study Population. J Adv Med Dent Scie Res 2013;1(2):171-74.

### INTRODUCTION

Atherosclerosis is associated with increased plasma LDL. Atherosclerosis leads to deposition of lipids within proliferated smooth muscle cells, macrophages, connective tissue matrix. Lipids include VLDL, LDL, triglycerides and HDL.<sup>1</sup> VLDL stands for very low density lipoprotein. Lipoproteins are made up of cholesterol, triglycerides, and proteins. They move cholesterol, triglycerides, and other lipids (fats) to around the body. VLDL is one of the three main types of lipoproteins. VLDL contains the highest amount of triglycerides. VLDL is considered a type of bad cholesterol, because it helps

cholesterol build up on the walls of arteries. Normal VLDL levels are from 2 to 30 mg/dl.<sup>2</sup>

LDL (low density lipoprotein) and VLDL (very low density lipoprotein) transport mostly fat and cholesterol. VLDL is made up of 55-65% triglycerides, 10-15% cholesterol, 15-20% phospholipid, and 5-10% protein. For comparison, HDL the "good" cholesterol is 45-50% protein. A desirable total cholesterol level for adults without heart disease is less than 200 mg/dL. An HDL cholesterol level of 60 mg/dL and above is considered protective against heart disease, while a level less than

50 mg/dL for women or 40 mg/dL for men is considered a major risk factor for heart disease.<sup>3</sup>

Exercise plays an important role in maintaining normal blood pressure and controlling normal lipid values. Exercise decreases the incidence of CAD. High level of physical fitness has lower rates of CAD. While decreased level of physical fitness increases risk of atherosclerosis. Regular aerobic physical training increases cardiac output, prostacyclin and decrease systemic vascular resistance. Regular exercise appears to have protective effect, which may be related to increase HDL cholesterol, lower blood pressure, reduce blood clotting and promote collateral vessels development.<sup>4</sup> This study was conducted to determine the lipid profile and central arterial stiffness in study population.

**MATERIALS & METHODS**

This study was conducted in the department of Physiology in year 2013. It included 120 healthy subjects with no history of smoking, diabetes and hypertension. All were informed regarding the study and written consent was obtained. Ethical approval for the study was taken from institutional ethical committee.

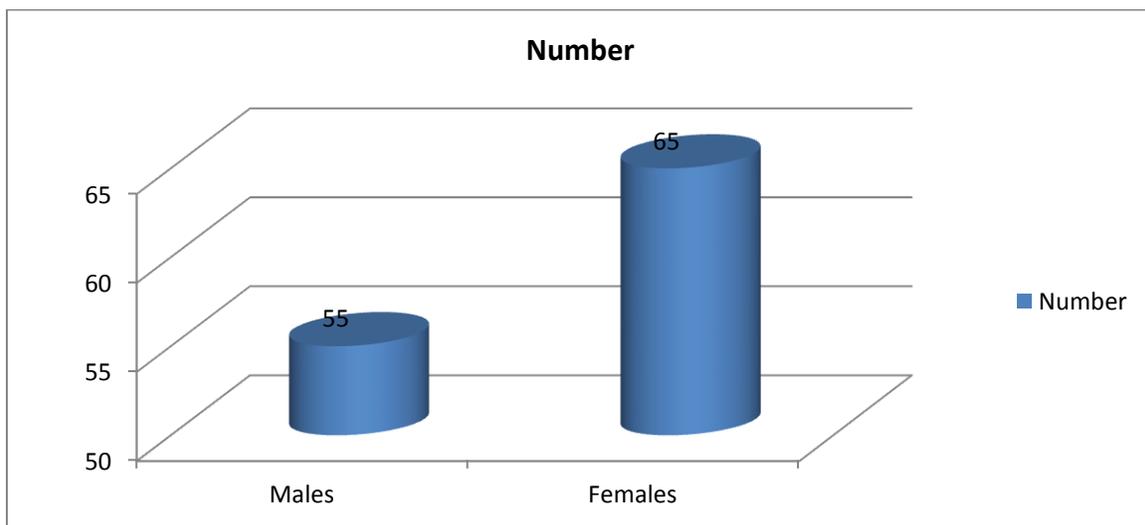
General information such as name, age, gender etc. was noted. Other parameters such as clinical examination, height, weight, biochemical analysis, pulse wave velocity and intima media thickness was done. Pulse wave velocity was determined by Periscop in an 8-channel real-time PC-based simultaneous acquisition and analysis system. CIMT (carotid intima media thickness) was measured from

outside the body, in larger arteries relatively close to the skin by ultrasound. Pulse wave velocity [PWV] is a gold standard for the measurement of arterial stiffness [AS]. Arterial stiffness [AS] is better determined by PWV, reliable and reproducible method. Among elderly patients PWV was the strongest predictor of cerebrovascular mortality. Several non-invasive methods have been developed for quantitatively evaluating arterial wall distensibility using the pulse wave analysis. Arterial stiffness [AS] may be measured using a variety of different techniques which measure either carotid-femoral PWV. Results were tabulated and subjected to statistical analysis. P value less than 0.05 was considered significant.

**RESULTS**

Graph I shows that out of 120 patients, males were 55 and females were 65. The difference was non-significant (P> 0.05). Table I shows s. cholesterol <150 was seen in males (22) and females (34), 150-250 in males (18) and females (20) and >250 in males (15) and females (11). Males have higher Av C-F PWV and Av CIMT as compared to females. The difference was significant (P< 0.05). Table II shows C-F PWV and CIMT according to LDL. It has been observed that both values were significantly higher in males as compared to females (P< 0.05). Table III shows distribution of C-F PWV and CIMT according to HDL. There was decrease in both values as HDL level increases. Level was more in males than females and difference was significant (P< 0.05).

**Graph I** Distribution of patients



**Table I** Distribution of C-F PWV and CIMT according to Serum Cholesterol

Males	Females	S. cholesterol	Av C-F PWV Male	Av C-F PWV Female	Av CIMT Male	Av CIMT Female
22	34	<150	1232.5 ± 262.5	762.5 ± 516.5	0.76 ± 0.23	0.54 ± 0.08
18	20	150-250	1355.3 ± 205.4	1025.2 ± 245.2	1.03 ± 0.25	0.67 ± 0.12
15	11	>250	1830.7 ± 650.2	1358.1 ± 210.1	1.38 ± 0.32	1.02 ± 0.13

**Table II** Distribution of C-F PWV and CIMT according to LDL

Males	Females	S. cholesterol	Av C-F PWV Male	Av C-F PWV Female	Av CIMT Male	Av CIMT Female
22	34	<100	1252.5 ± 222.5	942.5 ± 426.5	0.76 ± 0.23	0.54 ± 0.08
18	20	100-130	1585.3 ± 200.4	1258.2 ± 225.2	1.03 ± 0.25	0.67 ± 0.12
15	11	>130	1890.7 ± 150.2	1450.1 ± 120.1	1.38 ± 0.32	1.02 ± 0.13

**Table III** Distribution of C-F PWV and CIMT according to HDL

Males	Females	S. cholesterol	Av C-F PWV Male	Av C-F PWV Female	Av CIMT Male	Av CIMT Female
22	34	<30	1432.5 ± 302.5	1342.5 ± 226.5	1.26 ± 0.21	1.4 ± 0.18
18	20	30-40	1385.3 ± 250.4	1150.2 ± 285.2	0.93 ± 0.25	0.87 ± 0.20
15	11	>40	1160.7 ± 250.2	750.1 ± 150.1	1.08 ± 0.2	0.92 ± 0.15

## DISCUSSION

High-density lipoprotein (HDL) is positively associated with a decreased risk of coronary heart disease (CHD). As defined by the US National Cholesterol Education Program Adult Treatment Panel III guidelines, an HDL cholesterol level (HDL-C) of 60 mg/dL or greater is a negative (protective) risk factor. To calculate cholesterol ratio, divide high-density lipoprotein cholesterol number into total cholesterol number. An optimal ratio is less than 3.5-to-1. A higher ratio means a higher risk of heart disease.<sup>5</sup> This study determined the lipid profile and central arterial stiffness in study population.

In our study, we included normal subjects of both genders, with 55 males and 65 females. Males have higher Av C-F PWV and Av CIMT as compared to females. A study conducted by Tarchalski J<sup>6</sup> suggested that increased concentration of LDL cholesterol or decreased level of HDL cholesterol is a leading cause of coronary atherosclerosis.

We analyzed C-F PWV and CIMT according to LDL and found that both were significantly higher in males as compared to females. Ludwig M et al.<sup>7</sup> studied arterial stiffness assessed by PWV positively correlated with carotid media thickness, a marker of atherosclerotic burden in the cerebral arteries. Both intima-media thickness and PWV are non-invasive

marker of arterial wall alteration, increases with risk factors for cardiovascular disease.

We also assessed distribution of C-F PWV and CIMT according to HDL. There was decrease in both values as HDL level increases. Level was more in males than females and difference was significant. Penalva RA<sup>8</sup> in his study concluded that the TC/HDL ratio was a marker of severity of CAD in relation to the number of vessels affected, thus demonstrating that the lipid profile can be a determinant of severity in patients with ACS without ST-segment elevation.

Kim DE et al<sup>9</sup> in their study suggested that high-density lipoprotein cholesterol elevation, along with remnant lipoprotein cholesterol reduction and low apo-lipoprotein B/A-I, is associated with prevention of angiographic progression of symptomatic intracranial atherosclerotic stenosis.

## CONCLUSION

Increased level of LDL, VLDL and decrease level of HDL indicates increase in arterial stiffness and carotid intima media thickness.

## REFERENCES

1. Pais P, Pogue J, Gerstein H. Risk factors for acute myocardial infarction in Indians: a Case control study. *Lancet*. 1996;358-63.
2. Meaume S, Benetos A, Henry OF, Rudnichi A, Safar ME. Aortic pulse wave velocity predicts cardiovascular mortality in subjects > 70 years of age. *Arteriosclerosis, thrombosis, and vascular biology*. 2001; 21: 2046-50.
3. Naidu MU, Reddy BM, Yashmaina S, Patnaik AN, Rani PU. Validity and reproducibility of arterial pulse wave velocity measurement using new device with oscillometric technique: a pilot study. *Biomedical engineering online*. 2005; 23:49-55.
4. Mitani HM. A clinical study on PWV, ABI and HS-CRP of the group medical examination Shikoku *Acta Medica*. 2003; 59: 235-243.
5. Benditt EP, Benditt JM. Evidence for a monoclonal origin of human atherosclerotic plaque. *Proc Natl Acad Sci, USA*. 1973; 70: 1753-1756.
6. Tarchalski J, Guzik P, Wysocki H. Correlation between the extent of coronary atherosclerosis and lipid profile. *Molecular and cellular biochemistry*. 2003; 246: 25-30.
7. Ludwig M, von Petzinger-Kruthoff A, Von Buquoy M, Stumpe KO. Intima media thickness of the carotid arteries: early pointer to arteriosclerosis and therapeutic endpoint. *Ultraschall in der Medizin*. 2003; 24: 162-74.
8. Penalva RA, Huoya MD, Correia LC, Feitosa GS, Ladeia AM. Lipid profile and intensity of atherosclerosis disease in acute coronary syndrome. *Arquivos brasileiros de cardiologia*. 2008; 90: 24-30.
9. Kim DE, Kim JY, Jeong SW, Cho YJ, Park JM, Lee JH, Kang DW, Yu KH, Bae HJ, Hong KS, Koo JS. Association Between Changes in Lipid Profiles and Progression of Symptomatic Intracranial Atherosclerotic Stenosis. *Stroke*. 2012; 43: 1824-30.

**Source of support:** Nil

**Conflict of interest:** None declared

This work is licensed under CC BY: *Creative Commons Attribution 3.0 License*.