

Association of lipid profile and central arterial stiffness in the middle aged and the elderly

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Abstract: The Association of lipid profile and central arterial stiffness in the middle aged and elderly. Study comprised of 100 subjects from medical OPDMDM, Jodhpur. Both C-F PWV and CIMT increased with increase in serum cholesterol, serum LDL, & decrease with increase in serum HDL. So risk of atherosclerosis was found higher in males than females and was statistically significant. Increasing serum cholesterol, serum LDL and male gender and decrease inserum HDL level is associated with increase inarterial stiffness and carotid intima-media thickness (CIMT).

Keywords: Carotid-femoral pulse wave velocity (C-FPWV), carotid intima-media thickness (CIMT), arterial stiffness, lipid profile

INTRODUCTION

Both initiation and progression of lesion of atherosclerosis is some how associated in many individuals with markedly increased plasma LDL. The accumulation of lipids within proliferated smooth muscle cells, macrophages, connective tissue matrix, is a common finding in atherosclerosis [1].

Exercise plays an important role in maintaining normal blood pressure and optimizing lipid values. Patients who exercise regularly have been reported to show decreased 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 [2].

MATERIAL AND METHOD

Present study was conducted in the Department of Physiology, Dr. S.N.Medical College, Jodhpur. Participants after understanding the study protocol and procedure were asked to give their written consents for the study.

Inclusion Criteria

1.Healthy individuals with no atherosclerotic risk factors like high BP,diabetes,smoking etc.

2.Men and women of age 20- 50 years were included in the study.

Number Of Cases Studied-100.

Data Evaluation

This is an observational cohort study and the data was analyzed using student's t-test.

Evaluation On A Preformed Proforma History

Complete clinical examination, height,weight, routine biochemical analysis, and Pulse wave velocity and Intima-media thickness were measured.

METHOD

Pulse wave velocity was determined by Periscope (M/S Genesis Medical Systems, Hyderabad, India) in an 8-channel real-time PC-based simultaneous acquisition and analysis system. CIMT was measured from outside the body, in larger arteries relatively close to the skin by ultrasound.

Pulse wave velocity [PWV] is now recognized as a standard method for the measurement of arterial stiffness [AS]. Determination of PWV is most reliable and reproducible method among the various indices of arterial stiffness. [AS] [3-5]. In a cohort of 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 [accesses central arteries].

Observations

Table-1: Distribution of C-F PWV and CIMT According To Serum Cholesterol

Male	Female	S. CHOL	Av C-F PWV Male	Av C-F PWV Female	Av CIMT Male	Av CIMT Female
17	34	<150	1112.5 ± 294.5	866.4 ± 526.8	0.83 ± 0.17	0.58 ± 0.09
24	13	150-200	1329.3 ± 197.1	1081.3 ± 288.1	1.08 ± 0.28	0.79 ± 0.21
9	3	>250	1732.7 ± 680.3	1396.1 ± 239.1	1.31 ± 0.33	1.03 ± 0.12
50	50		1505.4 ± 508.9	1058.9 ± 391.7	1.16 ± 0.33	0.76 ± 0.22

Above table shows that both C-F PWV and CIMT were increased as S. Cholesterol increase. Both C-

F PWV and CIMT were higher in males as compared to females. It was statistically significant.

Tale-2: Distribution Of C-F PWV And CIMT According To LDL

Male	Female	LDL	Av C-F PWV Male	Av C-F PWV Female	Av CIMT Male	Av CIMT Female
17	34	<100	1268.6 ± 207.8	959.1 ± 416.9	1.1 ± 0.26	0.68 ± 0.17
24	13	100-130	1577.3 ± 253	1234.8 ± 223.6	1.14 ± 0.39	0.9 ± 0.19
9	3	>130	1938.9 ± 103.4	1427.6 ± 126.6	1.32 ± 0.24	1.06 ± 0.12
50	50		1594.9 ± 188	1207.1 ± 255.7	1.18 ± 0.89	0.88 ± 0.16

Above table shows that both C-F PWV and CIMT were increased as S. LDL rises. Both C-F PWV

and CIMT were higher in males as compared to females. It was statistically significant.

Table-3: Distribution Of C-F PWV And CIMT According To Serum HDL

Male	Female	S. HDL	Av C-FPWV Male	Av C-FPWV Female	Av CIMT Male	Av CIMT Female
17	34	< 30	1445.1 ± 307.6	1307.1 ± 216.3	1.2 ± 0.23	1 ± 0.17
24	13	30-40	1378.2 ± 256.4	1180.9 ± 280.4	0.96 ± 0.20	0.80 ± 0.22
9	3	> 40	1134.3 ± 245.2	786.3 ± 143.8	0.87 ± 0.17	0.62 ± 0.1
50	50		1319.2 ± 269.7	1091.4 ± 213.5	1.01 ± 0.2	0.80 ± 0.16

Above table shows that both C-F PWV and CIMT were decreased as S. HDL rises. Both C-F PWV and CIMT were higher in males as compared to females. It was statistically significant.

DISCUSSION

Ludwig M *et al.* [6] 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. Multivariate linear regression models showed that male gender, age and blood pressure were independent determinants of both CIMT and PWV while body mass index and LDL-cholesterol were independent determinants of CIMT only.

In the present study there was significant correlation found between a C-F PWV and CIMT and dyslipidemia. Both were elevated in CVA patients with deranged lipid profile. It is hold up by Mitoni Hiroki [7] who in Japan studied Atherosclerosis, especially, the pathogenesis of ischemic heart disease is exposed to attention the relations of chronic inflammations and its bio-maker. PWV was positively related to serum lipid levels.

Tarchalski J [8] concluded that, increased concentration of low density lipoprotein (LDL) cholesterol or decreased level of high density lipoprotein (HDL) cholesterol are important risk factors for coronary atherosclerosis. However, an independent association of triglycerides (TG) with atherosclerosis is uncertain. The study was conducted in 141 patients (53.6 +/- 7.8 years old; 32 female) who underwent a routine coronary angiography for CAD diagnosis. There

was a negative correlation between Gensini Score and HDL cholesterol ($r = -0.396$; $p < 0.001$). In angina pectoris patients with no previous myocardial infarction, the extents of coronary atherosclerosis is positively correlated with pro-atherogenic lipids, i.e. total cholesterol, LDL cholesterol and TG and negatively correlated with anti-atherogenic HDL cholesterol.

Penalva RA [9]. The relationship between coronary artery disease (CAD) and dyslipidemia in acute coronary syndromes has been rarely demonstrated in clinical and epidemiological studies. A total of 107 patients were included; 94(88%) had CAD, of which 50(53.2%) were males with predominance of multi-vessel disease. As regards the lipid profile, 64(59.8%) patients were observed to have $TC < 200$ mg/dl, 33(30.8%) had $HDL < 40$ mg/dl, and 38(35.5%) had $LDL < 100$ mg/dl. 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. Dong-Eog Kim [10] *et al.* studied predictors of progression of intracranial atherosclerotic stenosis that have not been clearly identified. They investigated whether poststroke changes in lipid profiles would affect the prognosis of symptomatic intracranial atherosclerotic stenosis. Prospective multicenter study to demonstrate 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.

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