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Study on Dyslipidemia in Young Adults (20-40 Yrs) and Its Relation to Various Risk Factors in Tertiary Centre of Lucknow, UP

Jyoti Verma

Assistant Professor, Medicine Department, Integral Institute of Medical Sciences and Research, Lucknow, UP, India

ABSTRACT

Background: Lipid disorders i.e., Dyslipidemia refer to abnormalities of cholesterol, including low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides.

Study objectives: To study the relation of dyslipidemia in young asymptomatic adults and to determine and correlate the significance of associated risk factors.

Patients: This was a cross sectional study consisting of consecutively selected 200 asymptomatic adults aged between 20-40 years who visited Integral institute of medical sciences and research, Lucknow, UP.

Patient information was collected with the help of the questionnaire after obtaining an informed consent and it included details such as; age, gender, anthropometric measurements, lifestyle related factors, clinical & family history, glucose and lipid analysis. Risk factors for dyslipidemia (high LDL-C, low HDL-C, high triglycerides) include physical inactivity, obesity, abdominal obesity, metabolic syndrome, hypertension,

Results: Our study showed that elevated serum lipids were more prominent in 31-40 year age group as compared to ≤30 years, 75% of total, which means the risk of dyslipidemia increases as the age advances. Dyslipidemia was found most significant amongst the alcoholic and the smokers (p value being significant < 0.05). Prevalence was more in males indicating Indian men being at a higher risk for dyslipidemia. Body mass index correlated with hypertriglyceridemia, increased LDL and increased VLDL (p value -0.001) than total cholesterol (p value- <0.05). The pattern of dyslipidemia correlated significantly with the history of alcohol consumption and smoking.

Those with diabetes has shown significant relation to dyslipidemia in this age group.

Conclusion: Dyslipidemia increases with age and high BMI, where males are more than females. Hypertriglyceridemia is more seen than increased total cholesterol. Alcohol and smoking significantly increases the proportion of lipid disorders. Dyslipidemia increased the episodes of hypertension and number of hypertensives.

Keywords: Dyslipidemia, Diabetes, Hypertension.

INTRODUCTION

Definitions used for this study:

Dyslipidemia: National Cholesterol Education Programme (NCEP) guidelines were used for definition of dyslipidemia as follows:

Hypercholesterolemia – serum cholesterol levels ≥200 mg/dl (≥5.2 mmol/l).

Hypertriglyceridemia – serum triglyceride levels ≥150 mg/dl (≥1.7 mmol/l).

Low HDL cholesterol – HDL cholesterol levels <40 mg/dl (<1.04 mmol/l) for men and <50 mg/dl (<1.3 mmol/l) for women.

High LDL cholesterol – LDL cholesterol levels ≥130 mg/dl (≥3.4 mmol/l) calculated using the Friedewald equation.

High total cholesterol to HDL-C ratio: This is defined as a total cholesterol to HDL-C ratio of ≥4.5.

Isolated hypercholesterolemia: Serum cholesterol ≥200 mg/dl and triglycerides <150 mg/dl; **Isolated hypertriglyceridemia**: Serum triglycerides ≥150 mg/dl and cholesterol <200 mg/dl; **Isolated low HDL-C**: HDL-C ≤40 mg/dl (male) and ≤50 mg/dl (female) without hypertriglyceridemia or hypercholesterolemia.

Diabetes: Individuals diagnosed by a physician and on antidiabetic medications (self-reported) and/or those who had fasting CBG ≥126 mg/dl (≥7 mmol/L) and/or 2-hr post-glucose CBG value ≥220 mg/dl (≥12.2 mmol/L).

Impaired fasting glucose [IFG]: Fasting CBG ≥110 mg/dl (≥6.1 mmol/L) and <126 mg/dl (<7 mmol/L) and 2-hr post-glucose value <160 mg/dl (<8.9 mmol/L).

Impaired glucose tolerance [IGT]: Two-hour post-glucose CBG ≥160 mg/dl (≥8.9 mmol/L) but <220 mg/dl (<12.2 mmol/L) and fasting value <126 mg/dl (<7 mmol/L).

Hypertension: Individuals diagnosed by a physician and on antihypertensive medications (self-reported) and/or those who had systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg – Joint National Committee 7 (JNC7) Criteria.

Obesity: Generalized obesity was defined as BMI ≥25 kg/m^2; overweight as BMI 23–25 kg/m^2 and abdominal obesity was defined as waist ≥90 cm (males), ≥80 cm (females) using Asia-Pacific guidelines for south Asians.

Coronary Artery Disease (CAD): CAD was diagnosed based on positive medical history (documented myocardial infarction (MI), angina pectoris and coronary artery bypass graft) and/or ischemic changes on a conventional 12-lead ECG which included ST-segment depression (Minnesota codes 1-1-1 to 1-1-7) or Q-wave changes (Minnesota codes 4–1 to 4–2).

The prevalence of obesity is rising to epidemic proportions at an alarming rate in both developed and less-developed countries around the world, [1] and many Indian studies have shown that the prevalence of overweight and obesity ranged between 30% and 65% among the urban population. [2] Body mass index (BMI; in kg/m^2) is widely used for the classification of overweight (BMI = 25 kg/m^2) and obesity (BMI = 30 kg/m^2) in men and women. [3]

Metabolic syndrome (MetS) is a complex web of metabolic factors that are associated with a 2‑fold risk of cardiovascular diseases (CVD) and a 5‑fold risk of diabetes. MetS is a constellation of multiple cardiometabolic abnormalities including truncal (central) obesity, borderline and high blood pressure (BP), high fasting glucose, high triglycerides (TGs), and low high‑density lipoprotein cholesterol (HDL‑C). [4-8]

Hypertension and dyslipidemia are important risk factors for cardiovascular disease. Coexistence of hypertension and dyslipidemia is often observed in daily clinical practice, and this empirical observation is consistent with baseline characteristics of clinical study participants. [9–12] Population-based epidemiological studies have also reported that gradual Increases in blood pressure (BP) or prevalence of hypertension are associated with increases in blood lipid levels. [13-16] One possible explanation for these relationships is that hypertension and dyslipidemia share common pathophysiological etiologies, such as obesity and the resulting dysregulation of adipocytokine release from adipose tissue. [17] Furthermore, dyslipidemia adversely affects functional and structural arterial properties and promotes atherosclerosis. [18-20] These changes may impair BP regulation, which, in turn, predisposes individuals with dyslipidemia to development of hypertension.

Dyslipidemia and hypertension were the two widely recognized independent key risk factors for development of CVD [21-23] and these may constitute Metabolic syndrome (MetS). [24,25] MetS is a group of clinical and biochemical abnormalities that confer a greater risk factor for type-2 DM and CVD. [26] The risk is associated with concomitant hypertension and dyslipidemia, is an additional sum of the individual risk factors. [27,28] Some of the studies found that the treatment of dyslipidemia has favorable effects on both coronary and cerebrovascular events, than to independent decrease the blood pressure benefit. [29,30]

Alcohol intake raises the levels of high-density lipoprotein cholesterol (HDL), [31-33] a fact that may explain, at least in part, its apparent protective effect against coronary heart disease. [34-35]

In patients with Type 1 diabetes in good glycemic control, the lipid profile is very similar to lipid profiles in the general population. [37] In contrast, in patients with Type 2 diabetes, even when in good glycemic control, there are abnormalities in lipid levels. [23-26] It is estimated that 30-60% of patients with Type 2 diabetes have dyslipidemia. [36,38] Specifically, patients with Type 2 diabetes often have an increase in serum triglyceride levels, increased VLDL and IDL, and decreased HDL cholesterol levels. Non-HDL cholesterol levels are increased due to the increase in VLDL and IDL. LDL cholesterol levels are typically not different than in normal subjects but there is an increase in small dense LDL, a lipoprotein particle that may be particularly pro-atherogenic. As a consequence there are more LDL particles, which coupled with the increases in VLDL and IDL, leads to an increase in Apo B. [39-41] Studies have shown that the anti-oxidant and anti-inflammatory functions of HDL isolated from patients with diabetes are reduced, indicating that HDL levels per se may not fully reflect risk. [42] Additionally, the postprandial increase in serum triglycerides is accentuated and elevations in postprandial lipids may increase the risk of cardiovascular disease. [39-41] It should be recognized that these lipid changes are characteristic of the alterations in lipid profile seen in obesity and the metabolic syndrome (insulin resistance syndrome). [43]

The effects of extended periods of sedentary behavior in otherwise physically active persons have begun to be elucidated, and they seem to be characterized by metabolic alterations commonly seen in diabetogenic and atherosclerotic profiles). numerous epidemiological studies have shown indicators of physical inactivity, such as TV viewing, driving in a car and sitting, are strongly related to the risk for developing dyslipidemia, [44,45] obesity ,type 2 diabetes ,hypertension metabolic syndrome and CVD. [45]

MATERIALS AND METHODS

Sample size: 200

Inclusion criteria:

- 20-40 years
- patients with dyslipidemia
- Fasting for 8-12 hours
- Risk factors like Smokers, alcoholic, Diabetics, hypertensives

Exclusion criteria:

- pregnancy
- patients with liver disease
- below 20 years and above 40 years
- the subjects in study were collected from people attending outpatient and from ward admitted in the integral institute of medical sciences and research, Lucknow, UP.

They were asked questionnaire that includes questions about certain socio-demographic variables (age, gender, occupation, marital status and educational level), history of chronic diseases for the patient and his family (DM, hypertension, IHD, stroke, and hyperlipidemia), history of intake of medications (steroids, contraceptive pills, B-blockers, diuretics), social history including (physical activity, smoking, drinking alcohol) Anthropometric Measures: * Weight was measured with subjects in light clothes without shoes.

* Height was measured with a tape. Subjects were requested to stand upright without shoes with their back against the wall, heels together and eyes directed forward.

* Body Mass Index: BMI is calculated as *BMI = Weight (kg) / Square Height (m^2).*

CLASSIFICATION:

Underweight (below 18.5 kg/m^2), normal range (18.5-24.9 kg/m^2), overweight (25-29.9 kg/m^2), class I obesity (30-34.9kg/m^2), class II obesity (35-39.9 kg/m^2) and class III obesity (>40 kg/m^2)

Blood Pressure Measurement: The study sample was assessed using standard criteria formulated by the US Seventh Joint committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension was diagnosed if there was a prior diagnosis by a physician, current use of blood pressure lowering medications, or measured blood pressure values of ≥140 mmHg systolic or ≥90 mmHg diastolic on ≥2 occasions (15). Measurement of fasting lipid profile and fasting blood sugar*:* Blood sample was drawn from an antecubital vein in all subjects after 9-12 hours fasting. Low-density lipoprotein (LDL) cholesterol was calculated according to the Friedewald formula: *LDL cholesterol = TC – (TG/5 + HDL cholesterol)*.
Hypertriglyceridemia was defined as a fasting plasma concentration > 150 mg/dl. Hypercholesterolemia was defined as a total cholesterol >200 mg/dl. Hypoalphali-poproteinemia was considered present if HDL-cholesterol was <40 mg/dl, LDL cholesterol is considered elevated if the values> 130 mg/dl and TC/HDL ratio is considered elevated if the value > 4.

Smokers and ex-smokers were included in the study. Diabetes was diagnosed if there was a previous medical diagnosis or in the presence of two readings of a fasting plasma glucose value > 126 mg/dl and no previous history of diabetes.

Statistical analysis

Various serum lipid levels were considered as primary outcome variables. Categorical variables were presented as frequencies and percentages. Quantitative variables were presented as mean and standard deviation. The lipid levels were compared between the hypertensive patients and the controls by unpaired t-test .The association between the categorical explanatory and outcome variables was done by cross tabulation and calculating the corresponding odds ratio and 95% CI. Chi square test was used to assess the statistical significance of the association. P value <0.05 was considered as statistically significant. IBM SPSS version 21 was used for statistical analysis. The Student's t-test was used for comparison between categorical variables, i.e. lipid profile, high-BMI and normal-BMI subjects at $P \leq 0.05$.

OBSERVATIONS

GENDER DISTRIBUTION-

Out of 200 adults, 120 are males and 80 are females.

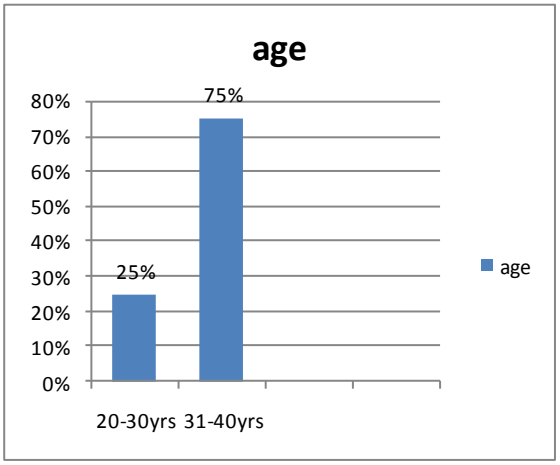

Fig./Table 1: dyslipidemia in age groups

Table 2: dyslipidemia in hypertensives

Parameters	Hypertensives (N= 110)	Healthy (N=90)	P-value
Total cholesterol	198+ 34.75	158+ 16.70	<0.001
Triglycerides	172+ 60.8	128+22.7	<0.001
HDL	39.78+6.66	55.2+ 4.8	<0.001
LDL	120 + 40	76 + 10.2	<0.001
VLDL	35 + 12.2	25.2 + 4.8	<0.001

Table 3: comparison of dyslipidemia to BMI in males

	High BMI (SD)	Normal BMI (SD)	P-value
TOTAL CHOLESTEROL	206.82(21.4)	175.21(18.4)	<0.05
LDL	125.55(14.3)	76.43(113.7)	<0.001
HDL	33.38(16.1))	35.72(14.9)	0.65
VLDL	42.31(15.8)	33.26(14.1)	<0.001
TRIGLYCERIDE	174.59(163)	119.23(15.4)	<0.001

Table 4: comparison of dyslipidemia to BMI in females

	HIGH BMI (SD)	NORMAL BMI(SD)	P-value
Total cholesterol	202.45 (19.1)	171.33(18.4)	<0.05
LDL	120.71(13..5)	73.58(15.1)	<0.005
HDL	32.51(12.8)	34.86(16.3)	0.64
VLDL	41.58(13.2)	30.18(14.1)	<0.001
TRIGLYCERIDE	170.98(17.1)	115.45(16.5)	<0.001

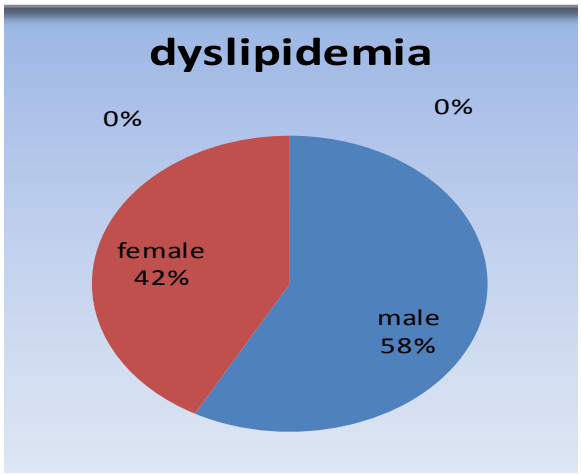

Fig./Table 5: dyslipidemia in males and female

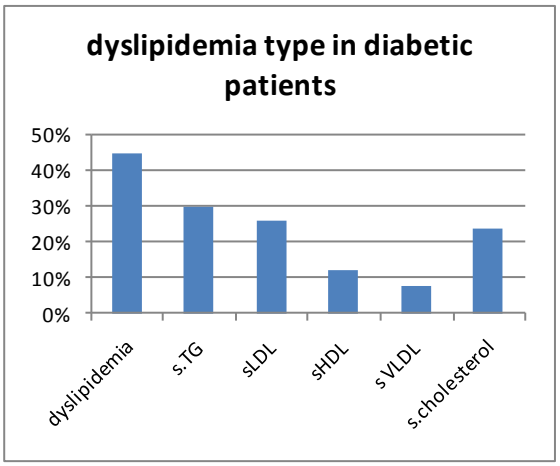

Fig./Table 6

Table 7: comparison of lipid profile among non-smokers and mild,moderate and severe smokers

Lipid profile	non-smokers	Heavy smokers	p-value	Moderate smokers	p-value	Mild Smokers	p-value
Total cholesterol	160.20	182+ 40	.001	172±30.17	.001	170±32	.001
Serum Triglyceride	92.88	190±37	.001	155±50.20	.001	120±40	.001
Serum LDL	88.90	138 ±35	.001	122±23.88	.001	118.30± 20	.001
Serum VLDL	20.86	42±80	.001	35.77±5.60	.001	32.38±4.1	.001
Serum HDL	45.50	32±92	.001	33.10±5.10	0.001	32.90±4.2	.001

Table 8: lipid profile among smokers and non-smokers

Lipid profile	Non-smokers	smokers	p-value
Total cholesterol	160.20	172.16±35.88	<0.01
s.triglyceride	92.88	150.90±82.90	0.001
s.LDL	88.80	98.80±22.30	<0.001
s.VLDL	20.86	34±8.02	<0.001
s.HDL	45.50	33.20±5.10	<0.001

RESULTS AND DISCUSSION

This study revealed higher prevalence of hyperlipidemia in young adults above 30 years i.e., 75% of total group.

Hypertension is an important risk factor for CVS disease and it becomes even more important when associated with hyperlipidemia. In the present study significant association was found between young adults and dyslipidemia (P<0.001).

Dyslipidemia is frequently associated with obesity no doubt, and it plays an important role in the development of atherosclerosis and thus cardiovascular disease in obese individuals.

All the components of the dyslipidemia including higher TGs, increased LDL and high VLDL have shown to be atherogenic and is significant (p<0.05- 0.001)

Dyslipidemia is higher in high BMI men than in women. An Indian study performed by Pandya et al. inferred that diabetic obese patients are more prone to develop dyslipidemias than the non-obese patients. The present study showed that cholesterol was significantly higher in high BMI people compared with people with normal BMI. These findings correlate well with the findings of Philip et al. From this study, it can be inferred that LDL-C was significantly higher in people with high BMI compared with people with normal BMI, while the values of HDL-C did not show any significant association between the two groups (high BMI and normal BMI); these findings correlate well with the studies of Grundy and Barnett. In our study, the TG levels were significantly higher among the high BMI group when compared with the normal BMI group, and the findings are in par with the study performed by Lemieux et al. In rural areas, the prevalence has increased in recent studies.

In our study most of the population are of low socio-economic background.

Smoking is the major risk factor in developing world. In the present study it was revealed that total cholesterol, LDL, VLDL, HDL and TG alteration were statistically significant in smokers as compared to non-smokers.

S.cholesterol and LDL were related to smokers significantly ($p<0.05$) when compared to non-smokers. However TG and VLDL were not significant ($p <0.01$) and HDL were higher in non-smokers than in smokers ($p<0.01$)

Study by Mokoto et al., has shown only mean TG level difference that was statistically significant ($p<0.05$).

Mean HDL levels were higher in non-smokers ($p<0.05$). Studies by Neki & Anile et al. also showed similar results. Thus, smoking induces dyslipidemia and has increased risk of CAD.

Along with dyslipidemia, diabetes is an important component of metabolic syndrome. In this study, dyslipidemia was present in 45% of young adults, of which maximum increase was observed in serum triglyceride, serum LDL and total cholesterol than low HDL and increased VLDL.

CONCLUSION

Significant associated risk factors with hyperlipidemia were age, BMI, newly diagnosed or uncontrolled diabetic and hypertensive patients, cholesterol/fat rich diet. This study revealed the high prevalence of hyperlipidemia in young adults individuals aged between (20-40) years old. Increased triglycerides, increased cholesterol was mostly observed than low HDL. Smoking directly influences dyslipidemia thus increasing the chances of CVS diseases.

Preventable risk factors will surely help in reducing the overall burden on hospitals. Healthy society needs early identification and management of such prevalent causes. In this study, as the various risk factors were present in all groups, relation that was established with dyslipidemia can vary when separate studies are made.

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