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Original Article

Serum lipid profile in prepubertal, reproductive and postmenopausal women

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ABSTRACT

The present study was undertaken to know serum lipid profile changes in prepubertal, reproductive and postmenopausal periods in women. The fasting blood samples were collected from 120 healthy volunteers from Bangalore and total cholesterol, triglyceride, VLDL, HDL were measured by enzymatic method. LDL was calculated using Friedewald's equation, HDL/LDL, HDL/cholesterol ratios were obtained. The results showed statistically significant increase in total cholesterol ($p<0.0001$) with age and also statistically significant increase in TG ($p<0.0001$), VLDL ($p<0.0001$) and LDL ($p<0.0001$) were observed between reproductive and postmenopausal women. HDL showed statistically significant decrease in postmenopausal women compared to reproductive age group. HDL/LDL and HDL/cholesterol ratios decreased with increasing age from reproductive to postmenopausal period ($p<0.0001$).Thus it can be concluded that serum lipid profile changes with possibly mediated by changing hormonal profile and sex steroids, especially estrogen which has role in lipid metabolism, thereby indirectly on coronary artery disease.

1. Introduction

With the increase in life expectation to 64 years in females in India[1], number of women living after menopause is increasing. Women from the time of intrauterine life till the end of life experience different stages in her reproductive life under the influence of female hormones, which is a physiological phenomenon.

These hormones, which are secreted in minute quantity, undergo a complex and ever changing milieu according to different phases of her reproductive life. These hormones not only play an important role in her reproduction but also influence other systems in the body. Metabolism is one system, which is affected in a significant way.

Oestrogen, one of the important female sex hormone has a role in lipid metabolism, which affects the serum cholesterol and lipoprotein levels thereby indirectly having a role in coronary heart disease, has a definite outcome in life expectation. Oestrogen undergoes a definite change from prepubertal to postmenopausal age and also its role in serum cholesterol and lipoprotein levels can be assessed by measuring them biochemically. In order to find the lipid profile changes after menopause who are at increased risk for cardiovascular disease, lipid profile of the prepubertal and reproductive women done for comparison. So this present study is done to study the serum lipid profile in prepubertal, reproductive and postmenopausal women of Bangalore City.

2. Material and Method

2.1. Subjects

Healthy attenders accompanying patients of Vani Vilas Hospital, Victoria Hospital, Bowring and Lady Curzon Hospital and students of Government Higher Primary School, Chamrajpet, Bangalore with no evidence of metabolic or endocrinal abnormalities , hypertension or coronary disorder were selected randomly for prepubertal, reproductive and postmenopausal group consisting of 40 subjects in each group. Subject should belong to the specified age range

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Prepubertal 8-10 years
Reproductive 15-45 years
Postmenopausal 47-55 years

Thorough history was taken including menstrual, obstetric, and gynaecologic and birth history in case of prepubertal subjects and clinical examination was done before accepting them as subjects for the study. Written informed consent was taken from the subjects.

2.2. Collection of Blood Samples

About 3 ml of fasting blood (minimum 12 hours fasting) was collected by using sterile disposable syringe in dry and clean centrifuge tube taking all sterile precautions , serum was separated from the clot within 30 minutes and analysed. Serum total cholesterol was estimated by dynamic extended stability CHOD-PAP method, HDL-cholesterol by phosphotungstic acid method, Triglycerides by dynamic extended stability with lipid agent clearing agent GPO-Trinder method.

LDL-cholesterol and VLDL–cholesterol were determined using Friedewald's Formula

LDL – cholesterol = Total cholesterol – [HDL cholesterol + (triglyceride / 5)]

VLDL – cholesterol = triglyceride / 5

2.3.Statstical treatment of the data

The data collected in this study was analyzed statistically by computing the descriptive statistics viz. mean, standard deviation, and 95% confidence interval. The comparison between the difference in mean of these groups prepubertal, reproductive and postmenopausal women were analyzed using analysis of variance (ANOVA) based on age and the student T-test has been used to compose lipid levels between prepubertal and reproductive ages, as well as reproductive and postmenopausal women. The results are considered statistically significant whenever p0.05.

3.Results

The table 1 shows the mean , standard deviation ,t-value & p-value for TC, TG, HDL cholesterol, LDL cholesterol VLDL cholesterol, HDL/LDL & HDL/Chol in prepubertal & reproductive females. Except for TC level no other values showed statistically significant difference. TC level increased from prepubertal to reproductive age groups with a mean value of 149.1014.14 to 162.4821.27 with t=0.3312 & 'p'value <0.001 which was statistically significant. So the TC is increasing with age.

Mean values of total cholesterol, TG, HDL cholesterol, LDL cholesterol, and VLDL cholesterol were 166.4826.36, 117.641.44, 42.933.96, 100.425.43 and 23.348.52 respectively.

The table 2 shows the mean, standard deviation, t-value & p-value for TC, TG, HDL cholesterol, LDL cholesterol ,VLDL cholesterol, HDL/LDL & HDL/Chol in reproductive & postmenopausal females.

'T' test showed significance for TC, TG, HDL cholesterol, LDL cholesterol , VLDL cholesterol, HDL/LDL & HDL/Chol between two age groups with t=4.783, 4.273, 2.272, 4.180, 3.400, 4.086, 4.937 respectively & p<0.001. TC, TG, LDL, VLDL values were increased in postmenopausal women compared to reproductive age group, while HDL- chol ,HDL/LDL & HDL/chol were decreased in postmenopausal women compared to reproductive age group.

Table 1. Comparison of pre-pubertal and reproductive ages

	Age (yrs)	No. of subjects	Mean	SD	t-value	p-value
TC (mg%)	8-10	40	149.10	14.14	3.312	<0.001
	16-45	40	162.48	21.27		
TG (mg%)	8-10	40	103.50	36.94	0.366	>0.716
	16-45	40	106.35	32.66		
HDL (mg%)	8-10	40	42.65	3.97	1.736	>0.087
	16-45	40	44.23	4.14		
LDL (mg %)	8-10	40	85.75	15.17	2.579	<0.012
	16-45	40	96.50	21.56		
VLDL (mg%)	8-10	40	21.25	7.7	0.016	>0.988
	16-45	40	21.28	6.51		
HDL / LDL	8-10	40	0.52	0.14	1.152	>0.253
	16-4	40	0.48	0.13		
HDL / Chol	58-10	40	0.29	0.04	0.994	>0.323
	16-45	40	0.28	0.05		

Table 2. Comparison of reproductive and postmenopausal ages

	Age (yrs)	No. of subjects	Mean	SD	t-value	p-value
TC (mg%)	16-45	40	162.48	21.27	4.783	<0.0001
	46-55	40	187.85	25.94		
TG (mg%)	16-45	40	106.35	32.66	4.273	<0.0001
	46-55	40	142.95	43.21		
HDL (mg%)	16-45	40	44.23	4.14	2.272	<0.008
	46-55	40	41.90	3.47		
LDL (mg %)	16-45	40	96.50	21.56	4.180	<0.0001
	46-55	40	118.95	26.25		
VLDL (mg%)	16-45	40	21.28	6.51	3.400	<0.001
	46-55	40	27.50	9.58		
HDL / LDL	16-45	40	0.48	0.13	4.086	<0.0001
	46-55	40	0.37	0.11		
HDL / Chol	16-45	40	0.28	0.05	4.937	<0.0001
	46-55	40	0.23	0.04		

4.Discussion

Goswami K and Bandyopadhyay A[2] showed that the average HDL-cholesterol was significantly higher in female (p<0.0001), the same change was not observed beyond 60 years. The females also showed high TC levels beyond 60 years (p<0.01), which was

further increased beyond 70 years with a constant increase in LDL-cholesterol.

Gandhi BM[3] – showed that TC increased with age in both sexes.. Triglycerides in plasma increased with age. Gupta KK et al[4] showed a continuous rise of cholesterol level in children with advancing age was observed on analysis of lipid values.

Bonithon-Kopp C[5] concluded that total cholesterol and LDL-cholesterol significantly increased in postmenopausal women. Gruchow et al[6], Farish E et al[7] explained that higher HDL-cholesterol levels among postmenopausal estrogen replacement may indicate a biologic mechanism by which postmenopausal oestrogen lowers the risk of coronary occlusion.

Nerbrand C et al[8] Smiti Nanda et al[9] suggested that loss of endogenous sex steroids contribute substantially to increased atherogenic lipid profile. Hormone replacement therapy may partly reverse these differences.

Notelovetz M et al[10] showed that cyclic esterone sulphate raised HDL-cholesterol, lowered LDL-cholesterol levels and raising the HDL / LDL ratio at 6, 9 and 12 months of treatment in postmenopausal women with elevated baseline total cholesterol.
Fahraeus L et al[11] – showed that oral administration of oestradiol-17 increased the HDL and decreased the LDL thus raising the HDL / LDL cholesterol ratio.

So the postmenopausal changes in lipid profile can be explained as follows. Estradiol level[12] in premenopausal (reproductive) women is 40-350 pg/ml as compared to 13 pg/ml in postmenopausal women. So the estradiol level decreases drastically after menopause. The high estrogen during reproductive age has a beneficial effect lowering the LDL-cholesterol by acting on LDL-receptors.[13]

The increase in HDL-cholesterol levels in response to HRT is caused by increased production of apolipoprotein A-I, the major apolipoprotein of HDL-cholesterol and by decreased hepatic lipase activity effects that enhance the uptake of HDL-cholesterol and the catabolism of HDL2 [14]. This may explain high values of HDL before menopause when oestrogen level are high. So as LDL increases, HDL decreases after menopause, HDL/LDL ratio decreases significantly after menopause. Before menopause the low value of serum cholesterol can be attributed to high estrogen level[15] and after menopause estrogen level decreases, this may contribute to increase in cholesterol level.[16] So after menopause HDL decreases and serum cholesterol increases leading onto significant decrease in HDL/cholesterol. Rise in TG and VLDL can be attributed to age.

5. Conclusion

At the end of this study we can conclude that total cholesterol increases significantly with age, which can be attributed to age.[2,3,4] Triglyceride, VLDL and LDL increases significantly from reproductive period to postmenopausal period. The raise in TG and VLDL can be attributed to age and the rise in LDL can also be attributed to age and the significant increase in LDL after menopause can be explained by changes in hormone levels. This in agreement with school of thought comprising Goswami K and Bandyopadhyay A, Gandhi BM, Bonithon Kopp C. HDL increases upto menopause and later decreases. The decrease in HDL after menopause can be attributed to hormonal changes.[2,3,5] HDL/LDL ratio and HDL/cholesterol ratio decreases with age, more between reproductive and postmenopausal age group. As HDL decreases and LDL cholesterol increases after menopause, HDL/LDL and HDL/Cholesterol decreases with age, which is self-explanatory. This in agreement with Notelovitz M et al and Fahraeus L et al.These changes may be attributed to postmenopausal changes in female sex steroidal hormonal profile, with decline in both estrogen and progesterone.

The best single indicator of the likelihood of developing atherosclerotic heart disease is not total plasma cholesterol but rather the ratio of plasma LDL to HDL-cholesterol, the lower the ratio lower the risk. LDL-cholesterol[17] is often designated 'bad' cholesterol since high levels of it in the plasma are associated with increased deposition of cholesterol in arterial wall and higher incidence of heart attacks, using the same criteria HDL-cholesterol has been designated good cholesterol.

Estrogen not only lowers total cholesterol and LDL but raises HDL, which explains in part why premenopausal women have so much less coronary artery disease than men. After menopause the cholesterol values and coronary artery disease rate in women become similar to those in men. Oestrogen, one of the important female sex hormone has a role in lipid metabolism, which affects the serum cholesterol and lipoprotein levels thereby indirectly having a role in coronary heart disease, has a definite outcome in life expectation. So hormone replacement in postmenopausal women has beneficial effects in maintaining lipid profile which is less atherogenic. Estimation of female sex hormones may reveal more facts, which was not undertaken due to practical problems.

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