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Original Research Article

Menopausal dyslipoproteinemia in breast carcinoma patients: a laboratory analysis

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ABSTRACT

Background: Seldom any precedent work has aimed to study the influence of age and menopausal status towards serum lipoproteins levels in breast cancer. Owing to influence of sex-steroids over lipid handling by oestrogen-dependent breast cells, their serum levels might reveal insinuating facts, if menopausal status-wise analysis is attempted. Ascertainment of serum lipid/lipoprotein aberrations in breast carcinoma; and substantiation of their behaviour with age and menopause among breast cancer and healthy subjects.
Methods: Laboratory analyses of serum total cholesterol (TC), high density lipoprotein cholesterol (HDL-c), low and very low density lipoproteins (LDL-c, VLDL-c) and triglycerides (TG) were done among breast cancer subjects (n=50) and healthy women (n=50) with respect to their age (3 sub-groups viz. 25-40, 41-55 and 56-75 years) and menopausal status. Grouped numerical data were subjected to intra- and inter-group comparisons using Student's unpaired-'t'-test and ANOVA with Post-Hoc comparisons using Tukey-HSD (Honestly-Significant-Difference) Test.
Results: Higher serum LDL-c and TC with lower HDL-c levels were observed among breast carcinoma subjects than healthy women (P=0.004, 0.003, 0.009 respectively). Serum LDL-c elevation in peri- and post-menopausal age (P=0.009), while lowered HDL-c specifically during post-menopausal age (0.004) were significantly evident in breast cancer subjects.
Conclusions: Breast carcinoma has obvious alliance with serum LDL-c, TC and HDL-c aberrations. LDL-c has specific variability during peri-menopausal and post-menopausal ages among breast cancer subjects. HDL-c alteration is mainly concerted for post-menopausal age; while total cholesterol could be a peril throughout. The odds of pooled effect of such aberrations in cancer causation cannot be underrated.

Keywords: Breast cancer, Cholesterol, HDL, Lipoproteins, LDL, Menopause

INTRODUCTION

Biosynthesized cholesterol and exogenous cholesterol have been suggested as sources of cholesterol for cells of the body. It has also been proven that if exogenous cholesterol supplementation is retarded; or cholesterol biosynthesis is inhibited, cellular growth can be blocked. This way the cellular growth has been resolutely linked to the availability of cholesterol.[1,2] Cholesterol has been notorious for its role in carcinogenesis owing to the fact that mammary tissue handles plasma lipids under the influence of sex steroid hormones.[3,4] Such hormonal influence may increase the uptake of cholesterol by oestrogen dependent tissue like breast, posing it to the augmented risk of carcinogenesis.[5] Based on these phenomena, a link has been established between breast cancer, serum lipids and oestrogen. As some earlier studies have already reported altered serum lipid and lipoprotein cholesterol levels in breast cancer subjects, the possible contribution of plasma lipids through oestrogenic influence on cellular metabolisms of plasma lipids in oestrogen-triggered carcinogenesis cannot be

underrated. Serum High Density Lipoprotein cholesterol (HDL-c) level regulation is sturdily affected by the body oestrogen status; while Low Density Lipoprotein cholesterol (LDL-c) has been particularly found susceptible to oxidation.[6,7] The 'oxidised-LDL' formed as a result of this may enhance the lipid peroxidation of cellular bio-membranes of oestrogen sensitive breast cells.[8] Age and menopause do play a very momentous role in woman's life towards determining serum oestrogen levels. Menopause in women is the point of switch from high to low serum oestrogen concentrations. Post-menopausal age is distinguished with ebbed serum oestrogen levels.[9] This contrast in oestrogen status might execute variable lipid and lipoprotein manipulation by oestrogen dependent breast cells.

In view of the same, hardly ever has any study focused on their analyses with respect to age and menopausal status in breast cancer women. Thus, bearing these specifics in mind, the present study was taken up to rule out if any significant variation exists in serum lipoprotein and lipid levels in breast cancer patients than healthy women. The comparisons have been further extended in this study to a detailed inter- and intra-group analyses with respect to age and menopausal status of subjects owing to the fact that menopause plays very crucial role towards oestrogen levels in blood.

Objective of present study was to evaluate serum lipoprotein and cholesterol levels in patients with breast cancer and healthy subjects; and their comparison and ascertainment of influence of age and menopausal status of women over serum cholesterol and lipoprotein levels among breast cancer subjects.

METHODS

The study was performed at the department of Biochemistry of a tertiary care medical institution located in central India. Having obtained the Institutional Ethics Committee approval and informed written consents from all the study participants, subjects' enrolment in the study was done as follows:

Study Sample Population

For recording the numerical data in this observational study, a cross-sectional case-control design was utilized. Sample size (N) was estimated with formula $N= 4Z\alpha^2S^2/W^2$ where Zα=Standard normal deviate (1.96), W=Desired total width of confidence interval (5%), S=Maximum standard deviation of the variables in related previous study in the area (=9), and Confidence level of 95%. A total of 100 participants were enrolled who were designated into two equal groups of 50 subjects each (Group A and Group B). Group-A (Cases) was inclusive of breast carcinoma subjects included after clinical diagnosis with cytopathology confirmation of breast carcinoma. Group-A subjects were included irrespective of tumour stage or grade, tumour type or metastasis; who were later sorted as per type of carcinoma and stage of cancer. It comprised of 40, 42, 12 and 6 percent subjects of stage I, II, III and IV respectively (As per TNM staging-grading criteria). Among these, 88 % were of intra-ductal type, while 12 % were intra-lobular type. Group-B (Controls) included 50 healthy females who were age-matched with Group-A subjects. Control subjects enrolled in the study were the apparently healthy women who belonged to similar geographic area and/or who accompanied with the patients. Controls only with no family H/O breast carcinoma were chosen.

All the study participants ranged between 25-75 years of age, who were divided and matched for age in three sub-groups (viz. 25-40, 41-55 and 56-75 years). All the participants were ruled out for nulliparity to avoid bias arising out of influence of parity upon the study outcome. Only women with multiparity or those who had at least one issue were enrolled in the study. Overall mean age of Group-A and Group-B had no significant difference. Subjects with BMI (Body Mass Index) within 18 to 25 Kg/m^2 were enrolled. The detailed demographic profile of participants was as per Table 1.

Table 1: Demographic profile of study participants.

Demographic parameter	Group- A (N=50)	Group-B (N=50)	P value summary
Mean age (Year)	44.2±10.3	44.7±10.1	0.401
Height (Meter)	1.59±0.04	1.61±0.05	0.151
Weight (Kg)	58.52±3.68	59.16±3.41	0.369
Body mass index (Kg/m^2)	23.03±1.43	22.77±1.59	0.397

(P <0.05- Significant difference); (P >0.05- Not significant difference), Statistical comparison- Student's Unpaired 't' test (Two tailed with 95% Confidence Interval).

For subsequent comparisons, both principal groups (A and B) were divided as:

- Age-wise groups: (viz. 25 to 40 years (corresponds with pre-menopausal age); 41 to 55 years (corresponds with peri-menopausal age) and 56 to 75 years (corresponds with post-menopausal age)
- Menopausal status-wise groups: (viz. Pre-menopausal and Post-menopausal)- Based on actual cessation of menstruation.

For individual parameters of serum lipids and lipoprotein cholesterols, comparisons were carried out in two types as:

- Inter-group comparisons (cases versus controls)
- Intra-group comparisons (within cases only; or within controls only)

Comparisons were performed independently for all age- or menopausal status-wise sub-groups for each serum

parameter in both intra- and inter-group types (Table 2 to 8).

Exclusion criteria

Subjects with kidney or liver impairment, infectious and inflammatory diseases, diabetes, prior known cardiovascular, cerebrovascular or stroke episodes; and those on medications like steroids, hypolipidemics, oral contraceptive pills, or thyroxin, etc. were excluded from the study as any of these factors may affect serum lipid and lipoprotein concentrations. Subjects with history of known risk factors for carcinoma of breast like nulliparity, obesity (Body Mass Index >25) and history of smoking/alcoholism were excluded from the study to avoid bias interference. For Group-A (Cases) particularly, subjects with benign breast lesion or with known tumour anywhere else in body; those who have received any of surgery, hormones, radiotherapy or chemotherapy mode of treatment for breast cancer were excluded from the study. Out of total 209 cases of breast cancer under consideration, 159 cases were excluded from enrolment owing to their disqualification as per exclusion criteria in the study.

Blood specimen collection

With all aseptic measures, 5 ml of fasting blood was collected from median cubital vein of every subject. Serum separation was encouraged by allowing these specimens to stand for 15 min before analysis. Centrifuged, non-haemolysed sera were instantaneously analysed for serum total cholesterol, HDL-c and TG levels.

Laboratory analysis of serum lipid and lipoprotein cholesterol levels

Serum cholesterol estimation was carried out using cholesterol oxidase and peroxidase end point enzymatic method,[10] while serum HDL-c estimation was done with Precipitation method- end point.[11] For estimation of serum triglycerides, glycerol phosphate oxidase and peroxidise, end point enzymatic method was used.[12] VLDL-c and LDL-c were calculated by indirect method with Friedewald equation.[13]

All estimations were carried out with XL-300 fully automated random access clinical chemistry analyzer (Transasia BioMedicals Ltd, Erba Diagnostics Mannheim GmbH, Germany) and Erba Chem 5 Plus semi-automated clinical chemistry analyzer (Transasia BioMedicals Ltd, Erba Diagnostics Mannheim GmbH, Germany).

Statistical analysis

Numerical data were subjected to multiple inter-group and intra-group comparisons. Statistical analyses and interpretations were made using Student's unpaired 't' test (for two group comparison) and one-way ANOVA (Analysis of Variance) (for three or more groups comparisons of variable sample size). For significant findings of ANOVA, analysis with Tukey HSD (Honestly Significant Difference) test was done for Post hoc comparisons. All the data have been expressed as Mean±SEM (Standard error of mean). The levels of significance were calculated for all inter- and intra-groups comparisons. Probability value 'P' above 0.05 was taken as a statistically non-significant difference; while, P value below 0.05 as significant; and P value below 0.001 as highly significant difference. All statistical analyses have been carried with a computer program, Graph Pad- Prism software for windows (Version 5.00- March 12, 2007. Inc.; 1992-2007).

RESULTS

Significant findings of the study are as listed below

Serum TC and LDL-c levels were raised in breast cancer subjects than healthy controls. But serum HDL-c levels were lower among cases of breast cancer than healthy controls (Table 2).

Table 2: Serum lipoproteins and cholesterol- breast carcinoma cases versus healthy subjects (Overall Inter-group comparisons).

Serum analyte (mg/dL)	Cases (Group-A) (n=50)	Controls (Group-B) (n=50)	P value
TC	193.9±4.9 *	174.2±4.4	0.003*
LDL-c	128.1±5.3 *	107.3±4.6	0.004*
HDL-c	39.0±0.9 *	42.5±0.8	0.009*
TG	133.8±5.9	121.7±4.4	0.106
VLDL-c	26.7±1.1	24.3±0.8	0.106
*= P <0.05 (Statistically significant difference); ** = P <0.001 (Highly significant difference)			
TC= Total Cholesterol; LDL-c= Low Density Lipoprotein Cholesterol; HDL-c= High Density Lipoprotein Cholesterol; TG= Triglycerides; VLDL-c= Very low density lipoproteins cholesterol Statistical comparison- Student's unpaired 't' test (Two tailed with 95% confidence interval)			

Both pre- and post-menopausal cancer subjects had higher serum TC and LDL-c levels than respective control groups; while serum HDL-c levels were found decreased only among post-menopausal group of breast cancer subjects than post-menopausal control subjects (Table 3).

Post-menopausal breast cancer subjects exhibited elevated serum TC and LDL-c levels, but lowered HDL-c levels than pre-menopausal breast cancer subjects (Table 4). No menopausal status-wise significant alteration of any of these serum parameters could be noticed among control subjects (Table 5).

Table 3: Menopausal status-wise inter-group comparisons- (cases versus controls).

Serum analyte (mg/dL)	Pre-menopausal			Post-menopausal		
	Cases (n=34)	Controls (n=30)	P Value	Cases (n=16)	Controls (n=20)	P value
TC	186.2±5.0*	170.0±5.3	0.032*	210.4±10.2*	180.5±7.5	0.021*
LDL-c	118.9±5.0*	102.8±5.6	0.037*	147.9±11.4*	113.1±7.9	0.015*
HDL-c	40.5±1.0	43.4±1.1	0.076	35.7±1.8*	42.1±1.2	0.005*
TG	133.8±5.9	118.9±5.2	0.069	133.7±13.7	125.9±8.0	0.609
VLDL-c	26.7±1.1	23.7±1.0	0.069	26.7±2.7	25.1±1.6	0.609
*= P <0.05 (Statistically significant difference); ** = P <0.001 (Highly significant difference) Statistical comparison- student's unpaired 't' test (2- tailed with 95% CI- (Confidence interval)						

Table 4: Menopausal status-wise intra-group comparison among cases.

	Serum analyte (mg/dL)	Menopausal groups		
		Pre-menopausal (n=34)	Post-menopausal (n=16)	P-value
Cases (Group-A)	TC	186.2±5.06	210.4±10.29*	0.021*
	LDL-c	118.9±5.04	147.9±11.47*	0.009*
	HDL-c	40.56±1.03	35.75±1.86*	0.018*
	TG	133.8±5.99	133.7±13.71	0.991
	VLDL-c	26.76±1.19	26.74±2.74	0.991
*= P <0.05 (Statistically significant difference); ** = P <0.001 (Highly significant difference) Statistical comparison- Student's Unpaired 't' test (2- tailed with 95% CI)				

Table 5: Menopausal status-wise intra-group comparison among controls.

	Serum analyte (mg/dL)	Pre-menopausal (n=30)	Post-menopausal (n=20)	P-value summary
Controls (Group-B)	TC	170.0±5.3	180.5±7.5	0.250
	LDL-c	102.8±5.6	113.1±7.9	0.281
	HDL-c	43.4±1.1	42.1±1.2	0.486
	TG	118.9±5.2	125.9±8.0	0.455
	VLDL-c	23.7±1.0	25.1±1.6	0.455
P < 0.05 (Statistically significant difference); P < 0.001 (Highly significant difference) Statistical comparison- Student's Unpaired 't' test (2-tailed with 95% CI)				

The 41-55 years (peri-menopausal) age-group of breast cancer cases displayed higher serum TC and LDL-c levels than controls of the same age group. Serum HDL-c levels were surprisingly found decreased among both 41-55 (peri-menopausal) and 56-75 years (post-menopausal) age-group cases than respective healthy controls (Table 6).

No stage-wise significant difference in any of these serum analytes could be identified among breast carcinoma cases (Table 7).

Among cases of breast cancer, subjects aged between 41-55 years (peri-menopausal) had higher LDL-c levels than subjects of other age-groups. But for HDL-c, subjects aged between 56-75 years (post-menopausal) had lower levels than other age group cancer subjects (Table 8).

Neither breast carcinoma subjects nor healthy control subjects exhibited any significant aberration in serum TG and VLDL-c levels. No particular significant alteration in their levels could be identified with respect to age or menopausal status of subjects of any of the groups (Table 2 to 8).

DISCUSSION

Serum lipoproteins and lipids though seem to have trivial significance in diseases other than cardiovascular, cerebrovascular, endocrine, oxidative stress disorders; they hold imperative association with some malignancies. Cancer of breast is the best exemplar of this.

The risk of breast carcinoma is primarily dependent on the intensity and duration of mammary epithelial exposure to oestrogen.[14] Elevated cholesterol levels, being the source for oestrogen synthesis, could be responsible for the risk of carcinogenesis in oestrogen dependent cells of breast.

The overall significantly raised TC and LDL-c with lowered HDL-c in breast cancer subjects (Table 2) in present study reiterates the substantial alliance of

cholesterol, LDL-c and HDL-c, either individually or unanimously with breast cancer.

Table 6: Age-wise intra-group comparison among cases and controls.

Age group	Serum analyte (mg/dL)	Cases (n=19)	Controls (n=18)	*P* Value
25-40 years	TC	178.2±5.4	167.9±8.0	0.292
	LDL-c	109.1±4.9	102.7±8.3	0.513
	HDL-c	41.7±1.3	41.3±1.4	0.840
	TG	136.8±8.4	119.3±7.3	0.129
	VLDL-c	27.3±1.6	23.8±1.4	0.129
	Serum analyte (mg/dL)	Cases (n=25)	Controls (n=27)	
41-55 years	TC	202.5±7.0*	172.9±5.2	0.001*
	LDL-c	139.0±7.9**	103.9±5.5	0.000**
	HDL-c	38.7±1.3*	44.5±1.1	0.002*
	TG	123.6±8.4	122.1±6.1	0.892
	VLDL-c	24.7±1.6	24.4±1.2	0.892
	Serum analyte (mg/dL)	Cases (n=06)	Controls (n=05)	
56-75 years	TC	208.2±20.6	204.0±13.6	0.876
	LDL-c	143.3±19.7	138.8±14.7	0.864
	HDL-c	31.5±1.5*	39.6±1.2	0.002*
	TG	166.7±17.3	127.8±16.5	0.144
	VLDL-c	33.3±3.4	25.5±3.3	0.144

*= *P* <0.05 (Statistically significant difference); ** = *P* <0.001 (Highly significant difference). Statistical comparison- Student's Unpaired 't' test (2-tailed with 95% CI)

Among cases of breast cancer, subjects aged between 41-55 years (peri-menopausal) had higher serum LDL-c levels than cancer subjects of other age-groups (Table 8). No such explicit age-wise pattern was identified for serum TC in breast cancer subject; indicating equal propensity of cholesterol at all ages as a risk factor (Table 8).

Elevated serum TC and LDL-c levels in both pre- and post-menopausal cancer subjects than healthy subjects; with significantly higher level among post-menopausal cancer cases than pre-menopausal cases indicate definite role of TC and LDL in breast cancer specifically during peri-menopausal and post-menopausal ages (Table 3, Table 4).

As no such significant difference could be identified for TC and LDL-c levels (between pre-menopausal versus post-menopausal comparisons) among healthy controls (Table 5), above finding again strengthens the coalition of TC and LDL-c with breast cancer, amid peri-menopausal and post-menopausal proclivity.

The cyclopentanoperhydrophenanthrine (CPPP) nucleus of cholesterol contributes to the synthesis of oestrogen.[15,16] Oestrogen, being a steroid hormone can cross cell membrane; and can easily bind with specific receptors. Such hormone-receptor complexes further bind over specific sites on DNA (Hormone Responsive Elements) which enhances transcription of genes, likely to be expressed in the form of unregulated cellular proliferation.[17]

Table 7: Stage-wise comparison among cases.

Serum analyte (mg/dL)	Stage I (n=20)	Stage II (n=21)	Stage III (n=12)	Stage IV (n=3)	P Value
TC	195.2±6.84	192.7±9.04	206.8±12.80	168.3±8.64	0.497
LDL-c	127.6±7.20	127.6±9.66	141.0±16.42	109.7±5.77	0.710
HDL-c	40.60±1.35	38.43±1.74	35.83±2.28	39.00±3.05	0.483
TG	134.8±6.79	133.3±11.22	149.8±16.32	98.00±16.50	0.385
VLDL	26.96±1.35	26.67±2.24	29.97±3.26	19.60±3.30	0.385
(P <0.05- Significant difference); (P >0.05- Not significant difference); Analysis with one-way ANOVA and Post Hoc comparisons using Tukey HSD (honestly significant difference) test					

On the other hand, Buchwald H and Soma et al have demonstrated that when cholesterol supplementation is retarded, cellular growth gets blocked.[1,2] This way excess available cholesterol may potentiate the risk as increased uptake of cholesterol by cells has been a documented peril factor for mammary carcinogenesis.

The elevated serum LDL-c, as noticed by us, has been demonstrated to be more susceptible to oxidation, and thus it may cause high lipid peroxidation in mammary epithelial cells. This resultant oxidative stress poses to the risk of cellular and molecular damage, thereby resulting in cell proliferation and malignant conversions.[8]

Overall decrease in HDL-c level in breast cancer subjects than healthy subjects yet again incriminates association of HDL-c with breast cancer (Table 2). But for serum HDL-c, breast carcinoma subjects aged between 56-75 years (post-menopausal) had lower levels than other cancer subjects (Table 8).

Table 8: Age-wise intra-group comparison among cases and controls.

Cases			Controls	
Serum analyte	**Age group exhibiting significant alteration**	**P-value**	**Serum analyte**	**Age group exhibiting significant alteration**
TC	No specific age group within CASES exhibited significant alteration	0.064	TC	No specific age group within CONTROLS exhibited significant alteration
LDL-c	41-55 years	0.019*	LDL-c	None
HDL-c	56-75 years	0.004**	HDL-c	None
TG	None	--	TG	None
VLDL-c	None	--	VLDL-c	None
(*P <0.05- Significant difference); (**P <0.001- Highly significant difference); (P > 0.05- Not significant difference (NS); Analysis with one-way ANOVA and post Hoc comparisons using Tukey HSD (Honestly significant difference) Test				

Significant lowering of serum HDL-c was recorded in post-menopausal cancer subjects than pre-menopausal cancer subjects (Table 4); but lowered HDL-c was noted only during post-menopausal comparison between cases and controls (Table 3). These findings, if jointly considered, obvious role of menopausal status in serum HDL-c manipulation in breast cancer gains attention.

Oestrogen has role in HDL-c handling at cellular level. Oestrogen may activate the receptor gene for HDL via binding with 'Oestrogen Response Elements' and through 'Sterol Regulatory Element Binding Protein-1A'.[7] Though the post-menopausal phase is typified with low bio-available oestrogen levels, there is a chance that oestrogen pool may be maintained if androgens are switched into oestrogens after menopause. It can be better justified through the postulation put forth by Bernstein and Ross.[18] They have proposed that aromatisation of androgens to estrogens by adipose tissue (especially during post-menopausal phase of life) which contributes to the post-menopausal pool of bio-available oestrogen that might pose to the risk of breast cancer. Excess manipulation of HDL-c by carcinomatous cells under effect of such estrogens may have role in HDL-c lowering.

Absence of significant pre- or post-menopausal differences with respect to all lipid and lipoproteins in healthy women indicated no role of oestrogen dependent (mammary) cells in lipid handling when they are in non-carcinomatous state (Table 5). But, the finding of significant alterations in these parameters, only among breast cancer subjects, points to a switch in the pattern of lipid handling by cancer cells specifically when they undergo carcinomatous transformation. In that case too, specific patterns of aberration in levels of serum TC, LDL-c and HDL-c in particular age group (peri-menopausal and/or post-menopausal), beyond doubt, insinuates the role of these parameters in cancer risk enhancement during that particular phase of life of a woman.

Few previous studies in the same research area have demonstrated variable findings related with serum lipid and lipoprotein cholesterols in breast carcinoma subjects. Yet our findings of significantly higher levels of TC and LDL-c; and their menopausal status-wise distribution gain fair support from few previous studies.[5,8,19,20] The finding of lower serum HDL-c level in breast cancer subjects found in the present study is in line with results of earlier researches.[5,20,21]

Though, whether the aberrations in serum lipid and lipoprotein levels are cause or effect of the disease still remains unclear; besides the fact that multiple hypotheses have been set forth till date; our study brings up with it certain alarming proclamation that post-menopausal lower HDL-c and/or increased serum TC and LDL-c during peri- and post-menopausal age have significant alliance with breast cancer, but serum total cholesterol may act as a constant menace throughout.

CONCLUSION

In conclusion, the present study indicates an obvious coalition of altered serum lipoproteins and cholesterol levels with breast carcinoma. Elevated serum LDL-c and reduced HDL-c levels during the peri-menopausal and post-menopausal age can be a significant risk factor towards breast cancer development; with major impact of lowered serum HDL-c specifically during post-menopausal age of a woman. Cholesterol probably acts as a constant risk, may it be pre-, peri- or post-menopausal age of a woman. This study prompts the possibility of collective effect of altered serum total cholesterol, LDL-c and HDL-c levels during peri-menopausal and post-menopausal age towards development of carcinogenesis in oestrogen dependent breast tissue. Serum TG and VLDL-c levels do not bear any obvious significance in breast carcinoma.

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