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Profiling and Determinants of Impaired Lipid Profile Parameters among Breast Cancer Women of Childbearing Age Living in Douala, Cameroon

Idriss Ntatou Lemouchele[1], Suzi Pascale Mbougang[1], Esther Dina Bell[2,3], Loick Pradel Kojom Foko[4], Eric Fouelifack Nzeko[1], Cecile Okalla Ebongue[2,3], Jean Pierre Nda Mefo'o[2,3], Martin Luther Koanga Mogtomo[1*], Rosalie Anne Ngono Ngane[1]

[1]Department of Biochemistry, Faculty of Science, The University of Douala, Douala, Cameroon
[2]Depatment of Biological Sciences, Faculty of Medicine and Pharmaceutical Sciences, The University of Douala, Douala, Cameroon
[3]General Hospital Douala, Douala, Cameroon
[4]Depatment of Animal Organisms, Faculty of Science, The University of Douala, Douala, Cameroon
Email: idrissntatou@gmail.com, mbougangsuzy@gmail.com, esther.dinabell@gmail.com, kojomloick@gmail.com, ericnzeko@gmail.com, cecileokalla@yahoo.fr, drnda41@yahoo.fr, *koanga@yahoo.com, angono@yahoo.com

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Abstract

Objective: Breast cancer in women of childbearing age (WCBA) is a major public health concern. This study aimed to determine variation and determinants in lipid profile among Cameroonian WCBA diagnosed with breast cancer. **Materials and Methods**: A case-control study took place at two reference hospitals in Douala, Cameroon. A total of 176 WCBA (88 cases and 88 controls) were finally enrolled. Interviewer-administered questionnaires were used to collect sociodemographic, behavioural, clinical and anthropometric data. Three millilitres of venous blood were collected for analysis of total cholesterol (TC), high density lipoprotein cholesterol (HDL-c), low density lipoprotein cholesterol (LDL-c) and triglycerides (TG). **Results**: Overweight was predominantly seen in cases and controls. Serum levels of TC and LDL-c were significantly lower in cancer women (167.02 ± 45.46 vs 183.12 ± 27.38 mg/dL, $p = 0.005$ for TC; 85.83 ± 40.34 vs 105.25 ± 29.95 mg/dL, $p = 0.0004$ for LDL-c), while HDL-c levels were higher in controls (45.09 ± 7.20 vs 38.24 ± 11.14 mg/dL, $p < 0.0001$). Dyslipidaemia were mainly represented by hypo-HDL-cholesterolemia and hypertriglyceridemia. Lipid profiles were not modulated by clinical staging of cancer disease. The risk of impaired CT level was increased by more than seven times (AOR = 7.32, 95% CI 1.01 - 58.82) in alcohol drinkers and by ~seven times (AOR = 6.81, 95% CI 1.74 - 26.63) in women under contraception. Cancer women had 24 times more chances of

hypo-HDL-cholesterolemia compared to controls (AOR = 24.23, 95% CI 5.06 - 116.00). **Conclusion**: This study suggests the influence of breast cancer on lipid profile parameters especially HDL-c and LDL-c, and possibly their putative clinical utility for early diagnosis in premenopausal women in Cameroon.

Keywords

Breast Cancer, Women of Childbearing Age, Lipoprotein, Impairment, Determinants, Cameroon

1. Introduction

Breast cancer is defined as a malignant tumour that develops in mammary glands. Forming cancer cells can invade nearby tissues, destroy them and spread to other organs [1]. This disease is a serious public health problem in both developed and developing countries. Its incidence has increased over the past decade in most parts of the world including sub-Saharan African countries where an estimated annual incidence ~50,000 cases and ~750,000 deaths were reported in 2020 [2].

In Cameroon, the incidence of breast cancer was ~39,906 cases in 2020, with 4170 (34.1%) new female cases [1]. Previous works outlined that its burden has increased these last 10 years in the country, and was mainly due to concomitant lifestyle changes of Cameroonian women [3] [4] [5]. Breast cancer moved from 2nd to 1st place of women cancers, with worryingly high rates in women of childbearing age (WCBA) in whom prevalence increased from 31.9% in 2014 to 43.6% in 2020 [1] [6]. Risk factors of breast cancer are multiple and several studies showed a strong link between breast cancer and oxidative stress [7] [8], hormonal profile [9] [10] and lipid profile [11] [12] [13] [14] [15].

The link between breast cancer and lipid profile is still elusive and controversial. Several studies have suggested a possible link between obesity, weight gain, high lipid diet and increased risk of breast cancer [16] [17]. Other studies have pointed out that lipid profile parameters—*i.e.* total cholesterol (TC), high density lipoprotein cholesterol (HDL-c), low density lipoprotein cholesterol (LDL-c) and triglycerides (TG)—could be used as biomarkers of breast cancer pathophysiology [13] [18] [19]. For instance, Lídia and colleagues showed that 1) LDL-c > 117 mg/mL was predictive of tumour stage and was positively associated with a poorer breast cancer prognosis, and 2) LDL-c > 117 mg/mL was predictive of metastases [13].

There is lack of studies on relation between lipid profile and breast cancer in Cameroon, especially in WCBA. We, therefore, conducted the present study to determine variation and determinants in lipid profile among WCBA diagnosed with breast cancer attending referral hospitals in the town of Douala, Littoral Region, Cameroon.

2. Materials and Methods

2.1. Study Design

A case-control study was conducted between July 2020 and July 2021 (one year) at oncology departments of two reference hospitals in the town of Douala viz. Bonassama District Hospital (BHD) and Douala General Hospital (DGH). These hospitals were chosen because of their strategic location, quality of technical facilities and medical staff available. In addition, they receive cancer patients from all regions of Cameroon and other countries [20].

2.2. Selection of Cases and Controls

Cases consisted of Cameroonian women aged < 45 years, in premenopausal period (*i.e.*, women having seen their menses within the last six months), with confirmed anatomopathological diagnosis of breast cancer, receiving or not specific cancer treatment, and willing to participate. Controls were recruited among women attending hospitals or accompanying cases, aged ≤ 45 years, in premenopausal period, without early menopause, with or without family history of breast cancer, and having agreed to participate in the study. Women with cancers other than breast cancer and those who could not be sampled, even though they agreed to participate, were excluded from this study. A total of 176 women, 88 cases and 88 controls, were finally included.

2.3. Questionnaire

An investigator-administered structured questionnaire was administered to each participant during 10 to 15-minute individual interviews to collect sociodemographic, clinical, behavioural and anthropometric information. Medical records were scrutinized and body measurements were performed to collect complementary information (Table 1).

Table 1. List of variables included in the analysis.

Variable Categories	Type of variables	Collection source
Sociodemographic data	Age, level of education, marital status, type of employment, age at menarche	Interview
Clinical data	Family history related to breast cancer, location of breast cancer, clinical stage, type of treatment and presence or absence of metastases	Interview and medical file
Anthropometric data	Weight, height, body mass index (BMI)	Measured by investigators using a balance and scale
Behavioural data	Alcohol consumption, tobacco consumption, fruit/vegetable consumption, physical activity, breast-feeding, use of contraceptives, physical inactivity	Interview

2.4. Blood Collection and Transport

Three millilitres (3 mL) of venous blood were collected into dry tubes to determine lipid profile parameters (TC, HDL-c, LDL-c and TG). Blood samples were taken on an empty stomach, and an appointment of maximum three days was made with those who were not fasting until the date of collection. Prior to blood collection, all tubes were labelled with an investigator-defined barcode. After blood collection, the tubes are gently inverted to avoid haemolysis and formation of air bubbles. Blood samples were then transported in a cooler to the Clinical Biology Laboratory of Douala General Hospital for further biological analyses.

2.5. Laboratory Procedure

Venous blood was centrifuged at 3500 rpm for 5 minutes. The resulting supernatant (serum) was separated from pellet and used for spectrophotometry based determination of TC, HDL-c and TG. The concentration of LDL-c was deduced from those of other lipids using the Friedewald formula [21] [22].

2.6. Operational Definitions

A set of operational definitions were used in this study as presented in **Table 2**.

2.7. Statistical Analysis

Data were keyed, coded, verified for consistency and then analysed with StatView v5.0 (SAS Institute, Chicago, Inc., IL, USA) and GraphPad v5.03 (GraphPad PRISM, San Diego, Inc., CA, USA). Quantitative and qualitative variables were summarized as mean ± standard deviation (SD) and percentages, respectively. Data are presented as figures and tables where appropriate. Quantitative variables were compared using Student, Mann-Whitney and Kruskal-Wallis tests while Fisher's exact and Pearson chi square tests were used to compare qualitative data. Bivariate and multivariate logistic regression models were used to quantify the association between participants' information and lipid profile variation by calculating odds ratio (OR), confidence interval at 95% (95% CI) and statistical significance level. A p-value < 0.05 was considered statistically significant.

Table 2. Operational definitions used in the study.

Terms	Definitions	References
Dyslipidaemia	Increased levels in TG or decreased levels in LDL-c and HDL-c	[23] [24]
Obesity	BMI ≥ 30 kg/m^2	[25]
Hypo-HDL-cholesterolemia	HDL-c level ≤ 45 mg/dL	[26]
Hypertriglyceridemia	TG level ≥ 150 mg/dL	[27]
Hypercholesterolemia	TC level ≥ 200 mg/dL	[25]
Hyper-LDL-cholesterolemia	HDL-c level ≥ 130 mg/dL	[25]

2.8. Ethics and Dissemination

This study was approved by Institutional Ethics Committee of the University of Douala (CEI-UDo) (N˚2198CEI-UDo/02/2020/M). The women are recruited after the study protocol has received authorization from the Ethics Committee of the Douala General Hospital (N˚ 254 AR/MINSANTE/HGD/DM/07/2020) and Bonassama District Hospital (N˚ 259 AR/MINSANTE/HDB/DM/06/2020). The information and informed consent form was developed in accordance with the requirements of the Institutional Ethics Committee of the University of Douala and presented to all participants before their inclusion in the study. The patients were reassured on the strict respect of the confidentiality of the data collected and the possibility of withdrawing at any time without any reprisals in their care.

3. Results

3.1. Demographical, Anthropometric and Lipid Profile

Comparative analysis of demographical, anthropometric and lipid profile data between cases and controls is summarized in Table 3. BMI values were significantly higher in cancer women compared to controls (29.68 ± 6.33 kg/m^2 vs 27.73 ± 5.45 kg/m^2, $p = 0.03$). Serum level of TC and LDL-c were significantly lower in cancer women while HDL-c levels were higher in controls. TG levels were statistically similar between cases and controls ($p = 0.27$) (Table 3).

3.2. Lipid Profile Parameters by Clinical Staging of Breast Cancer

We noted a decrease in TC with severity of breast cancer disease, with mean values of 171.11 ± 20.98 mg/gL in patients at stage 1 to 162.19 ± 36.13 mg/gL in patients at stage 4. The mean values of TC were significantly lower in breast cancer women, irrespective of clinical stage, compared to their control counterparts (Table 4). Similar trend was observed for LDL-c. In contrast, HDL-c level

Table 3. Selected demographical, anthropometric and lipid profile in clinical groups.

Characteristics	Breast cancer ($n = 88$)	Controls ($n = 88$)	p-value
Age (yrs)	38.46 ± 5.35	36.49 ± 7.43	0.04*
Height (m)	1.64 ± 0.06	1.63 ± 0.06	0.38
Weight (Kg)	78.75 ± 18.73	73.55 ± 15.69	0.04*
BMI (Kg/m^2)	29.68 ± 6.33	27.73 ± 5.45	0.03*
TC (mg/gL)	167.02 ± 45.46	183.12 ± 27.38	0.005*
HDL-c (mg/gL)	45.09 ± 7.20	38.24 ± 11.14	<0.0001*
LDL-c (mg/gL)	85.83 ± 40.34	105.25 ± 29.95	0.0004*
TG (mg/gL)	190.83 ± 30.74	195.22 ± 21.95	0.27

Data are presented as mean ± standard deviation, BMI: Body mass index, TC: Total cholesterol, HDL-c: High-density lipoprotein cholesterol, LDL-c: Low-density lipoprotein cholesterol, TG: Triglycerides, Student t test was used to compare the two groups, *Statistically significant at $p < 0.05$.

Table 4. Variation in levels of TC, HDL-c, LDL-c and TG according to clinical stages.

Variables	Cases—Stage 1	Cases—Stage 2	Cases—Stage 3	Cases—Stage 4	Controls
TC (mg/gL)	171.11 ± 20.98^{ab}	166.07 ± 52.46^{a}	168.83 ± 45.41^{a}	162.19 ± 36.13^{a}	183.12 ± 27.38^{b}
HDL-c (mg/gL)	49.86 ± 11.57^{a}	44.57 ± 4.67^{a}	44.82 ± 8.75^{a}	45.70 ± 5.10^{a}	38.24 ± 11.14^{b}
LDL-c (mg/gL)	83.54 ± 22.62^{ab}	84.66 ± 44.47^{a}	89.68 ± 41.05^{a}	77.08 ± 33.64^{a}	105.25 ± 29.95^{b}
TG (mg/gL)	188.57 ± 19.47^{a}	191.03 ± 35.94^{a}	188.92 ± 31.23^{a}	197.11 ± 18.08^{a}	195.22 ± 21.95^{a}

TC: Total cholesterol, HDL-c: High-density lipoprotein cholesterol, LDL-c: Low-density lipoprotein cholesterol, TG: Triglycerides, Data are presented as mean ± standard deviation (SD), One-way ANOVA and Duncan's post-hoc tests were used to do pairwise comparisons between clinical groups, For the same line, values with the same letter are not statistically significant at $p < 0.05$.

was significantly higher in breast cancer women compared to controls regardless of clinical stage of cancer disease. No statistical differences in TC, HDL-c, LDL-c and TG were observed between different clinical stages (Table 4).

3.3. Prevalence of Dyslipidaemia

As presented in Figure 1, dyslipidaemia cases were found in cases and controls. Dyslipidaemia were dominated by hypo-HDL-cholesterolemia and hypertriglyceridemia. The proportion of hypo-HDL-cholesterolemia was significantly higher in controls compared to that in cases (97.7% vs 95.5%, $p < 0.0001$) (Figure 1(b)). Likewise, higher proportion of LDL- and TG-related dyslipidaemia was reported in controls but differences were not statistically significant (Figure 1(c) & Figure 1(d)).

3.4. Serum Lipid Profiles by Characteristics of the Participants

Variation in TC and HDL-c between cases and controls were stratified by different participants' characteristics and depicted in Table 5. No statistical difference was noted for TC levels either between cases and controls, within cases or within controls for different age groups. HDL-c values were significantly higher in cancer women compared to controls in age groups of 26 - 30 years (45.50 ± 6.36 vs 38.08 ± 7.40 mg/gL, $p = 0.02$), 31 - 35 years (46.67 ± 5.19 vs 37.79 ± 8.41 mg/gL, $p = 0.0015$) and 36 - 40 years (46.32 ± 7.09 vs 31.91 ± 17.29 mg/gL, $p = 0.009$). We also found that TC, HDL-c and LDL-c levels between cases and controls were modulated by BMI and level of physical activity. For instance, levels of TC and LDL-c were found to be significantly increased in overweight controls compared to overweight cases: 155.46 ± 55.79 vs 185.69 ± 29.50 mg/gL, $p = 0.02$ for TC, and 77.53 ± 49.60 vs 112.88 ± 34.88 mg/gL, $p = 0.003$ for LDL-c.

Consumption of fruits/vegetables has modulated mean levels of all lipid profile parameters in cases and controls, with higher levels of TC and LDL-c in controls. Alcohol beverage uptake seemed did not influence levels of TC, LDL-c and TG in clinical groups. In contrast, data suggested the influence of alcohol beverage uptake on HLD-c levels as alcohol drinkers diagnosed with cancer had higher values of HDL-c compared to their cancer-free counterparts in control

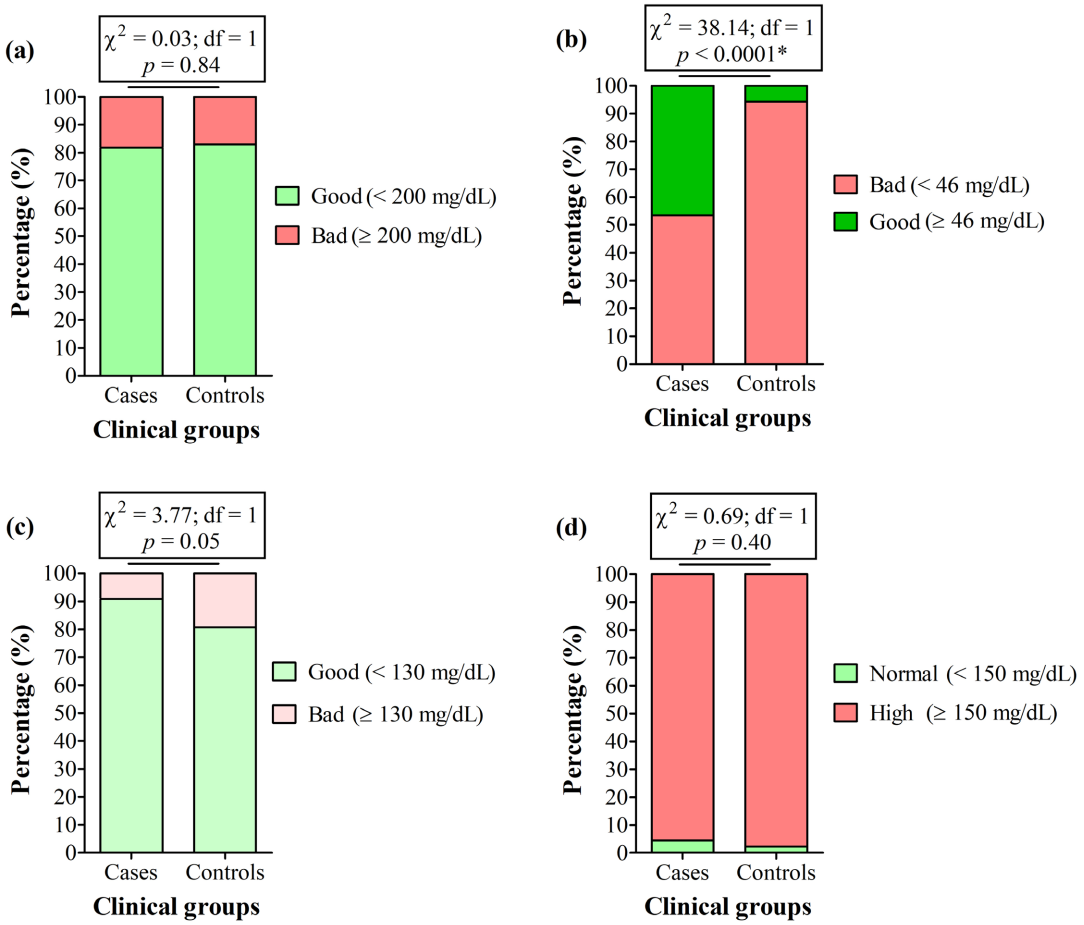

Figure 1. Prevalence of dyslipidaemia between cases and controls based on TC (a), HDL-c (b), LDL-c (c) and TG (d). TC: Total cholesterol, HDL-c: High-density lipoprotein cholesterol, LDL-c: Low-density lipoprotein cholesterol, TG: Triglycerides, df: Degree of freedom. Each bar represents proportion of dyslipidaemia for lipid profile parameters, Pearson's chi square (χ^2) test was used to compare proportion between cases and controls, *Statistically significant at $p < 0.05$.

Table 5. Variation of lipid profile parameters by characteristics of participants.

	TC (mg/gL)					HDL-c (mg/gL)				
	Breast cancer		Controls			Breast cancer		Controls		
Variables	*n*	Mean ± SD	*n*	Mean ± SD	*p*-value[$]	*n*	Mean ± SD	*n*	Mean ± SD	*p*-value[$]
Age (years)										
[20 - 25]	2	189.84 ± 0.00	5	188.52 ± 45.56	0.97	2	36.78 ± 0.00	5	43.74 ± 9.53	0.37
[26 - 30]	7	195.55 ± 35.04	18	180.60 ± 15.37	0.14	7	45.50 ± 6.36	18	38.08 ± 7.40	0.02*
[31 - 35]	15	166.43 ± 37.96	16	180.00 ± 27.83	0.26	15	46.67 ± 5.19	16	37.79 ± 8.41	0.0015*
[36 - 40]	28	157.30 ± 54.94	9	195.98 ± 30.56	0.05	28	46.32 ± 7.09	9	31.91 ± 17.26	0.009*
[41 - 45]	36	168.00 ± 41.87	40	181.92 ± 28.57	0.09	36	43.87 ± 8.04	40	39.23 ± 11.88	0.05
p-value[#]		0.38		0.56			0.16		0.38	
BMI (Kg/m²)										
Underweight	3	178.84 ± 30.08	0	-	-	3	47.13 ± 5.80	0	-	-

Continued

Normal	15	198.90 ± 23.36	33	184.35 ± 31.42	0.11	15	47.50 ± 5.11	33	38.56 ± 8.04	0.003*
Overweight	29	155.46 ± 55.79	28	185.69 ± 29.50	0.01*	29	44.30 ± 5.66	28	34.09 ± 12.87	0.003*
Obesity	25	160.54 ± 48.42	23	179.35 ± 20.38	0.09	25	46.29 ± 9.19	23	43.81 ± 10.25	0.38
Morbid obesity	16	165.91 ± 20.60	4	176.58 ± 9.06	0.33	16	42.03 ± 7.55	4	32.66 ± 14.40	0.08
p-value[#]		0.01*		0.88			0.21		0.19	
Fruits/Vegetables uptake										
Rarely	20	172.86 ± 54.38	20	170.92 ± 17.16	0.88	20	43.71 ± 8.02	20	40.84 ± 10.42	0.33
Occasionally	19	166.55 ± 53.88	36	192.85 ± 36.23	0.03*	19	45.37 ± 7.00	36	37.47 ± 10.71	0.005*
Always	49	164.81 ± 38.32	32	179.79 ± 14.95	0.03*	49	45.55 ± 7.01	32	37.48 ± 12.10	0.0003*
p-value[#]		0.61		0.03*			0.56		0.21	
Alcohol drinkers										
No	22	159.22 ± 37.33	23	177.84 ± 17.19	0.03*	22	80.24 ± 8.96	23	40.14 ± 15.35	0.13
Yes	66	169.61 ± 47.84	65	184.98 ± 30.06	0.02*	66	43.28 ± 6.58	65	37.57 ± 9.27	0.0001*
p-value[#]		0.24		0.38			0.22		0.11	
Physical activity										
Low	18	162.45 ± 41.39	27	183.12 ± 27.54	0.05	18	44.53 ± 4.20	27	34.60 ± 11.58	0.0012*
Moderate	25	176.41 ± 37.63	26	180.78 ± 23.84	0.62	25	44.47 ± 8.22	26	39.87 ± 11.17	0.11
Intense	45	163.63 ± 50.82	35	184.84 ± 30.21	0.03*	45	45.66 ± 7.63	35	39.85 ± 10.39	0.005*
p-value[#]		0.74		0.99			0.89		0.39	

	LDL-c (mg/gL)					TG (mg/gL)				
	Breast cancer		Controls			Breast cancer		Controls		
Variables	***n***	**Mean ± SD**	***n***	**Mean ± SD**	***p*-value[§]**	***n***	**Mean ± SD**	***n***	**Mean ± SD**	***p*-value[§]**
Age (years)										
[20 - 25]	2	115.67 ± 0.00	5	92.49 ± 10.80	0.03*	2	186.94 ± 0.00	5	174.70 ± 35.60	0.67
[26 - 30]	7	111.54 ± 33.56	18	103.70 ± 17.17	0.44	7	199.88 ± 12.99	18	194.10 ± 19.64	0.48
[31 - 35]	15	79.46 ± 36.71	16	103.29 ± 31.03	0.06	15	201.54 ± 16.84	16	194.59 ± 16.04	0.24
[36 - 40]	28	79.48 ± 44.59	9	124.61 ± 34.40	0.008*	28	180.06 ± 42.00	9	197.28 ± 18.35	0.24
[41 - 45]	36	86.77 ± 39.30	40	103.98 ± 33.70	0.04*	36	193.21 ± 26.10	40	198.08 ± 23.34	0.39
p-value[#]		0.17		0.31			0.19		0.28	
BMI (Kg/m²)										
Underweight	3	90.62 ± 32.02	0	-	-	3	205.44 ± 15.43	0	-	-
Normal	15	105.68 ± 36.35	33	105.67 ± 29.90	0.99	15	199.04 ± 12.26	33	192.75 ± 23.22	0.33
Overweight	29	77.53 ± 49.60	28	112.88 ± 34.88	0.003*	29	190.66 ± 37.42	28	193.59 ± 12.00	0.69
Obesity	25	82.41 ± 38.99	23	96.06 ± 22.45	0.14	25	187.51 ± 28.25	23	197.37 ± 27.66	0.22
Morbid obesity	16	86.70 ± 22.09	4	101.22 ± 24.86	0.86	16	185.92 ± 35.57	4	214.69 ± 26.16	0.14
p-value[#]		0.18		0.38			0.31		0.22	
Fruits/Vegetables uptake										
Rarely	20	93.19 ± 47.88	20	92.62 ± 20.18	0.96	20	194.93 ± 14.79	20	195.55 ± 23.51	0.92

Continued

Occasionally	19	86.02 ± 44.48	36	115.38 ± 36.45	0.01*	19	199.75 ± 12.94	36	187.96 ± 18.90	0.01*
Always	49	82.75 ± 35.59	32	101.76 ± 23.10	0.009*	49	185.71 ± 38.71	32	203.19 ± 21.99	0.02*
p-value#		0.75		0.06			0.50		0.005*	
Alcohol drinkers										
No	22	77.62 ± 32.54	23	97.98 ± 27.79	0.02*	22	189.58 ± 32.01	23	198.93 ± 13.41	0.21
Yes	66	88.57 ± 42.48	65	107.83 ± 30.47	0.003*	66	191.25 ± 30.55	65	193.91 ± 24.22	0.58
p-value#		0.41		0.22			0.61		0.11	
Physical activity										
Low	18	86.80 ± 35.30	27	110.80 ± 32.23	0.02*	18	187.44 ± 33.88	27	188.84 ± 19.68	0.86
Moderate	25	90.27 ± 43.26	26	102.27 ± 28.87	0.24	25	190.56 ± 29.89	26	193.22 ± 24.71	0.73
Intense	45	82.98 ± 41.17	35	103.19 ± 29.23	0.01*	45	192.34 ± 30.51	35	201.63 ± 20.25	0.12
p-value#		0.95		0.77			0.73		0.06	

Data are shown as mean ± standard deviation (SD), BMI: Body mass index, TC: Total cholesterol, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, TG: Triglycerides, §Student t test and Mann-Whitney test were used to compare the two clinical groups, #Kruskal-Wallis test was used to compare the two groups, *Statistically significant at *p*-value < 0.05.

group (43.28 ± 6.58 vs 37.57 ± 9.27 mg/gL, $p = 0.0001$). TG values were not modified by level of physical activity either between clinical groups or within clinical groups (*i.e.* cases and controls).

3.5. Variation of Lipid Profile by Type of Anti-Cancer Treatment

Mean serum levels of TC and HDL-c were significantly modulated by anticancer therapy. Lowest TC levels were seen in patients under radiotherapy (133.27 ± 39.25 mg/dL) and radiochemotherapy (142.05 ± 55.29 mg/dL) (**Figure 2(a)**). The same pattern was observed for HDL-c with values of 40.06 ± 12.77 mg/dL for patients under radiotherapy and 41.52 ± 5.82 mg/dL for those under radiochemotherapy (**Figure 2(b)**). Levels of LDL-c and TG were more decreased in patients under radiotherapy, but no statistically significant difference was found between groups (**Figure 2(c)** & **Figure 2(d)**).

3.6. Association of Breast Cancer Risk Factors with Lipid Profile

Alcohol uptake and contraceptive method usage were associated with an increased risk of TC ≥ 200 mg/dL based on univariate logistic regression (**Table 6**). The risk of HDL-c ≤ 46 mg/dL was increased by ~15 times in cases when compared to controls (COR = 14.81, 95% CI 5.35 – 39.17, $p = 0.0001$). In contrast, the risk of HDL-c ≤ 46 mg/dL was reduced by 70% in women having had their menarche at late age (COR = 0.30, 95% CI 0.14 – 0.61, $p = 0.001$), by 62% in those with intense level of physical activity (COR = 0.38, 95% CI 0.15 - 0.97, $p = 0.04$), and by 63% in those consuming fruits/vegetables (COR = 0.37, 95% CI 0.16 - 0.87, $p = 0.02$). The odds of LDL-c ≥ 110 mg/dL was increased by nearly

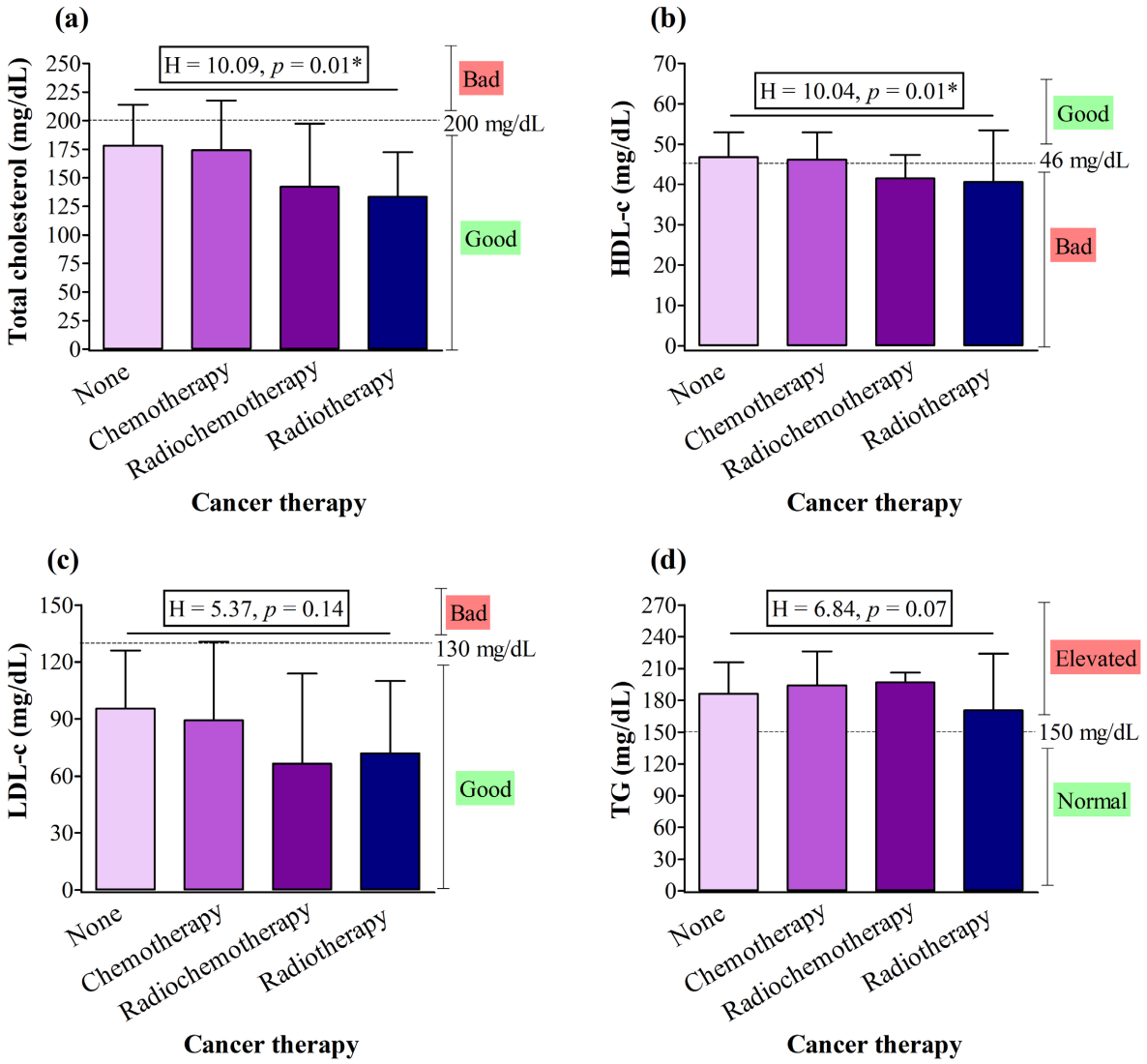

Figure 2. Variation in the mean concentration of lipid profile parameters depending on type of treatment (a) Total cholesterol, (b) HDL-c, (c) LDL-c and (d) TG.

five times in patients with BMI ≥ 25 Kg/m^2 (COR = 4.61, 95% CI 1.39 - 15.21, p = 0.01), and by more than four times in patients under contraception (COR = 4.02, 95%CI 1.33 - 12.13, p = 0.01). Conversely, the risk of LDL-c ≥ 110 mg/dL was decreased by 72% in those consuming fruits/vegetables (COR = 0.28, 95% CI 0.10 - 0.74, p = 0.01). No determinants were identified for TG levels (Table 6).

Based on multivariate logistic analysis, alcohol uptake and contraceptive method usage were associated with an increased risk of TC ≥ 200 mg/dL. Indeed, this risk was increased by more than seven times in those consuming alcoholic beverages (AOR = 7.32, 95% CI 1.01 - 58.82, p = 0.02), and by nearly seven times in those using contraceptive methods (AOR = 6.81, 95% CI 1.74 - 26.63, p = 0.005). The risk of HDL-c ≤ 46 mg/dL was 24 times increased in cases when compared to controls (COR = 24.23, 95% CI 5.06 - 116.00, p = 0.0001). No determinants were identified for LDL-c and TG (Table 7).

Table 6. Binary logistic regression for the relationship between anthropometric, behavioural and lipid profile parameters (n = 176).

Factors	Statistics	TC ≥ 200 mg/dL	HDL-c ≤ 46 mg/dL	LDL-c ≥ 130 mg/dL	TG ≥ 150 mg/dL
Clinical group: Case	*p*-value	0.84	**0.0001***	0.06	0.41
	COR (95%CI)	0.92 (0.43 - 2.01)	**14.81 (5.35 - 39.17)**	2.39 (0.97 - 5.88)	2.05 (0.37 - 11.48)
BMI ≥ 25 Kg/m²	*p*-value	0.84	0.21	**0.01***	0.98
	COR (95%CI)	0.58 (0.26 - 1.31)	0.60 (0.27 - 1.33)	**4.61 (1.39 - 15.21)**	0.65 (0.38 - 2.05)
Menarche ≥ 14 Years	*p*-value	0.84	**0.001***	0.87	0.21
	COR (95%CI)	1.15 (0.49 - 2.72)	**0.30 (0.14 - 0.61)**	1.08 (0.42 - 2.78)	0.35 (0.07 - 1.79)
Parity ≥ 4	*p*-value	0.84	0.29	0.71	0.37
	COR (95%CI)	1.45 (0.52 - 4.05)	0.63 (0.26 - 1.50)	1.26 (0.38 - 4.18)	0.45 (0.08 - .58)
Alcohol: Yes	*p*-value	**0.03***	0.09	0.24	0.61
	COR (95%CI)	**3.81 (1.10 - 13.20)**	1.86 (0.89 - 3.87)	1.96 (0.63 - 6.05)	0.57 (0.07 - 5.04)
Tobacco: Yes	*p*-value	0.74	0.97	0.56	0.97
	COR (95%CI)	1.54 (0.16 - 15.37)	1.03 (0.56 - 14.30)	2.01 (0.20 - 20.17)	1.98 (0.36 - 15.11)
Abortion ≥ 2	*p*-value	0.88	0.78	0.61	0.97
	COR (95%CI)	0.89 (0.19 - 4.30)	0.84 (0.24 - 2.90)	0.58 (0.07 - 4.78)	0.84 (0.33 - 3.89)
Physical activity: Intense	*p*-value	0.86	**0.04***	0.11	0.55
	COR (95%CI)	0.92 (0.37 - 2.32)	**0.38 (0.15 - 0.97)**	0.44 (0.17 - 1.19)	1.81 (0.25 - 13.34)
Contraception: Yes	*p*-value	**0.01***	0.17	**0.01***	0.98
	COR (95%CI)	**3.94 (1.37 - 11.35)**	2.87 (0.63 - 13.07)	**4.02 (1.33 - 12.13)**	2.44 (0.56 - 11.01)
Fruits & vegetables: Always	*p*-value	**0.01***	**0.02***	**0.01***	0.36
	COR (95%CI)	**0.34 (0.14 - 0.83)**	**0.37 (0.16 - 0.87)**	**0.28 (0.10 - 0.74)**	0.36 (0.04 - 3.28)

COR: Crude odds ratio, 95% CI: Confidence interval at 95%, BMI: Body mass index, TC: Total cholesterol, HDL: High density lipoprotein, LDL: Low density lipoprotein, TG: Triglycerides, *Statistically significant at *p*-value < 0.05.

Table 7. Multivariate logistic regression for the relationship between anthropometric, behavioural and lipid profile parameters (n = 176).

Factors	Statistics	TC ≥ 200 mg/dL	HDL-c ≤ 46 mg/dL	LDL-c ≥ 130 mg/dL	TG ≥ 150 mg/dL
Clinical Group: Case	*p*-value	0.42	**0.0001***	0.21	0.76
	AOR (95%CI)	0.64 (0.21 - 1.91)	**24.23 (5.06 - 116.00)**	2.42 (0.62 - 9.41)	0.73 (0.09 - 5.80)
BMI ≥ 25 Kg/m²	*p*-value	0.52	0.69	0.76	0.65
	AOR (95%CI)	0.70 (0.23 - 2.10)	1.26 (0.40 - 3.92)	0.81 (0.21 - 3.13)	0.59 (0.06 - 5.95)
Menarche ≥ 14 Years	*p*-value	0.22	0.38	0.08	0.57
	AOR (95%CI)	1.95 (0.66 - 5.74)	0.66 (0.26 - 1.68)	3.33 (0.85 - 13.01)	0.56 (0.07 - 4.29)
Parity ≥ 4	*p*-value	0.81	0.85	0.99	0.83
	AOR (95%CI)	1.15 (0.35 - 3.84)	1.11 (0.36 - 3.41)	1.00 (0.24 - 4.27)	0.78 (0.08 - 7.94)
Alcohol: Yes	*p*-value	**0.02***	0.29	0.12	0.68
	AOR (95%CI)	**7.32 (1.01 - 58.82)**	1.76 (0.61 - 5.06)	5.42 (0.62 - 47.58)	0.61 (0.06 - 6.44)

Continued

Tobacco: Yes	*p*-value	0.74	0.97	0.78	0.99
	AOR (95%CI)	1.59 (0.10 - 24.88)	1.13 (0.46 - 11.56)	1.51 (0.08 - 27.90)	-
Abortion ≥ 2	*p*-value	0.84	0.87	0.56	0.98
	AOR (95%CI)	1.19 (0.21 - 6.70)	0.88 (0.18 - 4.26)	0.49 (0.05 - 5.40)	-
Physical activity: Intense	*p*-value	0.82	0.26	0.27	0.55
	AOR (95%CI)	0.88 (0.30 - 2.59)	0.53 (0.17 - 1.62)	0.40 (0.08 - 2.06)	1.91 (0.23 - 16.03)
Contraception: Yes	*p*-value	**0.005***	0.64	0.88	0.98
	AOR (95%CI)	**6.81 (1.74 - 26.63)**	0.62 (0.08 - 4.75)	1.10 (0.28 - 4.29)	-
Fruits & vegetables: Always	*p*-value	0.07	0.34	0.07	0.51
	AOR (95%CI)	0.32 (0.09 - 1.10)	0.58 (0.18 - 1.81)	0.27 (0.06 - 1.14)	0.43 (0.04 - 4.96)

AOR: Adjusted odds ratio, 95% CI: Confidence interval at 95%, BMI: Body mass index, TC: Total cholesterol, HDL: High density lipoprotein, LDL: Low density lipoprotein, TG: Triglycerides, *Statistically significant at *p*-value < 0.05.

4. Discussion

Breast cancer is a public health problem in Cameroon especially in young WCBA. In this study, changes and determinants of lipid profile parameters were analysed among breast cancer WCBA attending two reference hospitals of Douala, Cameroon.

Serum HDL-c levels were lower in control women than those of breast cancer women. This finding is consistent with that from a study in UK [28], but contrasting with previous works from China, France, India and Morocco [14] [27] [29] [30]. Again, by stratifying cancer women with regard to clinical staging, HDL-c was significantly lower in controls when compared to different clinical stages. This result suggests that HDL-c, often considered beneficial, is actually involved in the pathophysiology of breast cancer as proposed earlier [31] [32] [33] [34]. Using gene-editing techniques, Christine and Philippe showed that by stopping activity of HDL receptors expressed on membrane of mammary cells the risk of developing breast cancer could be reduced while maintaining fundamental level of HDL in blood [31]. Besides, LDL-c levels were decreased in breast cancer WCBA, and this supports finding from a study conducted in China [29]. This is likely due to ability of cancer cells to absorb LDL-c into bloodstream for their growth. It was showed that cancer cells overexpress membrane LDL-c receptors as compared to normal cells [35] [36], and this leads to overuse of LDL-c in cancer women.

Main forms of dyslipidaemia were hypo-HDL-cholesterolemia and hypertriglyceridemia. These two types of dyslipidaemia are intrinsically related to two emerging public health concerns in Africa countries such as Cameroon (*i.e.* obesity and type 2 diabetes), and it would not be surprising to observe a sharp increase in burden of obesity and type 2 diabetes in Cameroonian WCBA in upcoming years. This reduced HDL-c level can be linked to increased obesity, decreased physical activity, malnutrition and pathologies such as cancer [37]. This

assumption is supported by some of our previous studies where we found that 49% of WCBA had low/moderate physical activity, 50% rarely or occasionally consumed fruits/vegetables and 60% were obese [20]. This result is in line with that reported in several Asian countries [37] [38] [39].

Women under cancer therapy had decreased TC and HDL-c levels, and this suggests a role of cancer therapy in appearance of dyslipidaemia as reported earlier with taxanes-containing chemotherapies [40] [41] [42]. Other studies showed that plasma lipid levels were temporarily affected in patients under cisplatin-based chemotherapy [43]. This decrease in TC and HDL-c levels would be attributed to chemical molecules used for chemotherapy which have the ability to inhibit the activity of lipoprotein lipase in adipose tissue [35]. In addition, cancer therapy, especially chemotherapy, is able to directly and/or indirectly cause formation of reactive oxygen species (O_2 and H_2O_2) [44]. To reduce this therapy-induced oxidative stress, liver upregulates the production of antioxidant molecules including HDL-c [35]; which will then be used by cancer cells for its proliferation.

The risk of hypo-HDL-cholesterolemia was reduced by 62% in women with intense level of physical activity. In fact, physical activity increases energy expenditure and improves activity of several tissues and organs such as brain. An improvement of mood and regulation of appetite is generally observed in physically active persons [45]. Again, physical activity increases consumption of cholesterol, utilization of fat as muscle fuel, and reduces collaterally the amount of fat depositing on artery walls or stored elsewhere in the body [45] [46].

The risk of hypo-HDL-cholesterolemia was reduced by 63% in those who were consuming regularly fruits/vegetables. Fruits do not contain cholesterol, and fresh/dried fruits and vegetables are protective factors against cardiovascular diseases. These are potential vitamin-rich antioxidants [47] [48]. Fruits and vegetables are able to lower blood cholesterol levels [49]. The bulk of scientific evidence on positive effects of fruits and vegetables is mainly supported by epidemiological studies in which fruits/vegetables can prevent metabolic alterations, reduce oxidative stress and appearance of breast cancer [20] [50] [51]. However, there is lack of knowledge on the nature of responsible bio-constituents and their mechanisms of action [47] [48].

Consumption of alcoholic beverages was associated with risk of increased TC levels. This result is not surprising as it is well documented that chronic alcohol consumption is associated with diverse metabolic disturbances such as glucose regulation, lipid profile, uric acid and nutritional status. Again, alcohol abuse can lead to overweight, obesity and protein-caloric malnutrition which are cause of increased TC levels [52] [53]. Maintaining normal cholesterol levels requires a healthy and balanced diet. Obesity is a common risk for breast cancer and dyslipidaemia [54] [55] [56].

5. Conclusion

In this case-control study, changes and determinants of impaired lipid profile

parameters (TC, HDL-c, LDL-c and TG) were determined in Cameroonian WCBA diagnosed with breast cancer. In conclusion, TC and LDL-c levels were reduced in cancer women while HDL-c were increased, as compared to controls. Impairment in lipid profile parameters were found in both controls and cases, with dyslipidaemia mainly represented by hypo-HDL-cholesterolemia and hypertriglyceridemia. TC, HDL-c and LDL-c were modulated by breast cancer disease and patients' characteristics such as age, BMI, fruit/vegetable uptake, alcohol beverage uptake and physical activity. Lipid profile parameters were not modulated by clinical staging of breast cancer. Cancer therapy influenced TC and HDL-c levels but not LDL-c and TG. The risk of impaired TC level was increased in women consuming alcohol beverages and practising contraception while the risk of hypo-HDL-cholesterolemia was increased in cancer women. This study suggests that breast cancer disease along with some patients' characteristics greatly influence lipid profile parameters. Further studies would be helpful to better understand relation between breast cancer and lipid profile parameters in WCBA, and possibly their clinical utility as biomarkers.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflict of interests.

Authors' Contribution

Authors INL, EDB and MLKM conceived the idea and designed the study. Authors INL and SPM collected and keyed field data. EDB and COE supervised collection of data at the hospitals. Author LPKF coordinated data keying, created figures, performed statistical analyses and helped in interpretation of results. INL drafted the first version of the manuscript with the help of LPKF. Authors EDB, LPKF, COE, EFN, JPNM, MLKM and RANN revised the paper for important intellectual content. Authors EDB, MLKM and RANN supervised the work at all stages. All authors read and approved the final paper before submission.

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Abbreviations

BC: Breast Cancer;

BMI: Body mass index;

CEI-UDo: Institutional Ethics Committee of the University of Douala;

DNA: Dehydroxyadenine Dinucleotide;

HDL-c: High Density Lipoprotein;

LDL-c: Low Density Lipoprotein;

TC: Total Cholesterol;

TG: Triglyceride;

WCBA: Woman of childbearing age.