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Research Article

Tamoxifen-Induced Dyslipidemia in Hormone Receptor-Positive Breast Cancer Patients: A Cross-Sectional Study among Kurdish Population

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Abstract

Background: Each patient's risk-benefit profile must be carefully assessed because tamoxifen's anti-estrogenic properties may have an impact on lipid metabolism. ***Objectives:*** This research looks into how tamoxifen affects Kurdish women's lipid profiles. ***Methods***: This cross-sectional study was conducted at the Azadi Hematology Oncology Centre in Duhok City, Kurdistan Region, Iraq, on 165 females with estrogen- and/or progesterone-positive breast cancer receiving adjuvant hormonal therapy with tamoxifen for three months or longer. ***Results***: The mean age was 42.4 years; 83.6% were married and multiparous (77.6%), with a BMI of 29.1 kg/m². 56.1% of participants had normal lipid profiles, 32.4% had elevated levels, and 11.5% had low levels. Age showed minimal impact on lipid biomarkers, with very weak correlations. Obese patients had normal TC (68.6%), HDL-C (68.6%), LDL-C (44.8%), TC/HDL-C (73.1%), and LDL-C/HDL-C (88.1%) ratios, as well as elevated TG (65.7%) and VLDL-C (77.6%), with weak correlations. Patients treated with tamoxifen for ≥ 24 months showed higher proportions of normal and high lipid profiles, with minimal impact of treatment duration. ***Conclusions***: Abnormal HDL-C, TG, VLDL-C, and TC/HDL-C were more frequent in obese patients, suggesting BMI had more impact on lipid profile than age or therapy duration. Tamoxifen had limited effects on lipid profile, with minimal associations with age or treatment duration.

Keywords: Lipid biomarkers, Hormonal positive breast cancer, Tamoxifen.

اضطراب دهون الدم الناتج عن تاموكسيفين لدى مريضات سرطان الثدي الإيجابي لمستقبلات الهرمونات: دراسة مقطعية في المجتمع الكردي

الخلاصة

الخلفية: يجب تقييم ملف المخاطر والفوائد لكل مريضة بعناية لأن خصائص التاموكسيفين المضادة للإستروجين قد تؤثر على استقلاب الدهون. **الأهداف**: بحث كيفية تأثير التاموكسيفين على ملف الدهون لدى النساء الكرديات. **الطرائق**: أجريت دراسة مقطعية في مركز آزادي لأمراض الدم والأورام في مدينة دهوك، إقليم كردستان، العراق، على 165 امرأة مصابة بسرطان الثدي الإيجابي للإستروجين و/أو البروجسترون واللواتي يتلقين العلاج الهرموني المساعد بالتاموكسيفين لمدة ثلاثة أشهر أو أكثر. **النتائج**: كان متوسط العمر 42.4 سنة؛ 83.6% كن متزوجات ومتعددات الولادة (77.6%)، مع مؤشر كتلة جسم 29.1 كغ/م². 56.1% من المشاركات كان لديهن ملفات دهون طبيعية، و 32.4% لديهن مستويات مرتفعة، و 11.5% لديهن مستويات منخفضة. أظهر العمر تأثيراً ضئيلاً على المؤشرات الحيوية للدهون، مع ارتباطات ضعيفة جداً. المصابات بالسمنة كان لديهن معدلات طبيعية للكوليسترول الكلي (68.6%)، الكوليسترول عالي الكثافة (68.6%)، الكوليسترول منخفض الكثافة (44.8%)، نسب الكوليسترول الكلي/الكوليسترول عالي الكثافة (73.1%)، والكوليسترول منخفض الكثافة/الكوليسترول عالي الكثافة (88.1%)، بالإضافة إلى الدهون الثلاثية المرتفعة (65.7%) والكوليسترول منخفض الكثافة جداً (77.6%)، مع ارتباطات ضعيفة. اللواتي عولجن بالتاموكسيفين لمدة 24 ≤ شهراً أظهرن نسباً أعلى من ملفات الدهون الطبيعية والمرتفعة، مع تأثير ضئيل لمدة العلاج. **الاستنتاجات**: كانت المستويات غير الطبيعية للكوليسترول عالي الكثافة، الدهون الثلاثية، الكوليسترول منخفض الكثافة جداً، ونسبة الكوليسترول الكلي/الكوليسترول عالي الكثافة أكثر تكراراً في المصابات بالسمنة، مما يشير إلى أن مؤشر كتلة الجسم كان له تأثير أكبر على ملف الدهون من العمر أو مدة العلاج. كان للتاموكسيفين تأثيرات محدودة على ملف الدهون، مع ارتباطات ضئيلة بالعمر أو مدة العلاج.

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INTRODUCTION

The incidence of breast cancer (BC) is rapidly increasing worldwide [1]. About 25% of all female cancer cases are BC, making it the most prevalent cancer diagnosed in women in the majority of countries [2]. Women have a 12.9% lifetime risk of developing BC, and the disease's incidence is increasing by 0.5% every year [3]. Nearly two-thirds of female BC patients with estrogen (ER) and/or progesterone receptor (PR) expression will benefit from anti-estrogen therapies [4]. For non-metastatic BC, tamoxifen, a non-steroidal medication, is utilized in adjuvant hormonal therapy. Instead of changing the manufacturing of estrogen, this selective estrogen receptor modulator (SERM) stops estrogen from binding to the estrogen receptor [5]. By

binding directly to the estrogen receptor, tamoxifen inhibits receptor-mediated transcriptional activity but does not alter estrogen synthesis [6]. SERMs are called "modulators" of estrogenic receptors rather than "agonists" or "antagonists" because they act as both agonists and antagonists of estrogen receptors in various tissues [7]. Individual differences in the therapeutic response to tamoxifen may be due to genetic and/or environmental variables that alter the drug's concentration and the amounts of its active metabolites in plasma [8]. For instance, menopausal status, body mass index (BMI), circadian rhythm, use of other drugs, contemporaneous meal consumption, and non-adherence to treatment all significantly impacted the drug's and its active metabolites' plasma concentrations [9]. Interestingly, few studies have indicated that adjuvant tamoxifen has distinct impacts on cancer patients' liver and lipid profiles. Significant estrogenic activity from tamoxifen decreases cholesterol and may reduce myocardial infarction risk [10]. Therefore, in addition to its anti-estrogenic effects, tamoxifen may lower the risk of cardiovascular disease (CVD) and associated mortality by changing lipid metabolism. Triglycerides (TG), total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C) have all been found to drop after taking tamoxifen [11]. Nevertheless, there are no clinical guidelines for lowering TG or TC with tamoxifen [12]. The effects of tamoxifen on lipid profiles are still poorly understood and inconsistently reported. To our knowledge, no similar studies on changes in lipid profiles in BC patients on tamoxifen have been conducted in our region, Kurdistan. This study hypothesizes that tamoxifen therapy influences lipid metabolism in Kurdish women with non-metastatic BC. The aim is to evaluate changes in the lipid profile associated with tamoxifen use and to determine whether these changes vary according to age, BMI, and treatment duration.

METHODS

Study design and setting

This cross-sectional study was carried out at the Azadi Hematology Oncology Centre (AHOC) in Duhok, situated in the Kurdistan Region of Iraq. The research spanned from October 2024 to January 2025. It was conducted in accordance with the ethical standards of the institutional and/or national research committee and with the Declaration of Helsinki and its later amendments (2000). Prior to commencement, ethical clearance was obtained from the Local Research Ethics Committee of the Duhok Directorate General of Health (Approval ID: 25092024-8-16; issued on August 16, 2024).

Participant selection

A cross-sectional study design was adopted. From a total of 250 BC patients assessed, 165 women with hormone receptor-positive (ER and/or PR receptor-positive) non-metastatic BC were eligible and included. All participants were on adjuvant tamoxifen therapy at a standard dose of 20 mg daily, having received treatment for a minimum of three months prior to enrollment.

Inclusion and exclusion criteria

Eligible participants were women diagnosed with non-metastatic BC who had been taking tamoxifen for at least three months, regardless of menopausal status. Patients were excluded if they had recently undergone surgery or radiation therapy. Additional exclusion parameters included: Male patients (because BC in men is rare and differences in hormones and body composition [e.g., BMI] could affect lipid metabolism and confound results), current use of lipid-lowering agents, and a history of diabetes mellitus, thyroid dysfunction, liver or kidney disease, or hypertension treated with medications known to influence lipid metabolism (e.g., beta-blockers, thiazide diuretics). Individuals who were active smokers were also excluded from the study.

Sample collection and laboratory analysis

Following an overnight fast of 9–12 hours, 2 mL of venous blood was drawn from each participant in the semi-recumbent (Fowler's) position. Blood samples were collected into sterile serum-separating tubes (gold-top) using standard aseptic techniques and a tourniquet to enhance venous access. After allowing the blood to clot for 30 to 60 minutes, samples were centrifuged for 10 minutes to isolate serum. The serum was analyzed for lipid profile parameters using the Cobas c501 module (part of the Roche Cobas 6000 system, Germany). All analyses were conducted in accordance with the manufacturer's guidelines to ensure consistency and reliability.

Sampling procedure

This study enrolled all patients (according to inclusion criteria) who have taken tamoxifen for three months or more. No random sampling was applied; rather, eligible participants were recruited through a purposive sampling approach during the study period. The primary outcome measures included key lipid profile components: TC, TG, High-density Lipoprotein Cholesterol (HDL-C), and LDL-C. Additional lipid indices were calculated using standard formulas: VLDL (mg/dL) = TG/5. VLDL-C was calculated using the Friedewald equation, which is applicable when TG

levels are below 400 mg/dL. Atherogenic indices, including the TC/HDL-C and LDL-C/HDL-C ratios, were calculated as the following:

$$\text{TC/HDL-C Ratio} = \frac{\text{Total Cholesterol}}{\text{HDL-C}}$$

$$\text{LDL-C/ HDL-C} = \frac{\text{LDL-C}}{\text{HDL-C}}$$

Anthropometric Measurements

Lipid profile biomarkers were assessed as part of the primary outcome measures. BMI was also recorded as a secondary variable. It was calculated using the standard formula: BMI (kg/m²) = Weight (kg) / Height² (m²)

Classification of BMI followed the World Health Organization (WHO) guidelines [13]: Normal weight: 18.5–24.9 kg/m², overweight: 25–29.9 kg/m², and obese: ≥30 kg/m².

Statistical analysis

Each participant was assigned a unique identification number to ensure anonymity and facilitate data organization. All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS), version 25 (IBM Corp., Armonk, NY, USA). Descriptive statistics were utilized to summarize patient characteristics and clinical parameters. Continuous variables were presented as means, standard deviations (SD) for normally distributed data, and as medians with interquartile ranges (IQR) for non-normally distributed data. Categorical variables were expressed as frequencies and percentages. To examine associations between categorical variables, the Pearson Chi-square test was applied. In cases where expected cell counts were below the required threshold, Fisher's exact test was used as an alternative. To explore the strength and direction of associations between continuous or ordinal variables, Spearman's rank correlation coefficient was employed. A two-tailed p-value < 0.05 was considered statistically significant for all analyses.

RESULTS

Among the 250 women treated at AHOC, Duhok, 165 met the inclusion criteria. Their mean age was 42.38 ± 6.73 years. Most were married (83.6%), multiparous (77.6%), and unemployed (78.2%), and 32.1% had a family history of cancer. Mean BMI was 29.10 ± 5.03 kg/m². Surgical treatment comprised lumpectomy (67.3%) and mastectomy (32.7%). All patients were hormone-receptor positive; 44.2% were HER2-positive and 55.8% were HER2-negative. The median time since cancer diagnosis was 24 months (13–36 months). The median duration of tamoxifen use was 22 months (12–36 months). All these details are demonstrated in Table 1.

Table 1: The demographic characteristics of the study population

Variables			Values
Age (year)	Mean±SD		42.38±6.73
	< 40 Y		41(24.8)
	40-49 Y		92(55.8)
	50-59 Y		32(19.4)
Marital Status	Married		138(83.6)
	Unmarried		27(16.4)
Parity	Nulli		37(22.4)
	Multi		128(77.6)
Occupation	Employed		36(21.8)
	Unemployed		129(78.2)
Family History of Cancer	Yes		53(32.1)
	No		112(67.9)
BMI (kg/m²)	Mean±SD		29.10±5.03
	Normal: 18.5–24.9		37(22.4)
	Overweight: 25–29.9		61(37.0)
	Obese: ≥ 30		67(40.6)
Surgery Type	Lumpectomy		111(67.3)
	Mastectomy		54(32.7)
Histochemical tests	HER2	Positive	73(44.2)
		Negative	92(55.8)
Time from diagnosis (month)	Median (IQR)		24(13-36)
	6-11		24 (14.5)
	12-17		34(20.6)
	18-23		11(6.7)
	≥24		96(58.2)
Duration of tamoxifen treatment (month)	Median (IQR)		22(12-36)
	3-9		32(19.4)
	10-16		30(18.2)
	17-23		21(12.7)
	≥24		82(49.7)

The values were expressed as frequency, percentage, median with IQR, and mean±SD.

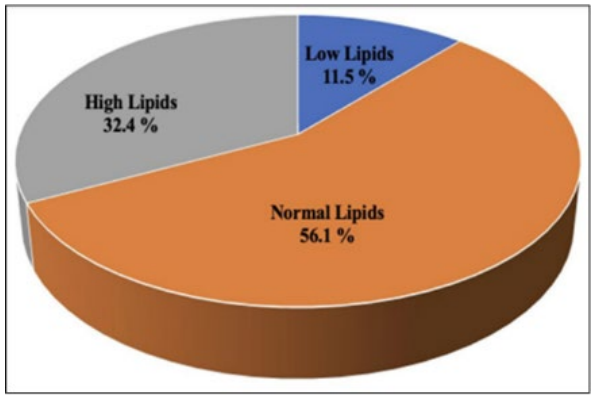

Figure 1: Prevalence of dyslipidemia among studied patients.

Lipid profile abnormalities were categorized into low, high, and normal values. Among participants, 11.5% exhibited low lipid levels, 32.4% had high lipid levels, and 56.1% maintained normal lipid profiles (Figure 1). Lipid values were interpreted based on the following reference ranges used in our clinical laboratory: TC (120–200 mg/dL), HDL-C (40–60 mg/dL), LDL-C (50–100 mg/dL), TG (35–150 mg/dL), VLDL-C (13–25 mg/dL), TC/HDL-C ratio (3.5–6.9), and LDL-C/HDL-C ratio (1.2–6.2). These ranges were applied to classify lipid profiles and guide the categorical analysis in this study. TC, HDL-C, and LDL-C were divided into three categories (low, normal, and high), while TG, VLDL-C, TC/HDL-C, and LDL-C/HDL-C were categorized into two categories due to fewer cases. This adjustment was made to ensure that each group had an adequate number of cases, allowing for meaningful statistical analysis despite the limited sample sizes in certain categories. Regarding the association between age groups and lipid categories, patients aged 40–49 years consistently showed the highest proportions across normal and abnormal levels of TC, HDL-C, LDL-C, TG, VLDL-C, and lipid ratios. None of these differences reached statistical significance, although TG (p= 0.053) and VLDL-C (p = 0.061) showed trends toward significance, but they are not statistically significant. All correlations were very weak (both positive and negative), indicating minimal age-related impact on lipid response to tamoxifen; all are shown in Table 2.

Table 2: Association of age groups with lipid biomarkers categories

Age groups (year)	Lipid markers								
	TC (mg/dL)			HDL-C (mg/dL)			LDL-C (mg/dL)		
	Low < 120	Normal 120-200	High > 200	Low < 40	Normal 40-60	High > 60	Low < 50	Normal 50-100	High > 100
< 40	1(2.4)	24(58.5)	16(39.1)	5(12.2)	27(65.8)	9(22)	3(7.3)	13(31.7)	25(61)
40-49	3(3.3)	60(65.2)	29(31.5)	18(19.6)	58(63)	16(17.4)	5(5.4)	39(42.4)	48(52.2)
50-59	3(9.4)	24(75)	5(15.6)	7(21.9)	21(65.6)	4(12.5)	4(12.4)	14(43.8)	14(43.8)
p-value		0.137			0.716			0.438	
Spearman's Correlation		rho = -0.181 p = 0.02			rho = -0.108 p = 0.166			rho = -0.117 p= 0.135	
	TG (mg/dL)			VLDL-C (mg/dL)		TC/ HDL-C		LDL-C/ HDL-C	
	Normal 35-150		High > 150	Normal 13-25	High > 25	Low < 3.5	Normal 3.5-6.9	Low < 1.2	Normal 1.2-6.2
< 40	24(58.5)		17(41.5)	19(46.3)	22(53.7)	16(39)	25(61)	6(14.6)	35(85.4)
40-49	35(38)		57(62)	25(27.2)	67(72.8)	37(40.2)	55(59.8)	8(8.7)	84(91.3)
50-59	11(34.4)		21(65.6)	8(25.0)	24(75)	12(37.5)	20(62.5)	5(15.6)	27(84.4)
p-value	0.053			0.061		0.962		0.423	
Spearman's Correlation	rho = 0.172 p = 0.027			rho = 0.163 p = 0.037		rho = 0.008 p = 0.919		rho = 0.002 p = 0.984	

Values are expressed as frequency and percentage. Chi-Square Test, Fisher's Exact Test, and Spearman's Correlation were performed for statistical analysis.

Regarding the association between BMI and lipid biomarkers, obese patients (BMI ≥ 30 kg/m²) had the highest proportions of normal TC (68.6%), HDL-C (68.6%), LDL-C (44.8%), and normal TC/HDL-C (73.1%) and LDL-C/HDL-C ratios (88.1%). They also had the highest proportions of elevated TG (65.7%) and VLDL-C (77.6%). Significant associations were observed for HDL-C (p = 0.001), TG (p = 0.001), VLDL-C (p = 0.001), and TC/HDL-C ratio (p = 0.005), though correlations were weak. Other markers, including TC, LDL-C, and LDL-C/HDL-C ratio, were not significantly associated with BMI; all data are presented in Table 3. Regarding the association between tamoxifen treatment duration and lipid biomarkers, patients treated for ≥ 24 months showed the highest proportions of normal and high TC (67.1% and 30.5%), normal and high HDL-C (69.5% and 13.4%), normal and high LDL-C (46.3% each), high TG (65.9%), high VLDL-C (78.0%), and normal and low TC/HDL-C (61.0% and 37.8%) and LDL-C/HDL-C ratios (12.2% and 87.8%). Among these, only VLDL-C showed a statistically significant association (p = 0.05); all other markers were non-significant, with very weak correlations, indicating minimal impact of treatment duration on lipid profile. These data are demonstrated in Table 4.

DISCUSSION

In this cross-sectional investigation, 165 Kurdish women with hormone receptor-positive BC had their lipid profiles evaluated in relation to tamoxifen.

Table 3: Association of BMI categories with the lipid biomarkers categories

BMI (kg/m^2)	Lipid markers								
	TC (mg/dL)			HDL-C (mg/dL)			LDL-C (mg/dL)		
	Low < 120	Normal 120-200	High > 200	Low < 40	Normal 40-60	High > 60	Low < 50	Normal 50-100	High > 100
18.5-24.9	1(2.7)	24(64.9)	12(32.4)	3(8.1)	20(54.1)	14(37.8)	1(2.7)	14(37.8)	22(59.5)
25-29.9	2(3.3)	38(62.3)	21(34.4)	10(16.4)	40(65.6)	11(18)	4(6.6)	22(36.1)	35(57.3)
≥ 30	4(6)	46(68.6)	17(25.4)	17(25.4)	46(68.6)	4(6)	7(10.4)	30(44.8)	30(44.8)
p-value		0.785			0.001			0.43	
Spearman's Correlation		rho= -0.088 *p*= 0.26			rho = -0.309** *p*< 0.0001			rho= -0.142 *p*= 0.07	
	TG (mg/dL)		VLDL-C (mg/dL)		TC/HDL-C		LDL-C/HDL-C		
	Normal 35-150	High > 150	Normal 13-25	High > 25	Low < 3.5	Normal 3.5-6.9	Low < 1.2	Normal 1.2-6.2	
18.5-24.9	26(70.3)	11(29.7)	22(59.5)	15(40.5)	22(59.5)	15(40.5)	3(8.1)	34(91.9)	
25-29.9	21(34.4)	40(65.6)	15(24.6)	46(75.4)	25(41)	36(59)	8(13.1)	53(86.9)	
≥ 30	23(34.3)	44(65.7)	15(22.44)	52(77.6)	18(26.9)	49(73.1)	8(11.9)	59(88.1)	
p-value	0.001		0.001		0.005		0.746		
Spearman's Correlation	rho= 0.235 *p*= 0.002		rho= 0.263 *p*= 0.001		rho= 0.251 *p*= 0.001		rho= -0.034 *p*= 0.663		

Values are expressed as frequency and percentage. Chi-Square Test, Fisher's Exact Test, and Spearman's Correlation were performed for statistical analysis.

Table 4: Association of tamoxifen treatment duration categories with the lipid biomarkers categories

Treatment duration (month)	Lipid markers								
	TC (mg/dL)			HDL-C (mg/dL)			LDL-C (mg/dL)		
	Low < 120	Normal 120-200	High > 200	Low < 40	Normal 40-60	High > 60	Low < 50	Normal 50-100	High > 100
3-9	1(3.1)	22(68.8)	9(28.1)	6(18.8)	19(59.4)	7(21.8)	1(3.1)	14(43.8)	17(53.1)
10-16	1(3.3)	18(60)	11(36.7)	6(20)	19(63.3)	5(16.7)	1(3.3)	9(30)	20(66.7)
17-23	3(14.3)	13(61.9)	5(23.8)	4(19)	11(52.4)	6(28.6)	4(19.0)	5(23.8)	12(57.2)
≥ 24	2(2.4)	55(67.1)	25(30.5)	14(17.1)	57(69.5)	11(13.4)	6(7.4)	38(46.3)	38(46.3)
p-value		0.451			0.722			0.146	
Spearman's Correlation		rho = 0.009 *p* = 0.905			rho = -0.04 *p* = 0.61			rho = -0.109 *p* = 0.165	
	TG (mg/dL)		VLDL-C (mg/dL)		TC/ HDL-C		LDL-C/ HDL-C		
	Normal 35-150	High > 150	Normal 13-25	High > 25	Low < 3.5	Normal 3.5-6.9	Low < 1.2	Normal 1.2-6.2	
3-9	16(50)	16(50)	15(46.9)	17(53.1)	13(40.6)	19(59.4)	3(9.4)	29(90.6)	
10-16	15(50)	15(50)	11(36.7)	19(63.3)	12(40)	18(60)	2(6.7)	28(93.3)	
17-23	11(52.4)	10(47.6)	8(38.1)	13(61.9)	9(42.9)	12(57.1)	4(19)	17(81)	
≥ 24	28(34.1)	54(65.9)	18(22)	64(78)	31(37.8)	50(61)	10(12.2)	72(87.8)	
p-value	0.203		0.05		0.99		0.591		
Spearman's Correlation	rho= 0.151 *p*= 0.053		rho= 0.213 *p*= 0.006		rho= 0.026 *p*= 0.737		rho= -0.045 *p*= 0.567		

Values are expressed as frequency and percentage. A Chi-Square Test, Fisher's Exact Test, and Spearman's Correlation were performed for statistical analysis.

Although the results of earlier studies on tamoxifen's impact on lipid metabolism have been conflicting, our findings provide fresh regional information on this developing subject. According to the analysis, older and obese patients had more noticeable lipid alterations, especially in TG, VLDL-C, and lipid ratios. For TG levels, statistical significance was only borderline, despite age group comparisons showing a tendency towards higher dyslipidemia rates in the 40–49 age range. These age-related results are not in tune with many previous studies; for example, Lin *et al*. (2014) reported that treatment with tamoxifen significantly decreased TC as age increased [14]. Interestingly, our investigation revealed lipid changes seemed to be more strongly correlated with BMI. TG, VLDL-C, and TC/HDL-C ratios were significantly greater in obese patients, but HDL-C levels were significantly lower as BMI rose. This is consistent with research that suggests tamoxifen may interact with hepatic lipid management, especially in patients who already have dyslipidemia, and supports the notion that body composition plays a significant role in lipid metabolism under tamoxifen medication. Almeida *et al*. (2005) [15]. Our findings contrast with previous reports of tamoxifen boosting HDL-C levels according to Ali *et al*. (2018) [4] results or showing no effect according to Patil *et al*. (2011) [16] results. Many cholesterol markers stayed within clinical reference ranges in spite of these modifications, which could indicate a more complex or patient-specific impact of tamoxifen on lipid metabolism. Two previous studies, Lin *et al*. (2014) [14] and Bourassa *et al*. (2016) [17], reported cardiovascular protection by improving lipid profiles, most notably by reducing TC and LDL-C, accompanied by the increase in HDL-C. Tamoxifen was linked to significant decreases in TC and LDL-C in a short-term study of postmenopausal women with BC that was done by Kusama *et al*. (2004) [18], although the observed increases in TG were not statistically significant. Similarly, a different study done by Gupta *et al*. (2006) [19] discovered that TC and LDL-C decreased

the most during the first three to six months of tamoxifen treatment, although TG levels did not significantly alter during the same time. In our findings, when evaluating different durations of tamoxifen medication, we did not find any statistically significant differences across TC, HDL-C, LDL-C, TG, TC/HDL-C ratio, or LDL-C/HDL-C ratio in our investigation, which included a broader treatment window (minimum 3 months to over 24 months). Overall, there were minimal associations between treatment duration and lipid levels, with the exception of VLDL-C and TG levels, which tended to increase with longer therapy, but not statistically significant. These findings are consistent with prior research done by Lin *et al*. (2014) [14] and Gupta *et al*. (2006) [19], which supported the idea that the most notable lipid changes, if any, occur early in tamoxifen treatment and eventually stabilize. The study of Ali *et al*. (2022) [20] has demonstrated a comparable rise in TG and VLDL-C; however, the elevated values were not statistically significant. While TG and VLDL-C levels were marginally elevated in the research done by Gupta *et al*. (2006) [19], they were not statistically significant. Contrary to our findings, the study of Lin *et al*. (2014) [14] showed that the TG levels did not change before or after tamoxifen treatment. Three months after starting the medication, tamoxifen raised the TG levels, according to Esteva and Hortobagyi (2006) [21]. A study by Cuzick *et al*. (2015) [22] has shown that tamoxifen may have adverse effects, such as a significantly increased risk of venous thrombosis, pulmonary embolism, and stroke. Furthermore, hypertriglyceridemia-induced acute pancreatitis has been associated with tamoxifen, as reported by Singh *et al*. (2016) [23]. Howard and Rossouw (2013) [24] explained the fact that tamoxifen is also a partial estrogen agonist, which is associated with an increased risk of endometrial cancer and thromboembolic events, may help to explain its possible adverse effects.

Study limitations

Nonetheless, this study has limitations, such as the absence of baseline (pre-treatment) lipid data, and a control group limits causal interpretation. Its cross-sectional design precludes assessment of changes over time, and unmeasured factors like diet, activity, and genetics may have influenced the results.

Conclusion

In high-risk women, tamoxifen, a hormonal therapy for hormone receptor-positive BC, has been shown to reduce recurrence risk. This study offers valuable regional insight into its effects on lipid profiles among Kurdish women. While tamoxifen did not significantly alter most lipid markers, BMI-related variations were notable, especially in obese patients with elevated TG, VLDL-C, and lipid ratios. Age and treatment duration had minimal associations, though TG and VLDL-C showed a non-significant upward trend with longer therapy. These findings underscore the importance of individualized lipid monitoring during tamoxifen treatment, particularly in patients with higher BMI. Health care providers are urged to regularly assess lipid levels and recommend lifestyle changes as needed. Further research with larger, longitudinal cohorts is essential to better understand tamoxifen's long-term impact on lipid metabolism.

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Conflict of interests

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Data sharing statement

Supplementary data can be shared with the corresponding author upon reasonable request.

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