Interactive role of breast cancer on dyslipidemia and hypertension metabolic risk according to treatment exposure and menopausal status

Research Article

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Abstract: **Background:** Breast cancer (BC) is the principal cause of cancer related deaths among women worldwide. The available evidence suggests that cardio-metabolic risk factors such as dyslipidemia and hypertension may contribute differently to breast cancer severity and pathogenesis. The aim of this study is to evaluate the interactive role of BC on dyslipidemia and HTN risk according to the type of treatment exposure and menopausal status. **Patients and methods:** Observational experimental design implemented; permit to include 134 newly-diagnosed patients who were naïve to any type of treatment interventions and 262 recently-diagnosed patients during their first three months of treatments' exposure including chemotherapy treatments. Patients with breast cancer were evaluated for dyslipidemia and hypertension biomarkers. **Results:** About 5.0% of breast cancer patients had dyslipidemia. The prevalence of increased triglycerides and total cholesterol were more frequent ($p < 0.05$) in recently-diagnosed group than in newly-diagnosed patients. While 23% of patients had overt hypertension, with higher ($p < 0.05$) prevalence in chemo group (28%), triglycerides was higher ($p < 0.05$) in postmenopausal than premenopausal BC patients (221.0 ± 5.9 vs. 195 ± 4.7 mg/dl). Similarly, the prevalence of abnormal systolic blood pressure (9% vs. 5%) and diastolic blood pressure (11% vs. 7%) was higher ($p < 0.05$) in postmenopausal patients. **Conclusions:** Dyslipidemia and hypertension biomarkers were prevalent among breast cancer patients and the risk increased in postmenopausal women and after treatments' exposure specially chemotherapy. This conclusion requires a closer attention by healthcare professionals in order to improve the outcomes after diagnosis and to enhance treatment exposure regarding postmenopausal women.

Keywords: Breast cancer • chemotherapy • hypertension • lipid profile

1. Introduction

Breast cancer (BC) is a disease characterized by the growth of malignant cells in the mammary glands. BC is the most frequent cancer in women, which is malignant in most cases, and is the leading cause of cancer related deaths among women worldwide. It accounts for about 28% of all cancers in Europe and United States.^[1] In Jordan, BC is the most frequent cancer type amongst female cancers, accounting for about 37% of all cancers and about 19% of all newly diagnosed cancer cases

followed by colorectal cancer. According to a published study in the United Kingdom, around 7% of female BC is related to modifiable lifestyle and environmental factors.

^[2] Although, many risk and prognostic factors of BC have been documented, an insight view of BC severity and prognosis is still not clarified.^[3] Evidences suggest that cardio-metabolic risk factors such as dyslipidemia and hypertension (HTN) may have different influences on BC severity and pathogenesis.^[4–6] Dyslipidemia is characterized by an elevation in total cholesterol concentration, low-density lipoprotein cholesterol

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(LDL-C), triglyceride (TG) concentrations and a reduction in high-density lipoprotein cholesterol (HDL-C). Mechanisms for such relationship between dyslipidemia and BC risk are unknown; however, previous studies proposed that abnormal lipid metabolism along with insulin resistance may be related to BC pathogenesis and BC subtypes.^[4] Moreover, BC tissues had faster TG turnover in comparison to normal tissue.^[6] Evidence of possible effect of dyslipidemia and HTN on BC risk is inconclusive and very limited.^[4,5,7]

In Jordan, dyslipidemia and HTN are significantly prevalent among Jordanian females and males.^[8–10] However, there are no studies on BC severity and HTN, nor on dyslipidemia risk factors. Furthermore, studies that evaluate the impact of treatment exposure on dyslipidemia and HTN are also lacking. Subsequently, the objective of the current study is to evaluate the interactive role of BC on Dyslipidemia and HTN risk among Jordanian women according to the type of treatment exposure and menopausal status.

2. Subjects and Methods

2.1. Study sample and design

In this study, 396 Jordanian female patients diagnosed with breast cancer (BC) aged between 25–65 years attending BC clinics at the Jordanian Royal Medical Services in Jordan for the management and follow-up of their conditions during the period from January 2013 to July 2014. Patients were screened for leptin hormone level. The experimental design was prospective observational that permitted to include 134 newly-diagnosed BC patients who were naïve to any type of treatment interventions and 262 recently-diagnosed BC patients (up to three months) who were exposed to any type of treatment interventions. Recently, group members were sub-divided into sub-groups to control exposure to chemotherapy. The study design also permitted to include pre- and postmenopausal BC patients for hormonal balance control. The sample size (396) was statistically sound and accounts for about 50% of BC cases in the year 2011. The median age of females with BC in Jordan is 51 years old, and 80% of the cases age ranged between the ages of 25 and 65 years old.^[11] Exclusion criteria was also determined; any clinical or laboratory evidence of congestive heart failure, coronary disease, chronic renal failure, polycystic ovary syndrome, thyroid diseases, pregnancy and lactation. Moreover, subjects below 25 or above 65 years of age, type I diabetes mellitus, epilepsy and those taking medical herbs were also excluded.

Also, any subject who did not fit the inclusion criteria was excluded. This study was conducted according to the Declaration of Helsinki (2008, including 2013 amendments) and willingly; all participants had read and signed an informed consent form at the start of the study. The Royal Medical Services Ethical Committee approved this study (reference number 1/2013).

2.2. Data collection

A validated and reliable questionnaire was adopted for data collection which included personal information, health, anthropometric and biochemical measurements.

2.2.1. Anthropometric measurements

Anthropometric indicators comprised of: height, weight, waist circumference (WC) and hip circumference (HC) were measured in duplicates with subjects lightly clothed and without shoes. These indicators were performed by the investigator following the methodological protocol.^[12] The body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. The BMI ≥ 30 kg/m² was considered obese.^[13] The waist to hip ratio (WHpR) was calculated as WC divided by HC, while the waist to height ratio (WHtR) was calculated as WC divided by Height.

2.2.2. Biochemical analyses

Blood samples were collected after minimum 12 hours of fasting. The serum had been harvested and stored at -80°C for analysis. Biochemical measurements were analyzed in Princess Eman Center for Laboratory Research and Science. The following laboratory measurements were performed in duplicates for each subject and the mean values were taken in subsequent calculations for biomarkers such as fasting blood glucose (FBG), fasting blood insulin (FBI) and C-peptide. For the analysis of triglycerides (TG), total cholesterol (TC), high density lipoprotein (HDL-C) and low density lipoprotein (LDL-C) in human serum, the enzymatic colorimetric assay was applied. The upper normal limits used for TC and LDL-C, were 200 mg/dl and 150 mg/dl, respectively. Lower limit for HDL was < 50 mg/dl. Plasma glucose was determined by the glucose dehydrogenase method (Wako Pure Chemical Industries, Ltd., Osaka, Japan). C-peptide was measured by a solid-phase, two-site chemiluminescent immunoassay (IMMULITE 2000 C-peptide assay, Siemens AG, Erlangen, Germany).

The fasting blood insulin levels were quantitatively determined by chemiluminescent microparticle immunoassay (CMIA) technology (ARCHITECT Insulin assay, Abbott Laboratories, IL, USA). The Manual Enzyme-Linked Immunosorbent Assay (ELISA) was used for the quantitative determination of leptin levels in serum by an enzyme immunoassay method (dbc-Diagnostics Biochem Canada Inc., Canada). The insulin sensitivity was then calculated using HOMA according to the following formula^[14]:

$$\text{Log (HOMA)} = \log [\text{FBG (mmol/L)} \times \text{FBI (}\mu\text{U/ml)} / 22.5].$$

2.2.3. Blood pressure

Two blood pressure readings were recorded in an upright sitting position by a licensed staff nurse using a standard mercury sphygmomanometer after seating the subjects for at least 15 minutes. The average value was recorded and blood pressure was considered high if SBP ≥ 130 mmHg and/or DBP ≥ 85 mmHg.^[15] The cut-off points for SBP, DBP, TG, and HDL-C were determined based on Alberti *et al.* definition of metabolic abnormalities.^[15] We consider high normal DBP and DBP levels among BC patients as a HTN risk as that of metabolic syndrome.^[15]

3. Statistical Analysis

Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS), version 10.0 (SPSS Inc., Chicago, USA). For all analyses, a probability value of < 0.05 was considered statistically significant. Results were expressed according to the study needs as either frequency distribution with their percentages (%) or means $\pm$ standard error of mean (SEM). Frequency distribution and percentages or means $\pm$ SEM were performed for the health characteristics, prevalence of dyslipidemia and hypertension, and the menopausal status was compared according to the type of treatment exposure. The independent sample t-test or the chi-squared test were used between high density lipoprotein (HDL-C) and low density lipoprotein (LDL-C), systolic and diastolic blood pressure, and menopausal status in addition to various BC status.

4. Results

Dyslipidemia and hypertension characteristics of the study sample according to treatment exposure are shown in Table 4.1. Systolic blood pressure (SBP) was

higher ($p < 0.05$) in chemo (123.0 ± 0.9 mmHg) than in non-chemo (119.5 ± 1.2 mmHg) and newly-diagnosed (119.1 ± 1.1 mmHg) groups. Total cholesterol (TC) and triglycerides (TG) were higher ($p < 0.05$) in recently-diagnosed (244.7 ± 3.0 vs. 211.1 ± 4.7 mg/dl) compared to newly-diagnosed (227.1 ± 4.3 vs. 195.7 ± 5.8 mg/dl) group. High density lipoprotein cholesterol (HDL-C) was lower ($p < 0.05$) in non-chemo (42.9 ± 1.0 mg/dl) compared to chemo (47.6 ± 0.7 mg/dl) and newly-diagnosed (47.8 ± 0.8 mg/dl) groups (Table 4.1).

Prevalence of the dyslipidemia risk factors and hypertension according to treatment exposure are presented in Table 4.2. The prevalence of elevated SBP (≥ 130 mmHg) was significantly ($p < 0.05$) higher in chemo group (19.3%) than non-chemo (9.3%) and newly-diagnosed (10.4%) groups (Table 4.9). The prevalence of increased TG (≥ 150 mg/dl) and TC (≥ 200 mg/dl) were more frequent ($p < 0.05$) in the recently-diagnosed group (82.4% vs. 80.9%) than in the newly-diagnosed patients (76.1% vs. 67.2%) for TG and TC, respectively. The prevalence of low HDL-C (< 50 mg/dl) was higher ($p < 0.05$) in non-chemo group (81.4%) than chemo (63.6%) and newly-diagnosed (61.2%) groups. No statistical significant difference was found with respect to the frequency of high DBP (18.4%), ($p \geq 0.05$) among the study groups (Table 4.2).

Prevalence of dyslipidemia risk factors and hypertension in pre and post-menopause according to treatment exposure are shown in Table 4.3. The prevalence of abnormal TG and HDL-C respectively, was significantly higher ($p < 0.05$) in the premenopausal (43.4% and 38.4%) than in the postmenopausal BC patients (36.9% and 28.3%). Compared to the premenopausal BC patients, the postmenopausal BC patients had significantly higher ($p < 0.05$) prevalence of abnormal SBP (9.3% vs. 4.8%) and DBP (11.4% vs. 7.1%) (Table 4.3).

Dyslipidemia and hypertension characteristic in pre and postmenopausal women according to treatment exposure are shown in Table 4.4. In postmenopausal BC patients, SBP was higher ($p < 0.05$) in the recently-diagnosed (136.2 ± 1.2 mmHg) compared to the newly-diagnosed group (121.1 ± 1.7 mmHg). Statistical significant differences were found ($p < 0.05$) between postmenopausal and premenopausal BC patients in TG (221.0 ± 5.9 vs. 195 ± 4.7 mg/dl), SBP (125.0 ± 1.0 vs. 118 ± 0.8 mmHg). However, no statistical significant difference was found with ($p \geq 0.05$) in HDL-C (47.0 ± 0.8 vs. 46.0 ± 0.6 mg/dl) and DBP (80.0 ± 0.7 vs. 78.0 ± 0.6 mmHg) among postmenopausal and premenopausal BC patients, respectively (Table 4.4).

Age-controlled partial correlation coefficients are presented in Table 4.5. The WHtR and WC respectively

Table 4.1: Dyslipidaemia and hypertension characteristics according to treatment exposure ⁽¹⁻³⁾

Character	Newly-diagnosed (n = 134)		Recently - diagnosed (n=262)						Whole sample (n = 396)	
			Non- chemo (n = 86)		Chemo (n = 176)		Total (n = 262)			
	Mean ± SEM		Mean ± SEM		Mean ± SEM		Mean ± SEM		Mean ± SEM	
SBP (mmHg)	119.1	1.1 ^a	119.5	1.2 ^a	123.0	0.9 ^b	121.9	0.8 ^a	120.9	0.6
DBP (mmHg)	78.4	1.0 ^a	78.0	0.8 ^a	79.6	0.7 ^a	79.1	0.5 ^a	78.8	0.5
HDL-C(mg/dl)	47.8	0.8 ^a	42.9	1.0 ^b	47.6	0.7 ^a	45.6	0.6 ^b	46.4	0.5
LDL-C (mg/dl)	126.9	9.0 ^a	141.0	3.4 ^a	128.8	2.8 ^a	132.8	2.2 ^a	130.8	3.4
TG (mg/dl)	195.7	5.8 ^a	221.8	8.9 ^b	205.9	5.6 ^b	211.1	4.7 ^b	205.9	3.7

1. Values are given as mean ± SEM.

2. Values in rows with different superscripts are significantly different (p < 0.05).

3. Abbreviations and definitions: newly diagnosed: newly- diagnosed: breast cancer patients who are not exposed to any type of interventions; recently-diagnosed: breast cancer patients within 3 months of diagnosis who are either exposed (chemo) or not exposed (non- chemo) to chemical therapy; SEM: stander error of mean; SBP: systolic blood pressure, DBP: diastolic blood pressure, TC: total cholesterol; TG: triglycerides; HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol.

Table 4.2: Prevalence of the dyslipidemia risk factors and hypertension according to treatment exposure⁽¹⁻⁴⁾

Variables	Cut-off points	Newly-diagnosed (n = 134)		Recently - diagnosed (n = 262)						Whole sample (n = 396)	
				Non- chemo (n = 86)		Chemo (n = 176)		Total (n = 262)			
		n	%	n	%	n	%	n	%	n	%
SBP (mmHg)	> 130	14	10.4 ^a	8	9.3 ^a	34	19.3 ^b	42	16.0 ^b	56	14.1
DBP (mmHg)	> 85	24	17.9 ^a	11	12.8 ^a	38	21.6 ^a	49	18.7 ^a	73	18.4
Overt hypertension*		31	23.1	10	11.6	49	27.8	59	22.5	90	22.7
HDL-C (mg/dl)	< 50	82	61.2 ^a	70	81.4 ^b	112	63.6 ^a	182	69.5 ^b	264	66.7
TG (mg/dl)	> 150	102	76.1 ^a	73	84.9 ^b	143	81.2 ^b	216	82.4 ^b	318	80.3
TC (mg/dl)	> 200	90	67.2 ^a	75	87.2 ^b	137	77.8 ^b	212	80.9 ^b	302	76.3
Overt dyslipidemia*		7	5.2	2	2.3	10	5.7	12	4.6	19	4.8

1. Values given as number of patients (n) and their percentages out of (N).

2. Values in rows with different superscripts are significantly different (p < 0.05).

3. Cut-off points were based on Albertiet al., 2009 definition of metabolic abnormalities.^[15]

4. Abbreviations and definitions: newly-diagnosed: breast cancer patients who are not exposed to any type of interventions; recently-diagnosed: breast cancer patients within 3 months of diagnosis who are either exposed (chemo) or not exposed (non-chemo) to chemical therapy; SEM: stander error of mean; SBP: systolic blood pressure, DBP: diastolic blood pressure, TC: total cholesterol; TG: triglycerides; HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol.

* The study patients who are on anti-hypertensive or lipid lowering agents.

Table 4.3: Prevalence of dyslipidemia risk factors and hypertension in pre and postmenopause according to treatment exposure⁽¹⁻⁵⁾

Metabolic risk	Newly-diagnosed (N = 134)		Recently-diagnosed (N = 262)		Whole sample (N = 396)	
	Premeno-pause (N = 80)		Postmeno-pause (N = 54)		Premeno-pause (N = 229)	
	n	%	n	%	n	%
SBP'(mmHg)	6	4.5	8	6.0	13	5.0
DBP'(mmHg)	8	6.0	16	11.9	20	7.6
HDL'(mg/dl)	48	35.8	34	25.4	104	39.7
TG'(mg/dl)	56	41.8	46	34.3	116	44.3

1. Documented international cut-off points: Alberti et al.^[15]

2. Values are given as number of patients (n) and their percentages out of (N).

3. (*) Significantly (p < 0.05) different between pre and postmenopausal women for each treatment exposure and whole sample.

4. Cross differences between treatment exposure groups were not significant (p > 0.05).

5. Abbreviations and definitions: newly diagnosed: breast cancer patients who are not exposed to any type of interventions; recently diagnosed: breast cancer patients within 3 months of diagnosis who are either exposed (chemo) or not exposed (non- chemo) to chemical therapy; SBP: systolic blood pressure; DBP: diastolic blood pressure; TG: triglycerides; HDL-C: high density lipoprotein.

Table 4.4: Dyslipidemia and hypertension characteristic in pre and postmenopause women according to treatment exposure⁽¹⁻⁵⁾

Metabolic risks	Newly-diagnosed (N = 134)				Recently-diagnosed (N = 262)				Whole sample (N = 396)			
	Premeno-pause (N = 80)		Postmeno-pause (N = 54)		Premeno-pause (N = 149)		Postmeno-pause (N = 113)		Premeno-pause (N = 229)		Postmeno-pause (N = 167)	
	Mean ± SEM		Mean ± SEM		Mean ± SEM		Mean ± SEM		Mean ± SEM		Mean ± SEM	
SBP^a(mmHg)	117.1	1.5	121.1	1.7 ^a	118.5	0.9	136.2	1.2 ^b	118.0	0.8	129.3	1.0
DBP(mmHg)	76.6	1.4	80.9	1.1	78.3	0.6	80.2	1.0	78.0	0.6	80.0	0.7
HDL-C(mg/dl)	47.9	0.9	47.7	1.6	44.8	0.8	46.7	0.9	46.0	0.6	47.0	0.8
TG^a(mg/dl)	182.5	6.9	215.3	9.6	201.7	6.1	223.5	7.4	195.0	4.7	221.0	5.9

1. Documented international cut-off points: Alberti, *et al.*^[15]

2. Values are given as mean ± SEM.

3. (*) Significant differences ($p < 0.05$) between pre and postmenopausal women for treatment exposure groups and whole sample.

4. Values in rows with different superscripts are significantly different among treatment exposure groups ($p < 0.05$). None of other values in rows show significant differences ($p > 0.05$).

5. Abbreviations and definitions: newly diagnosed: breast cancer patients who are not exposed to any type of interventions; recently diagnosed: breast cancer patients within 3 months of diagnosis who are either exposed (chemo) or not exposed (non- chemo) to chemical therapy; SBP: systolic blood pressure; DBP: diastolic blood pressure; TG: triglycerides; HDL: high density lipoprotein; SEM: stander error of mean.

Table 4.5: Age-controlled partial correlation coefficients of dyslipidemia risk factors and hypertension with obesity indices and selected biomarkers in the study sample⁽¹⁻²⁾

Metabolic risks	BMI	WC	WHpR	WHtR	leptin	HOMA	C-peptide
SBR(mmHg)	0.04	0.08	0.12	0.08	0.10	0.14**	0.03
DBR (mmHg)	0.01	0.17***	0.15**	0.13*	0.08	0.10*	-0.02
HDL-C(mg/dl)	-0.11*	-0.11*	-0.09	-0.13**	-0.02	0.02	0.03
TG (mg/dl)	0.05	0.16***	0.08	0.15**	-0.11*	0.03	0.02

1. *: ($p < 0.05$); **: ($p < 0.01$); ***: ($p < 0.001$).

2. Abbreviations and definitions: newly diagnose: BMI: body mass index; WC: waist circumferences cm; WHtR: waist to height ratio; WHpR: waist to hip ratio; HOMA: homeostasis model assessment according to the following formulas^[14]: Log (HOMA) as $\log [\text{FBG (mmol/L)} \times \text{FBI } (\mu\text{U/ml})/22]$ ^[15]

were significantly ($p < 0.05$) correlated with DBP ($r = 0.13$ vs. $r = 0.17$), HDL-C ($r = -0.13$ vs. $r = -0.11$) and TG ($r = 0.15$ vs. $r = 0.16$). The HDL-C was negatively correlated ($p < 0.05$) with BMI ($r = -0.11$), WC (-0.11) and WHtR (-0.13). While TG was negatively correlated ($p < 0.05$) with leptin level ($r = -0.11$). Both SBP and DBP were strongly correlated ($p < 0.05$) with HOMA ($r = 0.14$) and ($r = 0.10$), respectively (Table 4.5).

5. Discussion

5.1. Dyslipidemia and breast cancer

In this study, the prevalence of known cases of dyslipidemia was 5%. Nevertheless, high triglycerides (TG) and low high density lipoprotein cholesterol (HDL-C) were observed in 80% and 67% of study sample, respectively. Results from this study seem to be higher than those reported in non-BC patients in Jordan. Studies investigating dyslipidemia in breast cancer (BC) patients are scarce. A study by Hammoudeh *et al.* showed that the prevalence of high TG was 55% and

low HDL-C was 39%.^[8] Another study by Khader *et al.* found that 39% of women had high TG and 28% had low HDL-C.^[9] Furthermore, a resembling study conducted in Italy among BC patients reflected lower results; with prevalence of high TG and low HDL-C represented in the percentage of 34.7% and 38.4%, respectively.^[16] This variation emerged from several reasons. Improper data reporting provided by participants regarding the duration of fasting, the inconsistent definition of dyslipidemia, characteristics of the target group and different study design could all justify this variation.

In the current study, the prevalence of high TG and low HDL-C level were significantly different among recently-and newly-diagnosed BC patients. These differences may be due to possible medical interventions such as chemotherapy or surgery. Present study showed that prevalence of high TG and low HDL-C level in premenopausal women was higher than in postmenopausal women, but the mean value of TG level was significantly higher ($p < 0.05$) in postmenopausal BC women. In accordance, higher risk of dyslipidemia in BC patients over 60 years old has been reported.^[17,18] In a longitudinal prospective study

by Furberg *et al.* concluded that the serum HDL-C was only inversely related to the risk of postmenopausal BC in overweight women.^[19] In another study, HDL-C has been shown to be inversely associated with BC risk among premenopausal women, and positively associated with BC risk in postmenopausal women.^[20] Some studies demonstrated positive association between TG and postmenopausal BC risk,^[16,21] whereas, Bahl *et al.* established that lipid profile does not affect BC outcome.^[4]

5.2. Hypertension and breast cancer

In the present study, overt hypertension (HTN) was observed in almost 23% of BC patients. In Jordan, studies that investigate the link between HTN and BC in Jordan are absent. In a study by Hammoudeh *et al.*, the prevalence of HTN in Jordanian healthy population was 63%, which is much higher than the observed results in this study.^[8] The prevalence of high systolic blood pressure (SBP) in the present study was 14.1% and 18.4% for diastolic blood pressure (DBP). This prevalence is high compared to another study by AL-Odat *et al.*, in which it was found that 13.2% of adult non-BC female had high SBP and/or DBP.^[10] The variations may be due to the differences in ethnicity, study design, BC patient's characteristics and sampling technique.

In the present study, SBP, in recently-diagnosed BC and in those taking chemotherapy, was found higher than in newly-diagnosed patients. These findings are consistent with a study by Aparicio-Gallego *et al.*, which concluded high prevalence of HTN in patients taking chemotherapy.^[22] The positive association between exposure for chemotherapy and risk of HTN has been also observed by other studies.^[23]

In this study, the prevalence of both SBP and DBP is higher in postmenopausal than premenopausal women. This result is consistent with a Latin American study, where both SBP and DBP were associated with increased BC risk in postmenopausal women, a matter that suggests the role of increased insulin resistance and obesity.^[24] Similar results have been reported by other studies.^[25] However, in a study by Agnoli *et al.*, HTN was insignificantly associated to postmenopausal BC patients.^[16]

The available evidence regarding the relationship between HTN and BC risk are inconsistent and variable.^[26] In the present study, both SBP and DBP were correlated with HOMA, whereas DBP was strongly correlated to WC and central obesity; there are other studies that showed positive correlation between HTN and insulin resistance.^[27] Former Jordanian studies

established a high prevalence of insulin resistance and obesity indices among BC women.^[28,29] Obesity is a risk factor for both postmenopausal BC and HTN.^[24] Studies in which BMI is controlled, the relation between HTN and BC was found to be significant with 20% increased risk of BC in postmenopausal women with HTN.^[25] However, a study by Peeters *et al.* demonstrated the opposite results and findings.^[30]

In conclusion, dyslipidemic and hypertensive biomarkers were prevalent among BC patients. Moreover, the risk increased with obesity and age, as it was higher in postmenopausal BC women. Furthermore, treatments' exposure especially to chemotherapy increased the risk of dyslipidemia and hypertension. This may be considered as biomarker for BC prognosis after exposure to treatments and warranted a closer attention by health care professionals, in order to improved outcomes after diagnosis and treatment exposure with more concern regarding postmenopausal BC women.

To the best of authors' knowledge, this study is the first in Jordan to evaluate the concomitance between lipid profile and HTN among BC women. As for limitations, however, the study suffered from limited sample size since it was conducted in only one tertiary hospital. Moreover, there are some structural limitations regarding the study design.

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Conflict of interest

None declared.

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