

Research Article

Association between Serum Insulin and Interleukin-6 levels with Breast Cancer in Post-Menopausal Iraqi Women

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Abstract:

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Background: Obesity rates are increasing day by day affecting all populations at different ages. The most prevalent kind of cancer among women worldwide is breast cancer, with increasing rates in the present time and the future. Substantial connections between obesity and breast cancer are demonstrated. Elevated circulating levels of insulin and interleukin-6 have a substantial link to obesity and breast cancer development.

Objective: This study aimed to examine the serum levels of insulin and interleukin-6 in postmenopausal breast cancer patients and to study the connection between these biomarkers and breast cancer development.

Method: In this research, 120 postmenopausal women actively participated., 30 had a body mass index of $>30 \text{kg/m}^2$ diagnosed with breast cancer and considered as obese (group 1), 30 with a body mass index of $<30 \text{kg/m}^2$ with breast cancer and considered as not obese (group 2), 30 had a body mass index of $>30 \text{ kg/m}^2$ who were apparently healthy and were obese (group 3), and 30 participants with a body mass index of $<30 \text{ kg/m}^2$, were apparently healthy and were not obese (group 4). Five millilitres of venous blood were provided by each individual. Insulin and interleukin-6 levels in the blood were measured using ELISA (enzyme-linked immunosorbent assay) kits.

Results: Serum insulin levels were higher in the groups of obese participants (groups 1 and 3) than the groups of non-obese participants (groups 2 and 4), serum levels of interleukin-6 were the lowest in the healthy control group without obesity (group 4). Substantial moderate correlation was found between body mass index and both insulin and interleukin-6. Postmenopausal breast cancer risk is increased with elevations of serum insulin and interleukin-6.

Conclusions: Elevated levels of serum interleukin-6 and insulin were linked to higher body mass index. In addition, in addition, interleukin-6 elevated levels were linked to the cancer state of the non-obese patients. The Risk of breast cancer increases with higher levels of circulating insulin and interleukin-6. **Keywords:** Breast Cancer; Insulin; Interleukin-6; Obesity; Postmenopausal Women.

Introduction

Obesity rates worldwide have tripled since 1975, 650 million adults, 340 million adolescents, and 39 million children are obese. This magnitude continues to escalate. In 2025, the WHO predicts that 167 million individuals of different ages will end up being at risk of becoming less healthy due to being overweight or obese (1).

Breast cancer is reported to be the most frequently reported malignancy among women around the world, passing the 2.3 million documented cases and a range of 685,000 deaths in 2020, by the year of 2040 the newly diagnosed cases number will rise to about 3 million cases, and a rate of 1 million deaths making a huge burden on the healthcare institutes worldwide(2, 3). Many mechanisms have been attributed to the connection between breast cancer and obesity, and the role of leptin has been postulated and reviewed in many studies (5-7). Interleukin-6 is produced by adipocytes (8), tumor cells, endothelium cells, T cells, and B cells. IL-6 has a wide range of physiological effects on a range

* Corresponding author: mohammed.abd1200m@copharm.uobaghdad.edu.iq of cells (9). By increasing the levels of angiogenic and anti-apoptotic proteins in tumor cells, IL-6 can accelerate the growth of tumors (10).

The Janus kinase-2 (JAK2)/Signal transducer and activator of transcription- 3 (STAT3) signaling pathway appears to play a role in IL-6's ability to cause cancer. The IL-6 receptor directly activates this crucial anti-apoptotic and proliferative pathway in tumor cells (11).

Furthermore, since insulin can stimulate IL-6 flow into the bloodstream and to enhance TNF- α expression of genes in adipocytes, IL-6 offers intriguing connections between the occurrence of inflammation and insulin (12).

In addition to its key role as a growth factor by encouraging cellular division and migration and hindering apoptotic processes, insulin's principal action is to control the metabolic processes related to carbohydrates, lipids, and proteins. These effects may be amplified in situations of deregulated insulin sensitivity and further functional decline of insulin regulated metabolic processes.(13).

J Fac Med Baghdad 323 Vol.65, No4, 2023

Generally, the mitogenic actions related to insulin entail activating Ras and the mitogen-activated protein kinase (MAPK) pathway, whereas the phosphatidylinositol 3-kinase (PI3K) pathway mediates actions of insulin related to metabolism, such as glucose uptake by the cells, in the presence of insulin resistance and hyperinsulinemia, the PI3K pathway's ability to be stimulated by insulin is lost, but MAPK activation is increased and the amount of Ras protein that is prenylated by insulin is increased (14, 15). Through activating the PI3/Akt cascade, actions linked to direct stimulation are exerted by insulin, which affects the production of capillarylike tube and the migration of vascular endothelial (16). Moreover, insulin has its strong stimulatory effects on micro-vascular epithelial cells, including those of vessels associated with breast cancer stromal tissues (17).

The study aims to determine the involvement of the inflammatory state of obesity in breast cancer development by measuring and comparing the circulating levels of insulin and IL-6 in a sample of postmenopausal obese and non-obese Iraqi women diagnosed with breast cancer to their corresponding healthy control groups. In addition to discovering the predictive value of these biomarkers in postmenopausal breast cancer development.

Material and Method Study Design

This is a case-control study in which 120 participants were enrolled and were divided into four groups.: 30 with obesity state (BMI > 30 kg/m²) and breast cancer (group 1), 30 were not with obesity state (BMI < 30 kg/m²) but had breast cancer (group 2), and 30 were healthy and with obesity state (BMI > 30 kg/m²) (group 3), and 30 were healthy and without obesity (BMI<30mg/m²) (group 4).

Setting and Duration of the Study

Sample collection was done at the consultant clinic at the oncology teaching hospital in Medical City, Baghdad, Iraq in the period between October 2021 and February 2022. The purpose of the study was explained to each person who had signed up for it, and their agreement was obtained. In addition, details regarding participants' ages, medical history medication history, and menopausal status were gathered from them directly and recorded.

Target Population: Participants recruited in this study were postmenopausal women (with no menstrual cycle in the last 12 months), newly diagnosed (confirmed by mammography and/or PET scanning) with breast cancer of stages 1 and 2, postoperative, who are not using chemotherapy, biological therapy or hormonal therapy.

Inclusion Criteria: Postmenopausal women (with no menstrual cycle in the last 12 months), newly diagnosed (confirmed by mammography and/or PET scanning) with breast cancer of stages 1 and 2, postoperative, whom are not using chemotherapy, biological therapy or hormonal therapy. Subjects with no history of using medications that could

cause insulin resistance (such as certain some psychiatric medications: olanzapine, quetiapine, clozapine and resperidone and steroids) and medications that increase insulin sensitization (such as metformin, and thiazolidinediones), and medications that cause increased insulin secretion (sulphonylureas) in the last three months.

-Non-smokers.

Exclusion Criteria

- -Patients having metastasis to bone or other tissues (confirmed by PET scanning).
- -Male gender
- -Subjects with renal or liver diseases (which can affect the levels of studied biomarkers).
- -History of any autoimmune diseases (which may affect the levels of studied biomarkers).
- -Subjects with a history of cardiovascular disease (which may affect the levels of the studied biomarkers).

Study Variable:

A sample of venous blood measuring five milliliters (ml) was collected from each participant. To separate the serum for testing, it was then collected into a gel tube and centrifuged for 10 to 15 minutes at a speed of 4,400 rounds per minute (rpm). By the use of enzyme-linked immunosorbent assay (ELISA) kits, the levels of serum insulin and IL-6 were determined.

Body mass index was calculated for each participant enrolled in the study by using the following equation:

BMI (kg/m2) = weight / Height2

Each participant's waist circumference was also measured with a tape measure and recorded in (cm).

Statistical Analysis

The statistical evaluation was carried out using IBM SPSS Statistics 25 for Windows. To ascertain if the data were distributed normally, the Shapiro-Wilk test was employed. The median and interquartile ranges (IQR) for each participant were computed. Utilizing Kruskal-Wallis test, the groups were compared. For correlation investigations, the Spearman's correlation test was used. Binary logistic regression was used to determine the odds ratio of each marker with breast cancer.

Results:

Anthropometric results for the participants enrolled in the study are described in table 1. The current study demonstrated that group 2 showed the lowest median (IQR) of age (years) than the other groups involved in the study. The current study found that there was no recognizable difference between groups 1 and 3 and groups 2 and 4 in weight. The lowest median (IQR) in height (m) was group 3. Furthermore, results obtained in this study illustrated that groups 1 and 3 had higher median (IQR) of BMI (kg/m2) and waist circumference (cm) than groups 2 and 4.

| Variable | Group 1 Obese B.C. N=30 | Group 2 Non- obese B.C. N= 30 | Control Obese N-20 | Control Non Obese N=30 Group 3 | pValue |
|------------------------------------|-------------------------------|-------------------------------------|-----------------------|--------------------------------|--------|
| Age (year) | 54.5 (11.25) | 52.00 (6) | 57 (7) | 55.50 (5) | 0.015 |
| Weight | 85.00 | 61.50 | 80.00 | 66.00 | < 0.00 |
| (kg) | (9.5) | (8.5) | (12.5) | (12.25) | 1 |
| Height | 1.5850 | 1.6100 | 1.5800 | 1.6250 | 0.004 |
| (m) | (0.09) | (0.06) | (0.10) | (0.09) | 0.004 |
| BMI (kg/m²) | 31.8150 (3.64) | 24.0850 (5.43) | 32.665 0 (3.61) | 24.715 0 (4.91) | <0.00 |
| Waist Circumfe rence (cm) | 103.00 (12.25) | 98.00(8) | 107.5 (12.25) | 100.00 (7.5) | <0.00 |

Furthermore, current study found that groups 1 and 3 showed higher serum insulin levels than both groups 2 and 4, with no substantial difference between groups 1 and 3 and groups 2 and 4. In addition, IL-6 levels were lowest for group 4 compared to the other three groups involved in the study as shown in table 2, figure 1 and figure 2

Table 2 Median Serum Insulin and Interleukin 6 of study Groups

| | Group 1 | Group 2 Non | Group 3 | Group 4 |
|---------|-----------------------|----------------------------|----------------|--------------------------|
| | Obese | obese B.C | Obese | Non-Obese |
| | B.C | N=23 | Control | Control |
| | N=30 | | N=22 | N=21 |
| Insulin | 6.1160 ^{a,b} | 5.6560 ^{a,c} | $6.0250^{c,d}$ | 5.3760 ^{b,d} |
| (mIU/l) | (1.00) | (0.78) | (1.23) | (2.16) |
| IL-6 | 64.1950a | 61.8200 ^b (7.8) | 62.5825° | 53.4705 ^{a,b,c} |
| (ng/l) | (7.55) | | (3.27) | (12.03) |

Data presented in the table were written in terms of [Median (IQR)]. Subscriptions (a, b, c) indicate significant differences between groups.

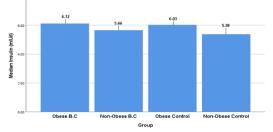


Figure 1. Median serum insulin (mIU/l) among the study groups.

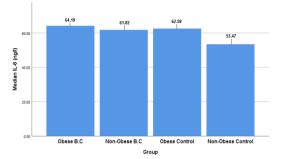


Figure 2 Median Serum Interleukin-6 among the Study Groups

This study demonstrated the existence of moderate positive correlation between BMI and both insulin and IL-6 serum levels, while a positive negative correlation was found between insulin and IL-6, as shown in table 3.

Table (3): Correlation Studies among variables investigated in the study

| | Correlation | Insulin (mIU/l) | IL-6 (ng/l) |
|------------|-------------|--------------------|-------------|
| BMI | р | 0.390 | 0.423 |
| (kg/m^2) | P-value | < 0.001** | <0.001** |
| Insulin | р | | 0.256 |
| (mIU/l) | P-value | | 0.005** |

^{**} Indicate correlation is significant at the 0.01 level (2-tailed).

Table (4): Association of Insulin and interleukin-6 in breast cancer risk

| Variable | Odds Ratio | P-value | |
|---------------|------------|---------|--|
| Insulin | 1.864 | 0.005 | |
| Interleukin-6 | 1.117 | 0.001 | |

Discussion

In this study, fasting serum insulin levels were higher in the groups of obese participants (groups 1 and 3) compared to the groups of non-obese participants (groups 2 and 4) shown in table 3. In addition, a positive correlation was found between insulin and BMI. Furthermore, this study found that elevations of serum insulin levels are associated with increased risk of breast cancer development. It has been suggested that the rise in plasma insulin level seen in obese individuals is an adaptation strategy to poor insulin sensitivity brought on by both increased insulin production and decreased insulin clearance (18), yet it has also been suggested that elevated insulin release from β -cells in obese individuals is a basic aberration that precedes and aids in the emergence of insulin resistance (19). Insulin plays a crucial role in the process of cell growth by encouraging cell division and migration and inhibiting apoptosis(13), these mitogenic processes are mediated by Ras protein and the mitogenactivated protein kinase (MAPK), during insulin resistance the prenylated Ras protein is elevated and MAPK activation is increased(15). By activating the PI3K/Akt pathway, insulin exerts its own specific stimulatory effects on capillary-like tube formation and vascular endothelial cell migration(16). Insulin also encourages neovascularization indirectly. First, it can stimulate the expression of vascular endothelial growth factor (VEGF) in vascular endothelial cells(20), which has an autocrine effect on the development and tube formation of microvascular endothelial cells, including those of vessels associated with breast cancer stromal tissues. Second, insulin can boost VEGF synthesis and release from adipocytes, allowing a paracrine relationship to form between adipocytes of the peritumor adipose tissue, the tumor capsule, and the tumor-associated vascular endothelial cells(21). Where in fasting insulin levels in two groups, obese patients and healthy control had a mean BMI of 30.4 (kg/m²) and found higher levels of serum insulin

(16.4 mIU/ml) in patients than the control group (13.7 mIU/ml). However the association of insulin resistance with breast cancer development and revealed that elevated insulin levels are associated with breast cancer development (OR, 1.29)(22).

The current study revealed that group 4 (healthy control without obesity) had the lowest IL-6 serum levels than the other groups. Also, a substantial positive moderate correlation existed between IL-6 and BMI. Furthermore, the study found that higher serum IL-6 levels are associated with higher risk of breast cancer development as shown in table 5. IL-6, a pro-inflammatory cytokine, is secreted by a variety of cells in the tumor microenvironment, including malignant cells. Tumor cells growth and differentiation are significantly influenced by IL-6 levels(23). In addition, the production of plasma IL-6 by adipose tissues makes up around one-third of the total. The non-adipocyte components of adipose tissue serve as the primary source of IL-6 in healthy adipose tissue, but pathological situations including obesity and cancer greatly increase the quantities of IL-6 released by adipocytes(24). While in study of the serological levels of IL-6 in a group of 80 patients with breast cancer and a group of 80 controls, results obtained were that the control group had lower levels of IL-6 compared to patients group(25) .in other hand compared the levels of IL-6 in a non-obese group of patients with breast cancer with a non-obese control groups and found that the levels of IL-6 are higher in the patients group than the control group(26). Furthermore, several studies found that the serum levels of IL-6 was higher in breast cancer group than the control group (27-29). The increased oestrogen synthesis from C19 steroids in obesity may be the result of an increase in the amount of IL-6-secreting macrophages infiltrating the adipose tissue, which raises the risk of breast cancer in postmenopausal obese women(30).

Conclusions:

Women whom are obese with or without breast cancer had higher levels of serum insulin. In addition, healthy women without obesity had lower levels of IL-6 than the other groups. Also, a significant correlation existed between BMI and both insulin and IL-6. Elevations of serum levels of insulin and IL-6 increase the risk of breast cancer development in postmenopausal women.

Authors' Declarations:

Conflicts of Interest: The authors declare no conflict of interest.

We confirm that all the Figures and Tables in the manuscript belong to the current study. Besides, the Figures and images, which do not belong to the current study, have been given permission for republication attached to the manuscript. Authors sign on ethical consideration's approval-Ethical Clearance: The project was approved by the local ethical committee in (Place where the research was conducted or samples collected and treated)

according to the code number (193220) on (10/4/2021).

Author's Contributions:

Study conception & design: Mohammed A. Abd , Shaymaa Abdulzahra Abbas. Literature search: Mohammed A. Abd. Data acquisition: Mohammed A. Abd. Data analysis & interpretation: Mohammed A. Abd. Manuscript preparation: Mohammed A. Abd, Shaymaa Abdulzahra Abbas Manuscript editing & review: Mohammed A. Abd, Shaymaa Abdulzahra Abbas

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الارتباط بين مستويات الأنسولين المصل والإنترلوكين 6 مع سرطان الثدي لدى النساء العراقيات بعد انقطاع الطمث

محمد عبد الحسن عبد¹ ، شيماء عبد الزهرة عباس¹ افرع العلوم والمختبرات السريرية، كلية الصيدلة، جامعة بغداد، بغداد، العراق

الخلفية: تتزايد معدلات السمنة يومًا بعد يوم مما يؤثر على جميع السكان في مختلف الأعمار. أكثر أنواع السرطانات انتشارًا بين النساء في جميع أنحاء العالم هو سرطان الثدي، مع تزايد معدلاته في الوقت الحاضر وفي المستقبل. تم اثبات وجود ارتباط جوهري بين السمنة وسرطان الثدي. ترتبط المستويات المرتفعة من الأنسولين والإنترلوكين 6 في الدم ارتباطًا جوهريًا بالسمنة والاصابة بسرطان الثدي.

الاهداف: تهدف هذه الدراسة إلى فحص تأثير مستويات الأنسولين والإنترلوكين 6 في الدم في مرضى سرطان الثدي بعد سن اليأس ودراسة العلاقة بين هذه المؤشرات الحيوية وتطور سرطان الثدي.

المرضى وطرق العمل: في هذا البحث ، شاركت 120 امرأة في سن اليأس، 30 امرأة كان مؤشر كتلة الجسم لديهن > 30 كغم / م 2 ومصابات بسرطان الثدي ويعتبرن مصابات بالسمنة (المجموعة 1) ، 30 امرأة مع مؤشر كتلة الجسم أقل من 30 كغم / م 2 مصاببت بسرطان الثدي و يعتبر 30 شخصًا غير بدينين (المجموعة 2) ، وكان مؤشر كتلة الجسم لدى 30 منهم> 30 كغم / م 2 وكانوا غير مصابين بسرطان الثدي و يعانون من السمنة (المجموعة 3) ، وكان 30 مشاركًا بمؤشر كتلة جسم أقل من 30 كغم / م 2 ، على ما يبدو أصحاء ولم يكونوا يعانون من السمنة المفرطة (المجموعة 4). تم سحب خمسة مليلتر من الدم الوريدي من قبل كل امرأة مشاركة في البحث . يتم قياس مستويات الأنسولين والإنترلوكين -6 في الدم باستخدام تقنية مقايسة الممتز المناعى المرتبط بالانزيم .

النتائج: كانت مستويات الأنسولين في مصل الدم أعلى في مجموعات المشاركين الذين يعانون من السمنة المفرطة (المجموعتان 1 و 3) مقارنة بمجموعات المشاركين 6 في المصل هي الأقل في المجموعة الغير مصابة بسرطان الثدي والتي لا تعاني من المسنة المفرطة (المجموعة 4). تم العثور على علاقة معنوية ايجابية معتدلة بين مؤشر كتلة الجسم وكل من الأنسولين والإنترلوكين 6

الاستنتاجات: ارتبطت المستويات المرتفعة من الانترلوكين 6 والأنسولين في مصل الدم بارتفاع مؤشر كتلة الجسم. بالإضافة إلى ذلك، تم ربط المستويات المرتفعة من إنترلوكين -6 بالحالة السرطانية للمرضى غير البدينين. يزداد خطر الإصابة بسرطان الثدي مع ارتفاع مستويات مصل الدم لكل من الأنسولين والانترليوكين -6.

الكلمات المفتاحية: السمنة، سرطان الثدي، النساء بعد سن اليأس، الانسولين، الانترليوكين-6.

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