

Inflammatory Signatures Associated with Obesity in Patients with Stage II Breast Cancer Receiving Doxorubicin-Cyclophosphamide Therapy

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Abstract

Background: Obesity is being considered as a biological factor in altering the progression of breast cancer, as well as potentially influencing the response to treatment. It is marked by persistent low-level systemic inflammation mediated by adipose tissue-derived adipokines and cytokines, which can affect the tumor microenvironment and tumor response to chemotherapy. The objective of the study was to investigate the correlation between body mass index (BMI) and obesity-related inflammatory biomarkers in stage II breast cancer patients on the doxorubicin-cyclophosphamide chemotherapy. **Materials and Methods:** An analytical study was conducted on 120 Iraqi women having stage II breast cancer that was confirmed by histology. Every patient was given doxorubicin and cyclophosphamide depending on the body surface area (BSA) and evaluated in the third round of chemotherapy. BMI was used in stratifying the patients on the basis of normal-weight, overweight, and obese conditions (40 per group). Leptin, interleukin-1 β (IL-1 β), high-sensitivity C-reactive protein (hs-CRP), monocyte chemoattractant protein-1 (MCP-1), and cancer antigen 15-3 (CA15-3) serum concentrations were determined by means of standardized ELISA kits. The BioTek ELx800tm microplate reader was used to read absorbance. **Results:** Leptin, IL-1 β , hs-CRP, and MCP-1 were much higher in overweight and obese patients than in normal-weight patients, which showed that systemic inflammation is progressive in obesity. The serum level of CA15-3 was materially greater in the overweight and obese breast cancer patients than in the normal-weight patients, and also, there was no significant difference between the overweight and obese groups. **Conclusion:** The adipokine-rich and inflammatory profile of doxorubicin-cyclophosphamide chemotherapy in obese women with stage II breast cancer is linked to obesity. The results point to the possibility of obesity-related inflammation in the formation of the biological context of chemotherapy and justify the use of inflammatory profiling in individual breast cancer management.

Keywords: Breast cancer- Obesity- Chemotherapy- Inflammatory biomarkers- Leptin

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Introduction

Breast cancer is now considered the most commonly diagnosed cancer in women across the world, with a significant variation in response to chemotherapy, irrespective of early detection and treatment [1]. Obesity has become an important treatment outcome modulator, an inflammatory process of chronic low-grade systemic inflammation triggered by adipose tissue-derived

adipokines, cytokines, and chemokines that jointly facilitate tumor progression and decrease chemotherapy sensitivity [2].

Chronic inflammatory responses associated with obesity have been suggested in several events that facilitate resistance to anticancer therapies, which include increased tumor cell growth, tumor angiogenesis,

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immune dysregulation, and the activation of pro-survival pathways [3]. Leptin, interleukin-1 β (IL-1 β), high-sensitivity C-reactive protein (hs-CRP), and monocyte chemoattractant protein-1 (MCP-1/CCL2) are some of the inflammatory mediators that are critical in the connection between adiposity and cancer development and treatment outcome [4].

Leptin is an adipokine released during obesity, which facilitates the proliferation of breast cancer cells, angiogenesis, and resistance to chemotherapy [5]. The pro-inflammatory cytokine IL-1 β is linked to tumor-associated inflammation, enhanced hostility, and resistance to cytotoxic therapy, and hs-CRP indicates the systemic presence of inflammation and underwhelming prognosis and response to chemotherapy in breast cancer [6]. MCP-1 also causes a pro-tumorigenic microenvironment by recruiting monocytes and macrophages to adipose and tumor tissues, and may further suppress the efficacy of chemotherapy [7].

Although the relationship between breast cancer and obesity has been well documented, little information is known about the effects of obesity-induced inflammatory markers in mediating chemotherapy response, specifically in Middle Eastern communities [8]. In Iraq, where the rates of obesity among women are rising, there is a need to understand the biological pathways through which obesity influences cancer treatment [9].

Thus, the research will profile the chronic inflammatory milieu caused by obesity in patients undergoing standard chemotherapy, using doxorubicin and cyclophosphamide in breast cancer patients with a stage II malignancy. This study aims to offer an understanding of inflammatory processes linked to obesity that might affect chemotherapy and can be used in shaping treatment plans to be more personalized when treating breast cancer patients by measuring a select set of inflammatory markers, such as leptin, IL-1 β , hs-CRP, and MCP-1.

Materials and Methods

Subjects and study design

This is a cross-sectional research conducted to determine the effects of the chronic inflammation caused by obesity on chemotherapy response in women with breast cancer at stage II. The participants were screened and recruited at special oncology centers in Iraq, between June 2024 and August 2025, a sample size of 243 women with a history of stage II breast cancer who are aged 29 to 48 years. (Figure 1) A total of 120 participants were eventually picked and divided into three different groups depending on the Body Mass Index (BMI) to obtain a balanced distribution of the inflammatory profiling, where 40 participants were selected in each group to attain a balance in the statistics. The groups were as follows: control group, which was made of 40 normal-weight women (BMI 18.5-24.9 kg/m²), 40 overweight (BMI 25.0-29.9 kg/m²), and 40 obese women (BMI $\geq$ 30 kg/m²). The other 123 screened patients were not included into analysis as they did not pass the special BMI-based selection criteria or appeared with confounding

clinical conditions. The other exclusion criteria included pregnancy, smoking, diabetes mellitus, hypertension, autoimmune or chronic inflammatory disease not predetermined by obesity, long-term corticosteroid/immunosuppressive treatment, severe liver or renal failure, and the presence of metastatic disease. All the subjects were assessed in the third cycle of chemotherapy in order to be given the same amount of treatment exposure. The doses of chemotherapy were estimated according to the body surface area (BSA) of the subject, with the actual body weight, according to the systemic therapy dosing in overweight and obese adult cancer patients [10].

Blood Sample Collection

All the participants were requested to provide their peripheral venous blood samples (10 mL) during the third chemotherapy cycle in sterile coagulation tubes to aid in the separation of serum. The samples were centrifuged at 1,000 x g for 10 minutes after being allowed to coagulate at room temperature for 30 minutes, then another round of centrifuging was done at a temperature of 4 °C to achieve the maximum serum separation and to preserve the analytes. The serum was subsequently aliquoted into sterile cryovials and stored at -80 °C until it was subjected to biochemical and inflammatory analysis. They were also observed to measure leptin, interleukin-1 β (IL-1 β), high-sensitivity C-reactive protein (hs-CRP), and monocyte chemoattractant protein-1 (MCP-1/CCL2) and the tumor-associated biomarker cancer antigen 15-3 (CA 15-3) as a marker of tumor activity.

Serum Biomarker Analysis

Serum leptin, IL-1 β , MCP-1, and hs-CRP concentrations were quantified using high-sensitivity ELISA kits from Elabscience® (China), while CA15-3 levels were measured via a quantitative ELISA kit from Calbiotech® (USA). All assays were performed in duplicate to ensure analytical accuracy. Optical density was measured using a microplate reader (BioTek ELx800™, USA) at the appropriate wavelength specified by each kit. Concentrations of biomarkers were calculated from standard calibration curves generated using known concentrations of standards supplied with each assay kit.

Statistical Analysis

The relationship between different groups and their influence on the study's parameters was determined using the Statistical Analysis System (SAS) software (2018). In this study, a T-test was used to test means.

Results

Body mass index differed significantly among the three study groups, as shown in Figure 2. The normal-weight group had a mean BMI of 22.01 ± 0.31 kg/m², which was significantly lower than that of the overweight group (27.86 ± 0.29 kg/m², $p < 0.001$). BMI was further increased in the obese group (34.92 ± 0.41 kg/m²), as shown in (Figure 3); A significant difference was noticed when compared with both the normal-weight and overweight

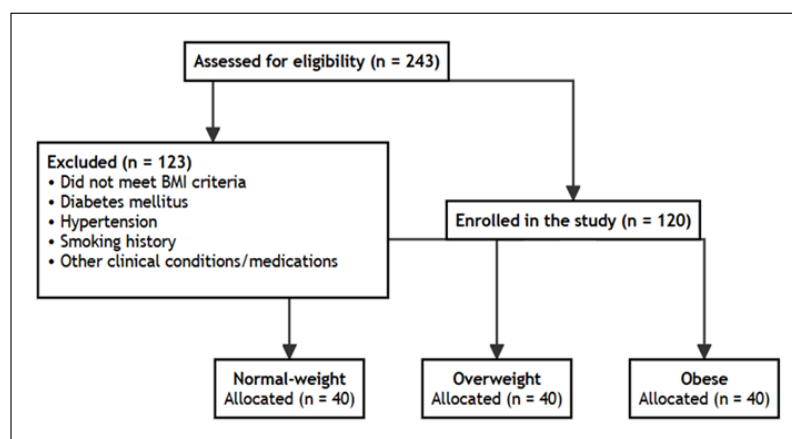


Figure 1. CONSORT-style Flow Diagram of Participant Recruitment and Selection

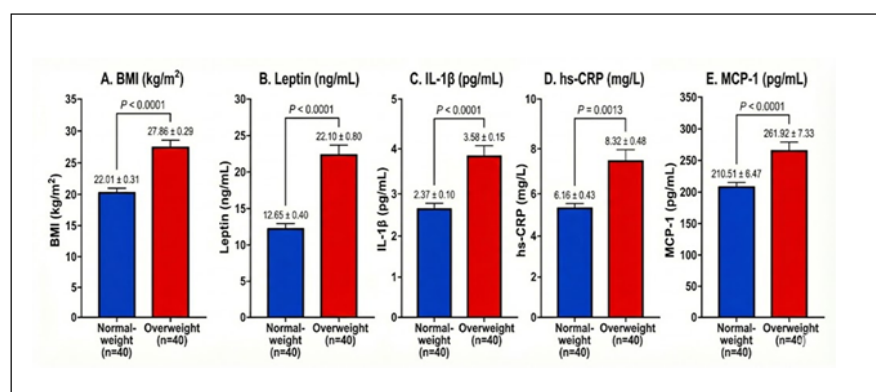


Figure 2. Comparison of the BMI and Inflammatory Biomarkers between the Normal-weight and Overweight Participants with Stage II Breast Cancer. Data are presented as mean ± SEM. ***p < 0.001

groups (p < 0.001 for both comparisons).

Serum leptin levels increased markedly with increasing BMI. Leptin concentrations were significantly higher in the overweight group (22.10 ± 0.80 ng/mL) compared with the normal-weight group (12.65 ± 0.40 ng/mL, p < 0.001) (Figure 2). The obese group exhibited the highest leptin levels (35.75 ± 1.24 ng/mL), which were significantly greater than those observed in both the normal-weight (p < 0.001) and overweight groups (p < 0.001) (Figure 3) and (Figure 4).

A significant increase in IL-1β levels was observed across BMI categories. The normal-weight group showed a mean IL-1β level of 2.37 ± 0.10 pg/mL, which increased significantly in the overweight group (3.58 ± 0.15 pg/mL, p < 0.001) (Figure 2). IL-1β concentrations were highest in the obese group (4.81 ± 0.19 pg/mL), with significant differences compared to both the normal-weight and overweight groups (p < 0.001 for both) (Figure 3) and (Figure 4).

Mean hs-CRP levels were 6.16 ± 0.43 mg/L in the normal-weight group and increased significantly in the overweight group (8.32 ± 0.48 mg/L, p = 0.001) (Figure 2). A further elevation was observed in the obese group (10.41 ± 0.56 mg/L), which was significantly higher than both the normal-weight (p < 0.001) and overweight groups (p = 0.007). (Figure 3) and (Figure 4).

MCP-1 concentrations differed significantly among all study groups. The normal-weight group had a mean

MCP-1 level of 210.51 ± 6.47 pg/mL, which increased significantly in the overweight group (261.92 ± 7.33 pg/mL, p < 0.001) (Figure 2). The highest MCP-1 levels were observed in the obese group (318.74 ± 8.41 pg/mL), showing significant differences compared with both the normal-weight and overweight groups (p < 0.001) (Figure 3) and (Figure 4).

In contrast to inflammatory and adipokine markers, (Table 1) showed that Serum CA15-3 levels were significantly elevated in overweight breast cancer patients compared to normal-weight patients (31.87 ± 0.72 vs. 24.20 ± 1.02 U/mL; t = -6.15, p < 0.001) and in obese patients compared to normal-weight patients (31.26 ± 0.74 vs. 24.20 ± 1.02 U/mL; t = -5.58, p < 0.001). No significant difference was observed between overweight and obese groups (31.87 ± 0.72 vs. 31.26 ± 0.74 U/mL; t = 0.60, p = 0.55, NS).

Discussion

This study provides evidence that obesity is associated with a distinct pro-inflammatory biological milieu in women with stage II breast cancer receiving standard doxorubicin–cyclophosphamide chemotherapy, with biomarker assessment performed during the third treatment cycle. The progressive elevation of obesity-related inflammatory and adipokine markers observed across increasing BMI categories highlights the role of

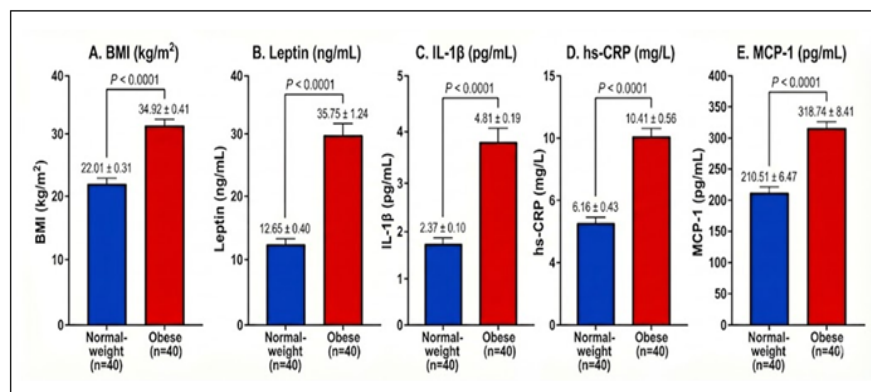


Figure 3. Comparison of the BMI and Inflammatory Biomarkers between the Normal-weight and Obese Participants with Stage II Breast Cancer. Data are presented as mean ± SEM. *** $p < 0.001$

excess adiposity as an active biological modifier during chemotherapy rather than a passive anthropometric characteristic.

Obesity is well known as a condition of chronic and low-grade systemic inflammation caused by adipose tissue growth, the infiltration of immune cells, and the imbalanced release of adipokines and cytokines [11]. In the current investigation, the indicators of this inflammatory condition, such as leptin, interleukin-1β, high-sensitivity C-reactive protein, and monocyte chemoattractant protein-1, were consistently high in overweight and obese patients as compared to the normal weight patients. Such findings are not new, as it is evidenced by the existing literature that adipose tissue is an endocrine and immunologically active organ that is able to modulate the tumor and host environment when treating cancer [12].

One of the most important adipokines, leptin, is reported to induce breast cancer cell proliferation, angiogenesis, and survival by stimulating several oncogenic signaling pathways, such as JAK/STAT, PI3K/Akt, and MAPK cascades [13]. Higher leptin concentration in patients with obesity could thus decrease the sensitivity of tumor cells to apoptosis caused by chemotherapy, they might decrease the usefulness of the cytotoxic drugs like doxorubicin and cyclophosphamide [14]. The leptin elevation that is significantly greater during active chemotherapy is an indication that endocrine signaling associated with obesity remains despite therapy and can disrupt optimal therapy [15].

On the same note, the presence of elevated levels of interleukin-1β demonstrates the elevated response of obese patients to inflammasome-related pathways [16]. IL-1β plays a pivot role in the connection between inflammation caused by obesity and tumor aggressiveness,

immune dysregulation, and anticancer therapy resistance [17]. IL-1β is upsurged in chemotherapy could be a contributing factor to a tumor microenvironment that supports survival, invasion and decreased drug sensitivity hence restricting treatment efficacy even with early-stage disease [18].

The increase in systemic inflammatory mediators like high-sensitivity C-reactive protein is also another indication of increased inflammatory load in overweight and obese patients [19]. Changing the drug metabolism, dysfunctional immune surveillance, and adverse cancer prognosis were linked to systemic inflammation [20]. Through the prism of chemotherapy, an increased inflammatory condition can affect the pharmacokinetics and pharmacodynamics, which results in inter-individual differences in the effectiveness and tolerance of treatment [21]. Although West and Asian studies have demonstrated that there is a high baseline inflammation (leptin and hs-CRP) in obese patients with breast cancer [22, 23] ours is the first to demonstrate that this inflammation continues severely through active cytotoxic therapy in an Iraqi population. As an example, leptin of our obese group was also extremely high following prolonged doxorubicin-cyclophosphamide administration. This highlights the fact that the intra-treatment inflammatory burden of Middle Eastern women is not reflected in the usual tumor markers such as CA153.

Moreover, elevated concentrations of monocyte chemoattractant protein-1 mean improved recruitment of monocytes and macrophages to the adipose and tumor tissues [24]. Infiltration by macrophages through MCP-1 helps in the formation of an inflammatory microenvironment of pro-tumorigenic phenotype (angiogenesis, immunosuppression, and remodelling of the ECM). These alterations have been observed to favor

Table 1. Comparison of Serum CA15-3 Levels among Normal-Weight, Overweight, and Obese Breast Cancer Patients with stage II breast cancer. Data are presented as mean ± SEM. *** $p < 0.001$

Comparison	Normal-weight (Mean ± SE)	Overweight (Mean ± SE)	Obese (Mean ± SE)	p-value
Normal-weight vs Overweight	24.20 ± 1.02	31.87 ± 0.72	—	< 0.001
Normal-weight vs Obese	24.20 ± 1.02	—	31.26 ± 0.74	< 0.001
Overweight vs Obese	—	31.87 ± 0.72	31.26 ± 0.74	0.55 (NS)

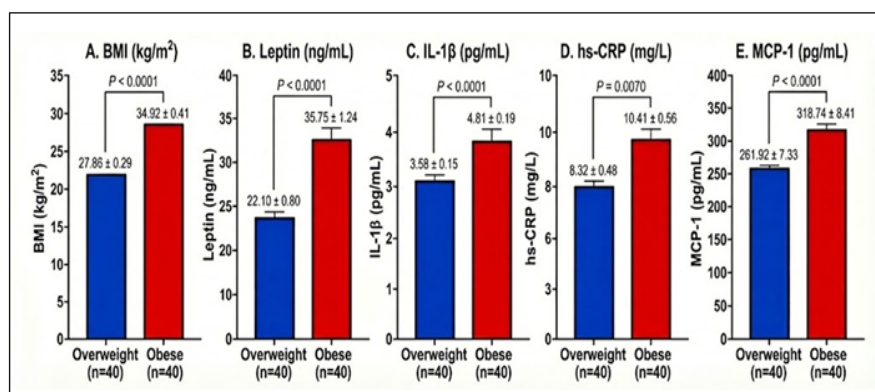


Figure 4. Comparison of the BMI and Inflammatory Biomarkers between the Overweight and Obese Participants with Stage II Breast Cancer. Data are presented as mean ± SEM. ***p < 0.001

tumor survival and can be antagonistic to the effect of chemotherapy in favor of tumor cell survival and repair processes [25].

Interestingly, there was a significant increase in the level of serum CA15-3 in the normal-weight to overweight/ obese breast cancer patients. However, it was not significant between the overweight and obese groups. The results show that CA15-3 can be used as a diagnostic tool to identify any BMI variations in tumor markers expression, whereas it highlights the weakness of using traditional tumor markers in isolation as a tool to understand the biological effects of obesity on chemotherapy [26]. The cause of the CA15-3 plateau between the overweight and the obese groups might be due to a biological saturation in tumor marker shedding or hemodilution due to an increased plasma volume in severe obesity. Also, CA15-3 levels could be affected by reduced hepatic clearance or a change in the liver during obesity. It is this non-linear relationship that justifies the addition of a dynamic inflammatory panel, in that it is possible that standard tumor markers might not reflect the true biological and inflammatory burden [27].

The combination of tumor markers and inflammatory biomarkers (leptin, IL-1β, hs-CRP, MCP-1) is capable of giving a complete evaluation of host-tumor-treatment interaction. Taken together, such results indicate that obesity sets up a state of enduring inflammatory and adipokine-filled environment when undergoing chemotherapy as a patient with the II stage of breast cancer [28]. Direct measurement of chemotherapy response was not carried out and yet, the resulting inflammatory profile is biologically aligned with those mechanisms that decrease chemosensitivity and favor resistance to treatment. These findings warrant the idea that the progression of obesity-related inflammation creates a biological environment that may negatively affect the efficacy of conventional chemotherapy treatments and provide support to the need to consider the host metabolic and inflammatory status during optimization of treatment options [29].

In conclusion, this paper reveals that obesity in women who have stage II breast cancer is linked to the presence of a chronic inflammatory and adipokine-filled biological condition during the doxorubicin-cyclophosphamide chemotherapy. The increase in leptin, IL-1β, hs-CRP,

and MCP-1 in overweight and obese patients indicates chronic systemic inflammation that could have an effect on the tumor microenvironment and potentially lower chemosensitivity, and CA15-3 levels rose significantly between normal-weight and overweight/obese patients. These results show the significance of the investigation of the role of obesity-related inflammatory status as an applicable biological determinant in the management and personalization of breast cancer chemotherapy.

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Statement of Ethics

The study was accepted by the scientific and ethics committee of Ibn Sina University of Medical and Pharmaceutical Sciences, Iraq, Baghdad.

Competing Interests

The authors do not have any financial or non-financial conflicts of interest.

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